INSEP

Département de la Recherche

Laboratoire Sport, Expertise et Performance
Équipe d'Accueil 7370

Cyril Schmit

LE PHÉNOMÈNE DE FATIGUE EN CONDITIONS EXTRÊMES : LE « FLUSH MODEL » À L'ÉPREUVE DES JEUX OLYMPIQUES

THÈSE DE DOCTORAT D'UNIVERSITÉ

Soutenue publiquement
le 21 novembre 2016

Jury composé de :

Romuald Lepers,
Professeur des Universités – Laboratoire INSERM U1093, Université de Bourgogne

Romain Meeusen,
Professeur des Universités – Université libre de Bruxelles

Jeanick Brisswalter,
Professeur des Universités – Université de Nice Sophia Antipolis

Charles-Yannick Guezennec,
Pr agrégé du service de santé des armées – Centre hospitalier de Perpignan

Christophe Hausswirth,
HDR – INSEP, Université de Nice Sophia Antipolis

Yann Le Meur,
Chercheur – INSEP, Université de Nice Sophia Antipolis

ÉCOLE DOCTORALE	SPÉCIALITÉ	DOMAINE SCIENTIFIQUE
Sciences du sport, de la motricité et du mouvement humain	Sciences du sport et du mouvement humain	Département sciences humaines et humanités

Université Paris-Descartes

UNIVERSITÉ PARIS DESCARTES

Liberté • Égalité • Fraternité
RÉPUBLIQUE FRANÇAISE
MINISTÈRE DE LA VILLE, DE LA JEUNESSE ET DES SPORTS

INSEP
Terre de Champions

UNIVERSITÉ PARIS DESCARTES

UFR des Sciences et Techniques des Activités Physiques et Sportives

École doctorale : Sciences du sport, de la motricité et du mouvement humain

Spécialité : Sciences du sport et du mouvement humain

Domaine scientifique : Département sciences humaines et humanités

THÈSE

en vue de l'obtention du grade de

Docteur de l'Université de Paris Descartes

LE PHÉNOMÈNE DE FATIGUE EN CONDITIONS EXTRÊMES : LE « FLUSH MODEL » À L'ÉPREUVE DES JEUX OLYMPIQUES

présentée et soutenue publiquement par

Cyril Schmit

le 21 novembre 2016

devant le jury composé de

Rapporteurs

Romuald Lepers, Professeur des Universités – Université de Bourgogne
Romain Meeusen, Professeur des Universités – Université libre de Bruxelles

Examinateur

Charles-Yannick Guezennec, Professeur agrégé du service de santé des armées, HDR – Centre hospitalier de Perpignan
Jeanick Brisswalter, Professeur des Universités – Université Nice Sophia Antipolis

Directeur et Co-directeur

Christophe Hausswirth, HDR – INSEP, Université de Nice Sophia Antipolis
Yann Le Meur, Chercheur – INSEP, Université de Nice Sophia Antipolis

REMERCIEMENTS

Une thèse sur l'étude du phénomène de fatigue t'amène par définition, inéluctablement à prendre la dimension de ton sujet. Travaille ! Écoute ! Analyse ! Explique ! Publie ! Repos ?? « Dans une prochaine vie, voire celle d'après… » La fatigue en théorie, et en pratique. Et l'expérience de ton sujet d'étude te rappelle chaque jour la persévérance nécessaire inhérente à une vie de doctorant. Dans ce cadre il n'est pas un moment où tu ne sois amené à mettre ta personne à l'épreuve du doute et des efforts de régulation qui l'accompagnent. Ces trois dernières années, ce doute a pourtant était absent d'une composante de ma vie : celle humaine. Et ce, pour la simple raison que les personnes ci-dessous ont à leur manière contribué à cette persévérance dont il a fallu faire preuve.

Christophe, merci pour ta confiance. Elle a déclenché et accompagné ces trois ans. Le chemin aurait été différent si tu n'avais pas réalisé ce pari initial. Merci pour l'ensemble des influences que tu as exercées à mon égard, de par ton statut ou plus subtilement par tes valeurs, et qui pèsent encore sur mes orientations.

Yann, merci. Pour beaucoup, mais surtout ton exemplarité. Elle m'accompagne depuis maintenant dix ans. Avancer avec toi a toujours été constructif, tant en science qu'en tant que personne. Merci d'avoir initié cette thèse et de l'avoir permise, en même temps que moi, de murir.

Erwan, Bertrand, Yolaine et Guy, merci de m'avoir suivi pendant ces trois ans, et permis de contribuer à faire avancer les choses. Vous aussi, vous êtes à l'origine de ce travail, et j'espère que celui-ci vous aidera encore à argumenter et rayonner comme vous le souhaitez.

Romuald Lepers, Romain Meeusen, merci pour le temps que vous allouez à ces travaux de thèse et l'expertise que vous acceptez d'y apporter. Charles-Yannick Guezennec, Jeanick Brisswalter, merci d'assister à la soutenance de cette thèse. Vous avoir *tous* à mes côtés pour conclure cette aventure demeure un privilège.

Anaël, Laurie-Anne, mes équipiers de doctorat légendaires. À 7h du mat' en short dans la Thermo, ou à se challenger pour la plus belle des « t'as pas fait une thèse si… », il restera grâce à vous de cette expérience une agréable atmosphère de soutien, et de rires, qui a été pour moi une ressource indispensable. Indispensable. Merci !

À Céline, Cyril, loulou et Manon, merci encore et toujours pour votre bienveillance et votre amour. À mes parents, merci pour votre soutien de toujours.

À Lo, Mo, Elo, pour m'avoir accompagné et compris, et encore aujourd'hui. À Gégé, Ness, pour votre curiosité, et nos échanges simples. À Anto, Mika, Kris, pour avoir fait de *notre* ballon rond la source de tout cela.

À Guigui, Séb, Laura G., Virha, pour votre authenticité. Vous êtes des repères pour moi.

À Chris. E.S., pour ton merveilleux enthousiasme et ta saisissante sincérité.

À Catherine, pour ton efficience et ton amour de la transmission auxquels j'ai, précocement et fort heureusement, pu bénéficier.

À nos triathlètes, Titi, Ben, Derville, The King, Manu, Greg et tous les autres. Qui de mieux que vous pour des protocoles longs de 4 mois, en manip à 6h du matin ou jusqu'à 22h ?! Assidus, motivés et rigoureux (souvent), la recette parfaite d'un cocktail de perfs… solides !

TABLE DES MATIÈRES

INTRODUCTION

La performance sportive dans les sports d'endurance de haut niveau porte en elle une signature : elle ponctue et authentifie les sollicitations psychologiques et physiologiques de plusieurs années de travail qui, pour ne pas demeurer désavouées, sont amenées à se sublimer lors d'une course unique. Face à un rapport coûts/bénéfices en effet souvent critique – bien qu'inhérent au quotidien de l'athlète – la réponse demeure pourtant aussi souvent évidente : le jeu en vaut la chandelle, et les Jeux Olympiques par-dessus tout !

Ce travail de thèse a eu pour ambition de mieux comprendre les facteurs psycho-physiologiques sous-tendant la performance sportive en endurance dans le contexte particulier de la préparation des athlètes aux Jeux Olympiques de Rio 2016. Ainsi, les travaux présentés plus loin ont été raisonnés à partir d'approches temporelles (chronique et aigüe) et fonctionnelles (rapportés aux conditions écologiques) de la performance. Dans cette perspective, le « Flush Model » de Millet (2011) a constitué un point d'appuis.

1. Le « Flush Model », à l'origine

a) À l'origine étaient les modèles catastrophiques

Le développement de facteurs physiologiques clés de la performance en endurance constitue un fil rouge des entraînements des athlètes de haut niveau (détails en point 2.). Dans ce type de performance, c'est donc logiquement que le phénomène de fatigue se trouve théorisé au regard de cinétiques physiologiques spécifiques (*e.g.,* la déplétion des réserves énergétiques, la température interne). Dans cette diversité de conceptions une convergence s'établit néanmoins, qui suggère que la mise en activité de l'organisme au sein de conditions de pratique données (dénivelé du parcours, conditions ambiantes, *etc.*) précipiterait le sujet vers l'atteinte inéluctable d'un plafond physiologique préétabli. De façon ultime, ce plafond interdirait tout effort supplémentaire (Abbiss & Laursen, 2005). Dès lors, un peu à l'image d'un jeu de dominos appliqué à la physiologie humaine, amorcer le système dans un contexte de pratique donné reviendrait à prédire ce qu'il adviendrait à l'instant t+1.

Dans cette logique, le modèle cardiovasculaire de la fatigue rationnalise la performance en endurance sur la base d'une incapacité du cœur à subvenir aux besoins en substrats énergétiques des muscles (Delp & Laughlin, 1998). Tout autrement, les modèles énergétique et thermorégulatoire rapprochent l'arrêt de l'exercice de l'épuisement des stocks d'adénosine triphosphate et de l'atteinte d'une température centrale critique (~40°C), respectivement (Nielsen et *al.*, 1997 ; Schulman & Rothman, 2001). Le modèle neuromusculaire comprend, pour sa part, la fatigue en tant que réductions de l'intensité de la commande motrice et de la propagation de l'influx nerveux jusqu'aux muscles (Millet et *al.*, 2002). Ces quelques propositions ne demeurent pas isolées (voir Abbiss & Laursen, 2005 pour une revue exhaustive) – ce qui dénote par là même leur applicabilité restreinte respective. En effet, plusieurs échos se sont soulevés face à ces modèles, respectivement en raison de :

i) la capacité du muscle à extraire l'oxygène du sang en plus grande quantité en cas de perfusion limitée (*e.g.,* Gonzàlez-Alonso & Calbet, 2003) ;

ii) la préservation des stocks d'adénosine triphosphate contre un déclin inférieur à ~40% des réserves initiales (*e.g.,* Green, 1997) ;

iii) l'observation chez certains athlètes de températures centrales dépassant (ou à l'inverse, loin de tutoyer) les 40°C lors d'épreuves compétitives (*e.g.,* Ely et al., 2009) ;

iv) la capacité évidente à réaliser des sprints finaux plus rapides que les allures moyennes de course.

Dans ce contexte, plus loin encore que renforcer le caractère partiel de chaque modèle dans la compréhension du phénomène de fatigue, ces inconsistances ont questionné le fondement « catastrophique » même des argumentaires, et invité à la prise en compte d'autres explications. Les études investiguant les corrélats psychobiologiques de la performance en endurance ont participé de cette démarche de rationalisation.

Les travaux de l'équipe de Samuele Marcora sont reconnus pour avoir outrepassé le focus physiologique de l'approche de l'état d'épuisement et magnifier le rôle de facteurs psychologiques dans la performance en endurance (*e.g.,* l'autosuggestion, l'amorçage

visuel, la fatigue mentale) (Blanchfield et *al.*, 2014a,b ; Marcora et *al.*, 2009). Dans cet éventail de travaux, une étude en particulier est venue invalider le fondement des modèles catastrophiques abordés. De façon spécifique, celle-ci requérait des participants d'abord de parcourir la plus grande distance possible à vélo à 80% de leur puissance maximale aérobie puis, au moment même d'abandonner délibérément l'effort, de poursuivre leur activité avec une épreuve de sprint court. Les résultats du sprint indiquent une puissance de pédalage trois fois supérieure à celle requise par l'épreuve en temps-limite (~731Watts vs. ~242Watts, respectivement ; Figure 2) (Marcora & Staiano, 2010). À noter qu'un modèle linéaire/catastrophique de fatigue ne concevrait pas un tel comportement chez un individu conduit à l'épuisement en raison de valeurs physiologiques devenues si critiques qu'elles annihileraient toute production de force.

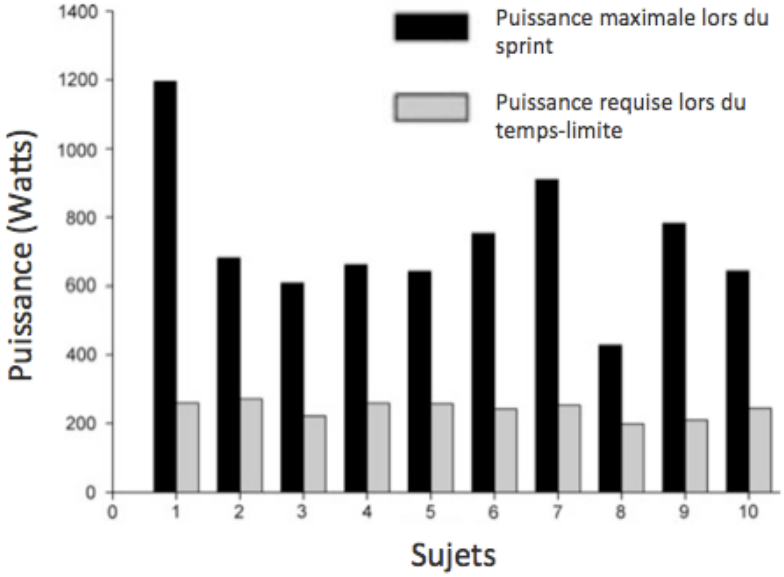


Fig. 2. Puissance de pédalage maximale volontaire de chaque sujet obtenue immédiatement après épuisement, et puissance correspondante à l'épreuve en temps-limite. D'après Marcora & Staiano (2010).

Alors, telle une révolution, ce type d'étude est venu redistribuer la compréhension du phénomène de fatigue en faveur d'approches systémiques de l'individu.

b) De la linéarité à la complexité : le Flush Model

La dimension partielle des modèles catastrophiques a conduit à envisager des perspectives moins unilatérales du phénomène de fatigue. Spécifiquement, le rationnel de Millet (2011) s'est constitué sur la base d'écueils relatifs à :

i) la méconnaissance du rôle des altérations physiologiques dans le cadre d'efforts sous-maximaux (*i.e.,* indépendamment des mesures de force maximale) ;

ii) l'influence de ces altérations dans le cadre d'épreuves librement gérées (*i.e.,* relativement aux stratégies de gestion d'allure) ;

iii) l'influence de paramètres *non* liés à l'exercice dans la détermination de la performance en endurance.

Trois éléments qui contrastent donc avec la dimension écologique d'une épreuve compétitive où, par exemple, les épreuves en endurance ne requièrent que rarement (voire jamais) le développement d'une force maximale et peuvent être influencées par de courtes nuits de sommeil. En conséquence, pour répondre à la problématique d'un éventuel transfert entre les résultats de laboratoire existants et la réalité du terrain, Millet (2011) a proposé un modèle illustré par le fonctionnement d'une chasse d'eau (Figure 3) et organisé autour de trois repères :

i) la pénibilité ressentie à l'exercice (RPE, pour '*rating of perceived exertion*'), en tant que régulateur princeps de la performance en endurance (*i.e.,* le flotteur dans la cuvette) ;

ii) l'instabilité de ce flotteur à partir d'un ensemble de facteurs, soit alimentant la cuvette en eau (*i.e.,* le tuyau de remplissage), soit participant à sa vidange (*i.e.,* la valve d'évacuation) ;

iii) l'arrêt de l'exercice dès l'atteinte par le flotteur d'une limite haute, elle-même modulable (*i.e.,* le réservoir de sécurité).

Chacun de ces points est développé plus en détails ci-dessous.

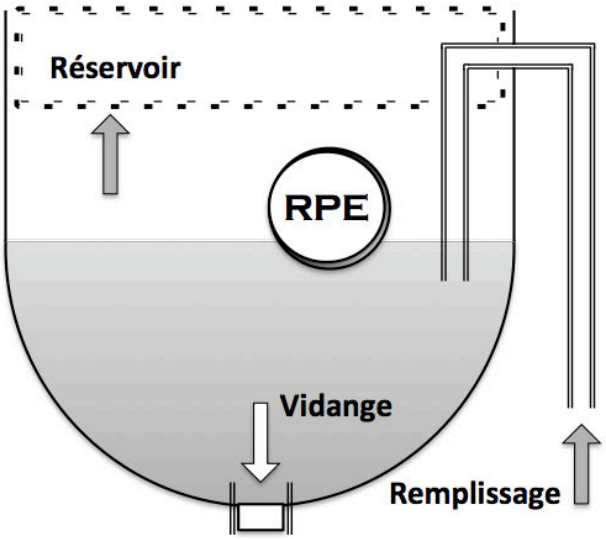


Fig. 3.

Représentation du Flush Model (Millet, 2011).

Notes. RPE = *Rating of Perceived Exertion.*

L'essence du Flush Model repose sur sa disposition à cristalliser la performance en endurance autour d'un construit largement employé en sciences du sport : la sensation de difficulté perçue à l'exercice, ou RPE. Cette sensation est généralement rapprochée de l'intensité des sollicitations cardiovasculaire, pulmonaire, musculaire et centrale à l'exercice (Borg, 1998) – apportant ainsi un écho aux modèles précédents. Dans la mesure où cette estimation est globale et personnelle, le modèle l'appréhende comme

un régulateur de la performance qui, selon sa magnitude, détermine si l'intensité d'exercice peut être augmentée ou diminuée par l'individu pour atteindre l'objectif visé (*e.g.,* gagner l'épreuve, terminer la course, *etc.*). En conséquence, la force de cette conception repose spécifiquement sur sa dimension prédictive. En effet, comme illustré en Figure 4 lors de performances en temps-limite, les valeurs RPE relevées à un instant prédéfini de l'épreuve permettent de présumer du moment d'arrêt de l'effort (Fig. A) et ce, quelque soient l'environnement ou l'intensité de l'exercice (Fig. B). De même, dans le cadre d'efforts librement gérés, la pénibilité à l'exercice (*e.g.,* 15 sur l'échelle de Borg, 1998) gouverne l'intensité consentie eu égard aux conditions de pratique *e.g.,* la température ambiante (Fig. C).

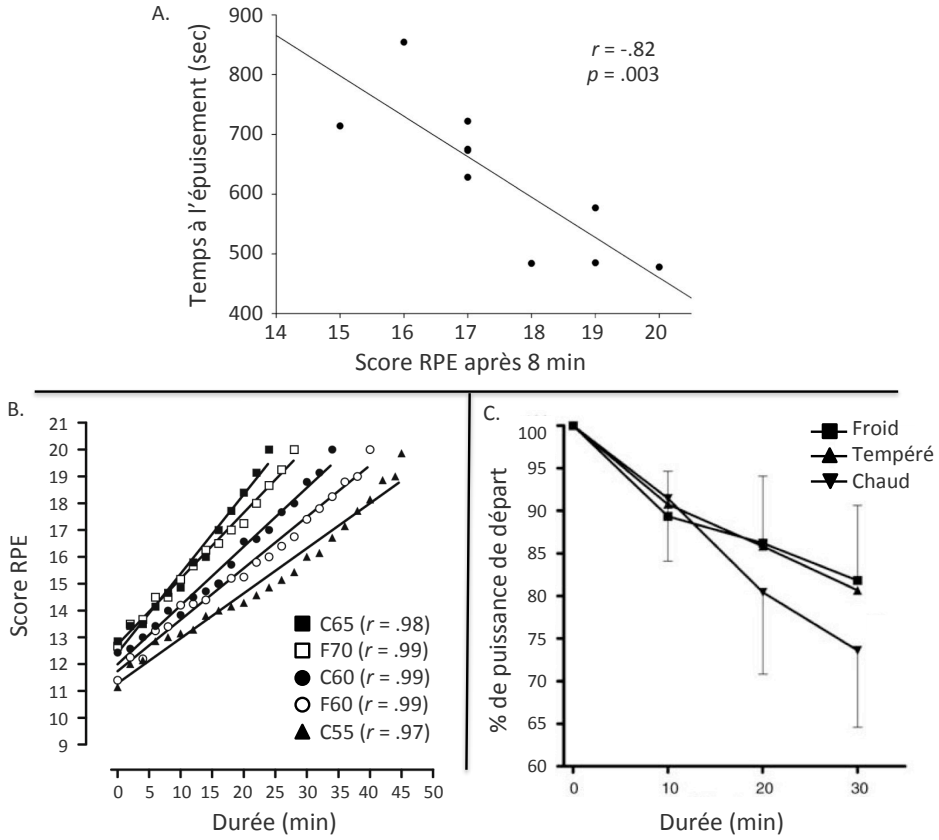
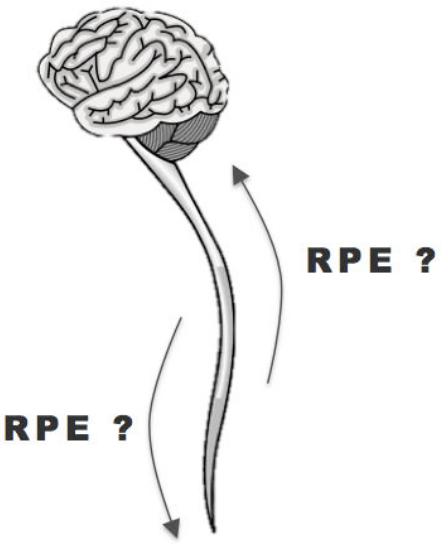


Fig. 4. Représentations graphiques du RPE comme outil de régulation de la durée/l'intensité d'exercice, à partir de A. Marcora & Staiano (2010), B. Crewe et *al.* (2008), C. Tucker et al. (2006).
Notes. En figure B. les conditions « C » et « F » représentent des ambiances thermiques Chaudes (35°C) et Froides (15°C), respectivement. Les nombres associés représentent l'intensité de l'exercice de pédalage (% de puissance maximale aérobie).

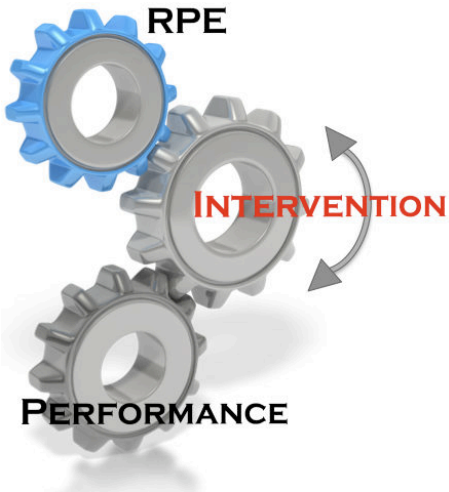


En contraste avec cette fiabilité de prédiction, les mécanismes à l'origine de l'évolution des valeurs RPE (*i.e.,* du remplissage de la cuvette en eau) restent affaires de débats. Brièvement, deux conceptions se sont récemment répondues. La première estime la pénibilité à l'exercice comme le résultat de l'intégration par le cerveau (possiblement le cortex cingulaire antérieur) des afférences propres aux différents récepteurs de l'organisme (nociceptifs, thermiques, de pression, métaboliques, osmotiques) (Amann et *al*., 2009). Cette représentation conclue des observations de réduction de la commande motrice dans des situations d'hypoxie (Millet et *al*., 2009), d'hypoglycémie (Nybo, 2003), d'hyperthermie (Tucker et *al*., 2006), ou de manipulations des fibres nerveuses III/IV (Amann et *al*., 2009). Mais les scores RPE pourraient ne pas être *directement* fonction de ces afférences périphériques et davantage corréler l'intensité de la commande motrice elle-même, renseignée aux aires sensorielles du cerveau via des décharges corticales parallèles (Marcora, 2009). Cette conception résulte pour sa part de travaux manipulant l'intensité des efférences et des afférences à l'exercice (Gallagher et *al*., 2001 ; Marcora et *al*., 2008) ou investiguant les pertes d'efficience dans la réponse motoneuronale et musculaire à l'influx nerveux (Millet, 2010). Ensemble, ces inconsistances théoriques complexifient la compréhension du phénomène de fatigue à l'exercice ; pour autant, comme développé ci-dessous, elles n'interfèrent pas avec le rôle médiateur effectif du RPE dans la détermination de la performance en endurance lorsque ce paramètre est manipulé expérimentalement.

D'un point de vue fonctionnel, le Flush Model est en effet rationnalisé sur la base d'interventions expérimentales ayant modulé le rapport 'RPE/intensité d'exercice' par rapport à des conditions contrôles. Cette modulation prend différentes formes.

De hautes valeurs RPE (*i.e.,* un haut niveau d'eau dans la cuvette) peuvent d'abord être précipitées *in situ* de l'exercice, en cas de conditions 'extrêmes' de pratique. Par exemple, des réserves musculaires pauvres en glycogène au départ de l'épreuve, ou un environnement thermique chaud pendant la course peuvent altérer le ressenti de l'individu et moduler progressivement (mais pas initialement) l'intensité d'exercice consentie (Baldwin et *al*., 2003 ; Racinais et *al*., 2015).

De façon intéressante, le niveau d'eau dans la cuvette peut aussi être modifié indépendamment du temps d'exercice effectif *i.e.,* en amont de l'effort, et induire un changement du niveau de performance. En effet, un écart de scores RPE est d'emblée observé suite à des interventions manipulant le niveau de ressources mentales du sujet par une activité cognitive prolongée préalable à l'exercice, un état de privation de sommeil ou l'annonce d'un événement décevant (Baden et *al*., 2005 ; Marcora et *al*., 2009 ; Martin, 1981). Dans ces conditions spécifiques, l'intensité d'exercice consentie pendant l'épreuve est alors revue à la baisse par l'athlète pour autoriser un ressenti consistant avec son plan initial de sensations.



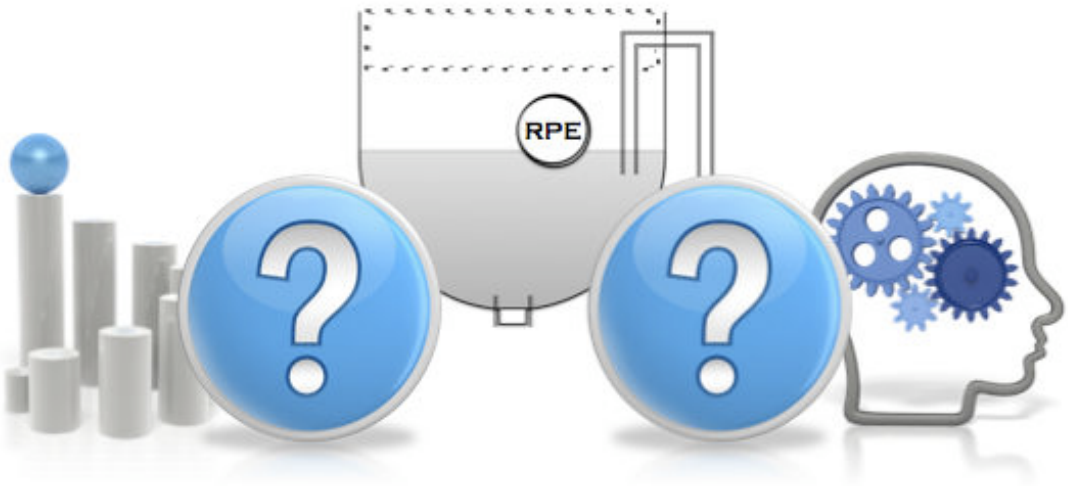
En contraste, une amélioration du niveau d'efficacité de l'athlète peut être envisagée sur la base d'interventions le soulageant de la pénibilité de l'exercice (*i.e.,* abaissant le niveau d'eau dans la cuvette). Dans ce cadre peuvent être considérées les stratégies nutritionnelles ou attentionnelles, potentiellement ergogéniques sous l'effet d'une altération des activations cérébrales (Chambers et *al*., 2009 ; Raglin, 2007). Spécifiquement, l'efficacité du rinçage de bouche à partir de boissons sucrées sur la performance en endurance pourrait s'opérer via la stimulation des neurones impliqués dans le « circuit de la récompense » (Chambers et a*l*., 2009), tandis que celle des stratégies de focus attentionnel pourraient s'effectuer via l'activation des zones corticales impliquées dans la gestion de la douleur (Lawrence et *al*., 2011).

En dernier lieu, le Flush Model définit la présence d'un 'réservoir de sécurité' estimé protéger l'individu d'éventuels traumatismes physiologiques liés à l'exercice. Autrement dit, l'atteinte de scores RPE maximaux (et donc l'arrêt de l'exercice) est estimée être concomitante de l'atteinte de ce réservoir (Kayser, 2003). De façon intéressante, le modèle accorde toutefois à cette limite subjective une marge de manœuvre, de sorte que l'individu pourrait parvenir à repousser l'arrêt de l'exercice lors de conditions exceptionnelles de pratique (*i.e.,* réduire la taille du réservoir). Des circonstances anecdotiques comme le défi d'un adversaire, la motivation d'une grande compétition (*e.g.,* les Jeux Olympiques) ou la recherche de survie, autoriseraient ce type de dépassement. Cette caractéristique illustre par là-même le fondement du Flush Model,

selon lequel c'est la volonté de se désengager de l'exercice qui conduit à arrêter la tâche, et non l'incapacité à la réaliser.

Ainsi, comparativement aux approches linéaires de la fatigue, la plus-value du Flush Model réside dans une approche interactive de leur fonctionnement respectif, qui plus est en parallèle de l'influence de variables psychologiques (parfois non liées à l'exercice) dans la détermination de la performance. Bien qu'hypothétique, cette représentation est forte d'appréhender le phénomène de fatigue dans une perspective écologique typique de la diversité des situations rencontrées par les athlètes en phase compétitive.

En revanche, malgré son ambition holistique, deux points font défaut à ce rationnel lorsqu'il est confronté à la performance dans le cadre de compétitions organisées (*e.g.*, les Jeux Olympiques). Précisément, la mise à l'épreuve du modèle à l'égard des problématiques d'entraînement reste non questionnée ; celles-ci ont spécifiquement trait aux notions de 'charges d'entraînement', de 'surcharge' et d' 'affûtage' et renvoient à une approche à moyen-terme de la performance. De plus, alors que la capacité d'un individu à rester lucide lors d'une épreuve conditionne aussi sa performance, la réponse cognitive à l'exercice n'est pas envisagée par Millet (2011). Cette réponse cognitive renvoie à l'efficacité des différentes opérations mentales requises lors de situations de prises de décision à l'exercice. Ces deux points sont abordés ci-dessous afin d'envisager plus avant les possibles liens entre les problématiques écologiques et le Flush Model.



2. Le Flush Model : extension à la performance en compétition

a) Une approche à moyen-terme de la performance

Les paramètres de consommation maximale d'oxygène ($\dot{V}O_{2max}$), de fraction d'utilisation de $\dot{V}O_{2max}$ et d'économie de course sont depuis bien longtemps reconnus pour limiter, et subséquemment prédire, la performance en endurance (*e.g.,* Bassett & Howley, 2000 ; Hill & Lupton, 1923). Leur développement représente ainsi un fil rouge des entraînements des athlètes en endurance de haut niveau ; pour autant, leur intégration au sein de problématiques d'entraînement plus larges demeure un enjeu contemporain.

Depuis quelques années, se multiplient en effet les investigations scientifiques structurées autour de la gestion de l'état de fatigue de l'athlète (*e.g.,* Bosquet et *al*., 2007 ; Seiler & Kjerland, 2006 ; Seiler, 2010). Ces travaux tentent d'objectiver les moyens permettant d'élever le potentiel de performance de l'athlète (*e.g.,* la régulation des charges d'entraînement), comme ceux facilitant l'expression de ce potentiel (*e.g.,* la régulation des stratégies d'allures). Cette problématique a ainsi la spécificité de concerner tant les composantes d'amélioration de la condition physique en amont de la compétition, que celles relatives à l'état de forme de l'athlète lors de la phase compétitive. Ces deux points sont abordés ci-dessous.

L'élévation du potentiel de performance de l'athlète en phase précompétitive suppose la planification de lourdes périodes d'entraînement, généralement effectuées dans l'espoir d'induire un gain d'efficacité consécutivement à la période d'affûtage (Meeusen et *al*., 2013). L'état de fatigue accompagnant ces périodes de surcharge peut néanmoins parfois dépasser la capacité de récupération de l'athlète, entraînant alors une baisse temporaire de son niveau de performance. Les travaux de notre laboratoire préalables à cette thèse ont montré l'absence d'intérêt d'une telle programmation d'entraînement pour la performance en endurance, en ce qu'elle s'accompagne de l'instauration d'un état de surmenage inhibant le rebond de performance subséquent (Aubry et *al*., 2014). En revanche, un gain d'efficacité est effectivement constaté chez des sujets conduits à un état de « fatigue aigüe », c'est-à-dire soumis à une surcharge d'entraînement qui n'engendre pas les

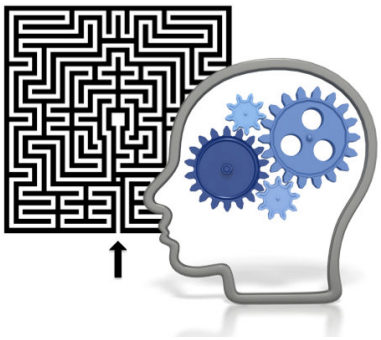
symptômes d'un état de surmenage. Ces observations soulèvent l'intérêt de mieux comprendre, pour prévenir, le développement du phénomène de surmenage susceptible de subvenir en phase précompétitive.

D'autre part, l'atteinte ponctuelle d'une performance maximale suppose l'optimisation des composantes psycho-cognitives relatives aux efforts en endurance. Comme présenté en partie 1, il est aujourd'hui établi que c'est l'*effort* investi par le sujet qui détermine sa performance et oriente la régulation de sa stratégie d'allure (Marcora et *al.*, 2009 ; Marcora & Staiano, 2010 ; McCarron et *al.*, 2013). Pour autant, quand bien même les sollicitations nerveuses à l'origine de cette sensation d'effort demeurent inhérentes au fonctionnement de l'organisme à l'exercice, leur implication dans la performance – et en particulier la performance cognitive – reste à préciser. Ces zones d'ombre laissent transparaître la nécessité d'une compréhension plus approfondie du fonctionnement psycho-cognitif à l'exercice physique, afin de faciliter l'expression par l'athlète de son plein potentiel.

Dans ce contexte, une partie de nos travaux s'est orientée vers l'identification chez des athlètes bien entraînés des soubassements psycho-cognitifs liés à l'instauration d'un état de fatigue induit par une période de surcharge d'entraînements, ou par l'exercice physique aigu. Eu égard à la vaste dimension de la problématique 'exercice-cognition', la partie suivante vise à clarifier ses liens possibles avec le Flush Model.

b) Le Flush Model : implications cognitives

Malgré son caractère multidimensionnel, le Flush Model n'est pas dédié à expliquer la réponse cognitive à l'exercice physique. Pourtant, certains de ses mécanismes pourraient être intriqués dans l'évolution de la capacité d'autorégulation à l'exercice, si précieuse pour diriger efficacement le comportement vers un but. En effet, le développement du phénomène de fatigue en course librement gérée est partiellement sous-tendu par la charge cognitive *i.e.,* la quantité d'opérations cognitives à résoudre (McCarron et *al.*, 2013). Ainsi, des liens peuvent être envisagés entre le rationnel de Millet (2001) et la réponse cognitive à l'exercice, afin de compléter la fonctionnalité du Flush Model.



Les fonctions exécutives représentent la facette spécifique des processus cognitifs sous-tendant la capacité d'autorégulation *i.e.,* les opérations permettant d'aligner délibérément le comportement vers un but (Hofmann et *al.*, 2012). Suivre un schéma de course prédéfini ou s'adapter avec efficacité aux aléas d'une épreuve peuvent constituer certains de ces buts et, consécutivement, requièrent de telles fonctions. À l'inverse des processus simples (*e.g.,* stimulus-réponse), les fonctions exécutives sont identifiées pour être des opérations lentes, coûteuses en effort et limitées en capacité (Evans, 2008). Parmi elles, la mise à jour de la mémoire de travail, la capacité d'inhibition comportementale ou de switch attentionnel demeurent les fonctions les plus couramment testées en laboratoire, et renvoient à des processus relatifs à l'implémentation d'un control cognitif orienté, la retenue d'impulses automatiques, et le changement rapide de schémas mentaux, respectivement (Jacobson & Matthaeus, 2014 ; Miyake et *al.*, 2000 ; Vestberg et *al.*, 2012). Les performances relatives à ces fonctions peuvent être mesurées par l'intermédiaire de tests 'exécutifs' (*e.g.,* tâche de Stroop, d'Eriksen, de N-*back*) et dépendent d'un recrutement spécifique et systématique du cortex préfrontal (PFC) (Miyake et *al.*, 2000).

À l'exercice physique, la réponse de performance relative aux fonctions exécutives peut être appréhendée sous deux angles :

i) une approche *aigue* de l'exercice suggère des effets contrastés selon les intensités, durées, et modes de pratique, ainsi que selon la condition physique de l'individu. Plus précisément, dépendamment de ces caractéristiques, l'exercice physique pourrait induire une élévation du niveau d'efficacité cognitive ou, à l'inverse, initier une détérioration des performances exécutives (Chang et *al.*, 2012 ; Lambourne & Tomporowski, 2010). Cette hétérogénéité de résultats dans l'analyse de l'interaction exercice-cognition est aujourd'hui reconnue, de même que restent à clarifier les mécanismes à l'origine de la dynamique des fonctions exécutives à l'exercice. Par exemple, quand bien même la réponse d'oxygénation cérébrale à l'exercice évolue relativement à certains des facteurs énoncés plus haut (*i.e.,* intensité d'exercice, condition physique ; Rooks et al., 2010), cette variable pourrait ne pas être

directement associée à la performance cognitive à l'exercice (Ando et *al.*, 2011 ; Ogoh et *al.*, 2014). À l'inverse, la réponse biochimique de l'organisme à l'exercice (McMorris et *al.*, 2016) et/ou l'intensité des afférences et efférences impliquées dans l'effort pourraient davantage se poser. En effet, l'activité du PFC est connue pour moduler la réponse de l'amygdale aux stimuli aversifs de l'organisme, comme la soif ou la douleur (Buhle et *al.*, 2013 ; Ochsner & Gross, 2008) ; l'activité du PFC évolue de plus parallèlement à l'augmentation de la commande motrice avec la durée de l'exercice (Fuster, 1997 ; Jenkins et *al.*, 1994). De telles implications neuronales posent alors les bases de possibles chevauchements entre les mécanismes propres au Flush Model (*i.e.,* à l'origine des variations de RPE) et ceux impliqués dans le fonctionnement exécutif à l'exercice.

ii) d'un point de vue *chronique*, deux temporalités sont à dissocier. À moyen-terme (3-4 semaines), la réponse cognitive à l'entraînement renvoie aux problématiques d'état de forme de l'athlète et, plus précisément, à la question de sa tolérance aux variations de charge d'entraînement précompétitive (*i.e.,* périodes de surcharge et d'affûtage). À cet égard, les données de la littérature démontrent une détérioration des performances cognitives (exécutives et non-exécutives) chez des sujets menés à un état de surmenage temporaire, cependant sans soubassements mécanistiques identifiés (Decroix et *al.*, 2016 ; Dupuy et *al.*, 2014 ; Le Meur et *al.*, 2013). Malgré cet écueil, une opinion consistante demeure, qui consiste à théoriser ce déclin de performance cognitive en conditions de surmenage relativement à une capacité réduite du cerveau à intégrer et traiter les stimuli afférents (Satz, 1993). Dans ce cadre, la compréhension plus fine de l'effet des variations de charge d'entraînement sur le fonctionnement cérébral demeure à investiguer, et pourrait rapprocher la performance cognitive du fonctionnement de « remplissage/vidange » propre au Flush Model.

Par ailleurs, à plus long terme (plusieurs mois), la réponse cognitive à l'entraînement fait écho à l'accélération du traitement de l'information et au développement neuronal inhérents à l'amélioration de la condition physique (Hillman et *al.*, 2008). À noter, cette composante à long-terme n'a pas fait l'objet d'investigations au sein de cette thèse.

La préparation aux Jeux Olympiques (JO) se dessine donc comme un chemin de croix en imposant, on l'a vu, une préparation à moyen-terme de la compétition (chronique puis aigüe). Pour autant, de façon plus particulière, cette préparation suppose aussi la prise en compte des composantes spécifiques à la compétition. Eu égard à l'éventuelle expérience par les athlètes de températures ambiantes inhabituelles lors des JO de 2016, la question de l'effort en chaleur s'est donc naturellement formulée.

c) La performance en ambiance olympique

De façon spécifique à Rio de Janeiro, nous avons entrepris un recensement des données météorologiques locales enregistrées chaque jour (plage horaire 10h-20h) de chaque mois d'août 2010 à 2015, et relevé des conditions environnementales proches de 24 ± 1°C et de 61 ± 4% d'humidité relative (Figure 1). *A priori*, ces valeurs n'incitent pas à une préparation spécifique des athlètes à la compétition ; cependant, les incidences effectivement observées de ce type de conditions sur le niveau de performance des athlètes en endurance invitent à une toute autre perspective (voir plus bas). De plus, une analyse plus précise des heures les plus chaudes de la journée (11h-15h) indique aussi l'éventualité pour les athlètes d'être confrontés à des ambiances thermiques atteignant 37°C et 100% d'humidité. En conséquence, l'occurrence possible de fortes chaleurs n'est apparue nullement à écarter.

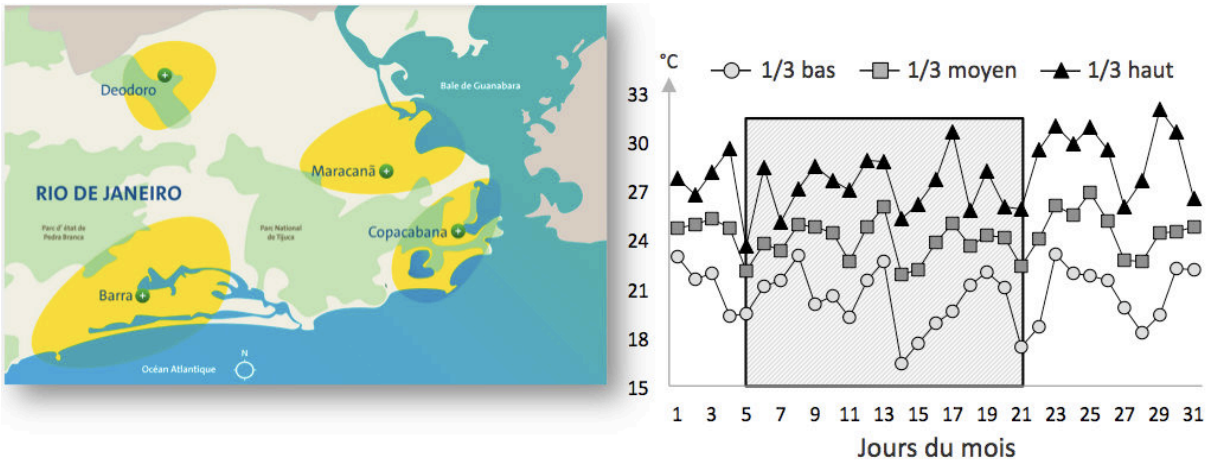


Fig. 1. Représentation des moyennes de température à Rio de Janeiro par journée du mois d'août lors des années 2011 à 2015.
Notes. Les zones de l'image coloriées en jaune représentent les différents sites de compétition. La case grisée du graphique représente la période de déroulement des Jeux Olympiques. Chaque 1/3 de température affiché correspond à la moyenne des valeurs basses, moyennes et hautes par journée (plage horaire 10h-20h).

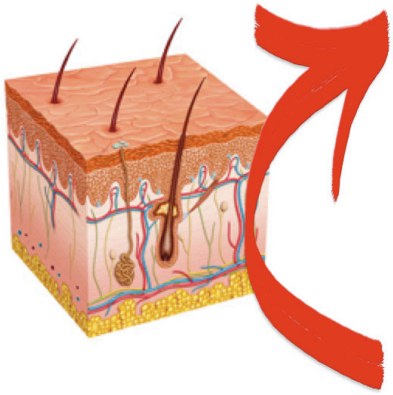
Des sports olympiques comme l'aviron, l'athlétisme, le cyclisme sur route, le football, le hockey sur gazon, le pentathlon moderne, le rugby, le tennis ou le triathlon représentent certaines des activités les plus exposées aux effets délétères de conditions ambiantes d'exercice chaudes. La performance dans ces activités suppose en effet une forte production de chaleur endogène (*i.e.,* libérée par l'activité musculaire) stockée en sus d'une accumulation de chaleur exogène (*i.e.,* issue de l'environnement). Par ailleurs, des expositions passives et prolongées à la chaleur sont aussi susceptibles d'affecter le niveau d'efficacité des athlètes au sein d'autres activités (*e.g.,* tir, tir à l'arc).

Le constat lié à la chaleur (>25°C) reste en effet sans appel : la performance en endurance est globalement détériorée comparativement à des conditions plus fraîches (<25°C) (Guy et *al*., 2014). De façon plus précise, les recherches investiguant les corrélats physiologiques de l'état d'hyperthermie suggèrent qu'une performance optimale est atteinte à des températures ambiantes proches de 10°C, et que chaque degré de température supplémentaire à ce seuil induit une baisse de 0,3-0,9% de la performance (Cheuvront et *al*., 2010 ; Racinais et *al*., 2015). Si cette donnée justifie à elle seule le caractère préjudiciable de conditions considérées « tempérées » (*e.g.,* 24°C), elle suppose aussi les influences concomitantes d'autres 'facteurs thermiques' sur la performance en endurance. Par exemple, en rapport à la production de chaleur métabolique évoquée plus haut, la durée de l'épreuve apparait influencer la décroissance de performance *e.g.,* par ~2%, ~7% et ~16% pour des performances initiales de ~6min, ~30min et ~70min en environnement tempéré, respectivement (Altareki et *al*., 2009 ; Tatterson et *al*., 2000 ; Racinais et *al*., 2015). Ensemble, ces observations évoquent une relation étroite entre l'état thermique de l'athlète en compétition et son niveau d'efficacité, partiellement médiée par sa capacité à thermoréguler.

Le phénomène de thermorégulation est inhérent au déséquilibre de la balance thermique de l'Homme. Spécifiquement à la chaleur (*vs*. des conditions fraiches), les incidences d'un tel phénomène sur la limitation de la performance en endurance sont généralement expliquées au regard du type d'effort à fournir (Nybo, 2012). Dans le cas

d'efforts maximaux (*e.g.,* le 10 000m), c'est une limite cardiovasculaire qui est avancée : le travail du cœur ne parviendrait progressivement plus à subvenir aux perfusions parallèles musculaire et cutanée induites par les besoins énergétiques et de refroidissement, respectivement (Périard et *al.*, 2011). Dans le cas d'efforts sous-maximaux (*e.g.,* le marathon), la performance serait davantage soumise au dictat d'une désactivation du système nerveux central, protégeant ainsi l'organisme d'une dérive critique de la température interne en élevant la pénibilité d'effort associée à l'exercice (Nybo & Nielsen, 2001).

Au-delà de cette schématisation, les soubassements explicatifs de la décroissance de performance en chaleur s'étayent plus précisément au regard des interactions entre les réponses cardiovasculaires, métaboliques, perceptives, cognitives, et thermorégulatoires à l'exercice (Nybo et *al.*, 2014). Ces dernières dénotent effectivement des systèmes d'influence en cascade *e.g.,* l'impact d'une plus ample vascularisation de la peau sur les contributions cardiaques puis métaboliques subséquentes. Dans cette complexité, les ajustements de l'organisme à la chaleur apparaissent néanmoins tous raisonner autour d'un dénominateur commun. En particulier, la température cutanée de l'individu pourrait demeurer un paramètre consistant susceptible de limiter la baisse de performance observée (Cuddy et *al.*, 2014 ; Ely et *al.*, 2009). Plus précisément, une réduction de la température cutanée (pour une température centrale donnée) est connue pour augmenter le flux de chaleur entre le centre du corps et la peau, et diminuer ainsi les réponses de perfusion sanguine cutanée et métaboliques associées (Cuddy et *al.*, 2014 ; Sawka et *al.*, 2012). Rapporté au contexte plus large de la performance sportive, l'impact ergogénique d'une diminution de la température de la peau a été démontré par l'intermédiaire d'une plus grande tolérance psychophysiologique à l'exercice physique en chaleur (*e.g.,* Byrne et *al.*, 2006 ; Cheuvront et *al.*, 2010 ; Sawka et *al.*, 2012).



L'ensemble de ces éléments invoque le caractère indispensable de préparer les athlètes en endurance à l'exercice en chaleur – comme à des ambiances thermiques moins chaudes (*e.g.,* ~20°C) – notamment en améliorant la capacité de dissipation de chaleur de l'organisme. Alors, en cohérence avec l'approche à moyen terme de la compétition énoncée plus haut, cette préparation requiert d'être programmée relativement aux contraintes effectivement rencontrées par les athlètes en phase précompétitive (logistiques, financières, de temps, *etc.*). Dans cette logique, si possible, les problématiques d'acclimatation à la chaleur sont à anticiper en concomitance à celles de gestion de charges d'entraînement. À l'inverse, dans l'optique d'un manque de moyens/temps à allouer à des expositions répétées à la chaleur, des interventions ponctuelles, écologiques et propices au rafraichissement du corps le jour de l'épreuve sont à envisager. L'optimisation et l'interaction de ces deux types de stratégie (chronique et aigüe) de lutte contre les effets de la chaleur ont constitué certains des points d'intérêt de nos travaux.

Cette toile de fond théorique nous amène à envisager le phénomène de fatigue selon un double versant psycho-physiologique, et sous des angles de développement à court- et moyen-terme. De plus, l'éventualité d'un stress thermique en phase compétitive à Rio invite à étendre cette approche chronologique à des ambiances thermiques chaudes *i.e.,* à l'état ponctuel d'hyperthermie et aux expositions répétées à la chaleur. Ensemble, les travaux liés à ce type d'approche pourrait participer à compléter le fonctionnement du Flush Model de Millet (2011).

3. Enjeux de la thèse

La performance sportive en endurance à haut niveau porte en elle une signature, celle d'une gestion programmée, millimétrée, des phases précompétitive et compétitive. Et comme un amplificateur, le phénomène de fatigue permet de rendre explicite les diverses composantes de cette signature *i.e.,* directes et indirectes (*e.g.,* le rôle de la température cutanée), importantes et minorées (*e.g.,* la capacité à thermoréguler en ambiance chaude *vs.* fraîches), immédiates et durables (*e.g.,* les charges d'entraînement précompétitives). Spécifiquement aux Jeux Olympiques de Rio 2016, nous avons appréhendé ce phénomène avec un regard fonctionnel, en tentant de le comprendre, et en tentant d'y remédier, au travers des questions suivantes :

- La familiarisation à la chaleur constitue-t-elle une stratégie ergogénique pour un athlète d'endurance ne disposant ni de temps (*i.e.,* acclimatation à la chaleur) ni de moyens (*i.e.,* stratégies de refroidissement) dans le cadre de sa préparation à une compétition en ambiance chaude ?

Cette question répondra à une problématique réelle de terrain permettant de quantifier et qualifier l'effet d'une stratégie économique (logistiquement, financièrement, et en temps) pour la préparation à une épreuve en ambiance chaude.

- Les bénéfices sur la performance en environnement chaud, liés aux stratégies de refroidissement demeurent-ils toujours effectifs chez un athlète acclimaté à la chaleur ?

Cette question permettra de définir si les mécanismes affectés par le refroidissement post-acclimatation sont les mêmes que ceux impactés pré-acclimatation, et de quantifier leur potentiel effet ergogénique.

- Les athlètes d'endurance ont-ils intérêt à réaliser des sessions d'entraînement à haute intensité en conditions chaudes dans le cadre d'une phase d'acclimatation précompétitive afin de maximiser les adaptations positives d'entraînement ?

Cette question autorisera à élucider une hypothèse de la littérature fondée sur la spécificité physiologique des athlètes entraînés en endurance (*i.e.,* partiellement

acclimatés), de même qu'à déterminer l'intérêt d'une période d'affûtage post-acclimatation programmée dans l'optique de simuler des conditions écologiques.

- Quelles sont les relations entretenues entre l'état d'hyperthermie de l'organisme et la performance cognitive ? Existe-t-il des moyens d'adresser le déclin du fonctionnement cognitif induit par l'élévation de la température interne ?

Ces questions viseront à compléter la cartographie connue des effets physiologiques de l'état d'hyperthermie par une composante cognitive (moins appréhendée, et peu modélisée), afin de déboucher sur des recommandations utiles pour le maintien de la lucidité de l'athlète.

- Quelle(s) est(sont) la(les) réponse(s) du fonctionnement exécutif à l'exercice d'endurance. Comment cette(ces) réponse(s) peu(ven(t)-elle(s) être expliquée(s) ?

Cette question aura vocation à élucider l'inconsistance de résultats recensée au sein de la littérature, pour tenter d'apporter un éclairage et proposer un modèle explicatif de l'ensemble de ces observations.

- Quels sont les soubassements neuronaux associés aux altérations de la performance cognitive chez des athlètes conduits à un état de surmenage fonctionnel ?

Cette question aura vocation à compléter la cartographie métabolique identifiée de l'état de surmenage, par ses implications nerveuses centrales et ses incidences comportementales.

Nous avons tenté de répondre à ces questions en nous inspirant des caractéristiques du Flush Model de Millet (2011). Ainsi, plus loin dans notre Discussion générale, comprendre et remédier au phénomène de fatigue s'est entendu comme…

(Approche aigue de la fatigue)

i) …réguler la vitesse de remplissage de la cuvette,

en améliorant la stratégie d'allure dans une épreuve en endurance librement gérée en conditions chaudes, par une familiarisation à ce type d'environnement.

Schmit, C., Duffield, R., Hausswirth, C., Coutts, A. J., & Le Meur, Y. (2015). Pacing adjustments associated with familiarisation: Heat vs. temperate environments. *International journal of sports physiology and performance*.

ii) …réguler le niveau d'eau initial de la cuvette,

en réduisant dès l'échauffement les contraintes liées à la chaleur, par l'utilisation d'un gilet réfrigérant de type CryoVest®.

Schmit, C., Le Meur, Y., Duffield, R., Robach, P., Oussedik, N., Coutts, A. J., & Hausswirth, C. (2015). Heat-acclimatization and pre-cooling: a further boost for endurance performance?. *Scandinavian journal of medicine & science in sports*.

iii) …repousser les limites de la réserve,

en approfondissant la réponse cognitive à l'exercice physique, en ambiances tempérées et chaudes, pour envisager des moyens possibles d'influer sur sa dynamique.

Schmit, C., Davranche, K., Easthope, C. S., Colson, S. S., Brisswalter, J., & Radel, R. (2015). Pushing to the limits: the dynamics of cognitive control during exhausting exercise. *Neuropsychologia*, *68*, 71-81.

Schmit, C., & Brisswalter, J. Executive functioning during prolonged exercise: A fatigue based-neurocognitive perspective. *Under review* (in *International Review of Sport and Exercise Psychology*).

Schmit, C., Hausswirth, C., Le Meur, Y., & Duffield, R. (2016). Cognitive functioning and heat strain: performance responses and protective strategie. *Sports Medicine*.

(Approche chronique de la fatigue)

iv) …réguler la vitesse de remplissage de la cuvette,

en améliorant la tolérance de l'organisme aux contraintes de l'exercice en endurance (en ambiance tempérée ou chaude) par une acclimatisation à la chaleur.

Schmit, C., Le Meur, Y., Duffield, R., Robach, P., Oussedik, N., Coutts, A. J., & Hausswirth, C. (2015). Heat-acclimatization and pre-cooling: a further boost for endurance performance?. *Scandinavian journal of medicine & science in sports*.

v) …repousser les limites de la réserve, et réguler le niveau d'eau initial de la cuvette,

en manipulant la charge d'entraînement en période précompétitive, pour des épreuves en ambiance tempérée ou en chaleur

Schmit, C., Blain, B., Hausswirth, C., Pessiglione, M., & Le Meur, Y. Neural correlates of functional overreaching in endurance athletes.

Schmit, C., Duffield, R., Hausswirth, C., Brisswalter, J., & Le Meur, Y. Optimizing heat-acclimation for performance peaking: high- vs low-intensity training. *Submitted* (in *International journal of sports physiology and performance*)

TRAVAIL DE THÈSE

Schmit, C., Duffield, R., Hausswirth, C., Coutts, A. J., & Le Meur, Y. (2015). Pacing adjustments associated with familiarisation: Heat vs. temperate environments. *International journal of sports physiology and performance*.

Résumé en français

Cette étude a eu pour objectif de décrire l'effet de l'expérience perceptive initiale de la familiarisation à la chaleur sur le profil d'allure d'un contre-la-montre (CLM) librement géré, comparativement à des conditions tempérées. Deux groupes de triathlètes bien-entraînés ont réalisés deux CLM soit en conditions chaudes (35°C et 50% d'humidité, n=12), soit en conditions tempérées (21°C et 50% d'humidité, n=22). Un entraînement standardisé était effectué pour chaque groupe avant chaque CLM. Afin de n'induire aucune acclimatation physiologique entre les deux CLM, les épreuves ont été séparées de 11 ± 4 jours. L'amélioration de performance entre les CLM en chaleur (11 ± 24W) est apparue comparable à celle en environnement tempéré (8 ± 14W, p = .67). Cependant, la redéfinition spécifique du pacing en chaleur a été sensiblement différente de la condition tempérée, avec un changement d'une stratégie d'allure initialement 'positive' à 'stable'. En conclusion, l'altération de la perception en chaleur, davantage que l'acclimatation physiologique en soi, pourrait expliquer les changements initiaux du pacing et de la performance en chaleur. Ces résultats mettent en lumière la nécessité pour les athlètes ne possédant pas un temps suffisant pour s'acclimater à la chaleur de s'y familiariser, afin de réduire l'incertitude des éléments susceptibles d'impacter la performance.

Ce travail de thèse a permis de caractériser l'effet et l'utilité de l'expérience à la chaleur sur la performance, comme un moyen simple mais efficace d'anticipation de l'état d'hyperthermie. Les futurs travaux tendront à compléter cette intervention d'autres stratégies ergogéniques pour l'exercice en chaleur.

International Journal of Sports Physiology and Performance, 2016, 11, 855-860
http://dx.doi.org/10.1123/ijspp.2015-0572

Human Kinetics
ORIGINAL INVESTIGATION

Pacing Adjustments Associated With Familiarization: Heat Versus Temperate Environments

Cyril Schmit, Rob Duffield, Christophe Hausswirth, Aaron J. Coutts, and Yann Le Meur

Purpose: To describe the effect of the initial perceptual experience from heat familiarization on the pacing profile during a free-paced endurance time trial (TT) compared with temperate conditions. ***Methods:*** Two groups of well-trained triathletes performed two 20-km TTs in either hot (35°C and 50% relative humidity [RH], n = 12) or temperate (21°C and 50% RH, n = 22) conditions, after standardization of training for each group before both trials. To ensure no physiological acclimation differences between conditions, the TTs for both groups were separated by 11 ± 4 d. ***Results:*** Performance improvement in the heat (11 ± 24 W) from the 1st to 2nd trial appeared comparable to that in temperate conditions (8 ± 14 W, $P = .67$). However, the specific alteration in pacing profile in the heat was markedly different than temperate conditions, with a change from "positive" to an "even" pacing strategy. ***Conclusions:*** Altered perceptions of heat during heat familiarization, rather than physiological acclimatization per se, may mediate initial changes in pacing and TT performance in the heat. These results highlight the need for athletes without time for sufficient heat acclimatization to familiarize themselves with hot conditions to reduce the uncertainty from behavior-based outcomes that may impede performance.

Keywords: hot environments, pacing strategy, cycling, time trial

Endurance performance is reduced in the heat.[1] Compared with time trials (TTs) in temperate conditions, reductions in self-paced cycling endurance performance have been reported to be ~2%,[2] ~7%,[3] and ~16%[4] for short, medium, and long events, respectively. This impairment typically manifests through a progressive down-regulation of intensity (ie, pacing), resulting in a redistribution of work in a manner that allows athletes to complete the required work in the context of the accumulating heat strain.[5] Whereas traditional explanations for these performance reductions in the heat typically focus on physiological adjustments, contemporary models also emphasize the importance of behavioral adjustments that account for the athlete's cognitive interpretation of the environment, thermal state, or perceived effort.[6]

Heat acclimatization (HA)—that is, undertaking repeated exercise bouts in hot environments—is commonly used to prepare athletes for endurance competitions in hot conditions.[7] Indeed, ~2 weeks of HA in well-trained cyclists has been demonstrated to offset the heat-related reductions in performance, mainly through reestablishing the pacing profile adopted to a level comparable to cool conditions.[4] Although it is well established that ≥14 days of heat exposure is required to induce complete HA, shorter exposure periods (eg, intermittent HA) may provide partial acclimatization responses.[8] Indeed, it has been proposed that the adjustments in cardiovascular, metabolic, and thermoregulatory functions mediate HA according to a dose-response relationship.[9] However, despite this knowledge, the relationship between the amount of heat exposure and the physiological and performance outcomes at the level of the individual athlete remains unclear.[10] For example, Keiser et al[10] reported that the highly individual physiological responses to a training camp did not correlate to individual performance outcomes. These observations suggest that mechanisms other than physiological adaptations, such as perceptual adaptations, may also contribute to the improved endurance performance commonly observed with heat exposure.

It was recently demonstrated that the alliesthesial variations of skin temperature in response to heat stress were sufficient to alter the subjective state of the individual and the subsequent ability to self-regulate behavior.[11] However, of the studies that investigate HA-related effects on self-paced endurance performance, few have examined the specific role of perception in behavioral adaptations.[4,12,13] In these studies, the initial testing bouts performed in the heat were not preceded by familiarization in thermally stressful environments. The importance of familiarization in research studies is well established and is essential for minimizing the learning effects on outcome measures. Therefore, it is possible that the perceptual familiarization to exercise in hot environments may influence exercise behavior and performance, independent of the common physiological responses to HA. At present, however, although the role of previous experience in behavioral self-regulation during exercise is often proposed to factor in acute performance improvement in the heat,[14] no studies have yet examined the importance of this factor independent of physiological responses. However, athletes without time for sufficient HA may benefit from such experience to better apprehend the specificity of heat stress during the competition.

Within this framework, the aim of this study was to determine the effect of the initial heat familiarization as related to perceptual experience on the pacing profile during an unfamiliar free-paced endurance TT as compared with in temperate conditions. We hypothesized that well-trained non-HA cyclists would redistribute power output during a 20-km cycling TT performed in the heat after an initial experience in this context, whereas a similar population performing in temperate conditions would not alter the pacing profile.

Schmit, Hausswirth, and Le Meur are with the Laboratory of Sport, Expertise and Performance (EA 7370), French National Inst of Sport, Expertise and Performance (INSEP), Paris, France. Duffield and Coutts are with the Sport & Exercise Discipline Group, University of Technology Sydney, Sydney, Australia. Address author correspondence to Cyril Schmit at cyril.schmit@gmail.com.

Methods

Two data sets from previous studies were used for the present work. In 1 study, 22 male triathletes performed repeated 20-km TTs in temperate conditions (temperate group), whereas in the other study, 12 male triathletes performed repeated 20-km TTs in the heat (heat group). Apart from the environmental conditions, there were no methodological differences between the 2 protocols, and as such we here describe a single experimental design.

Subjects

The characteristics of the 2 groups are presented in Table 1. All subjects had at least 3 years of prior competitive experience, were training a minimum of 7 sessions/wk, had no HA in the previous 5 months (commencement of both studies in February in Paris, France), and were not familiar with the specific 20-km TT. Before inclusion in the study, participants were medically examined by a cardiologist to ensure normal electrocardiograph patterns and obtained general medical clearance. All respective data collection was performed in accordance with the Helsinki Declaration. After comprehensive verbal and written explanations of the study, all subjects gave their written informed consent for participation in respective studies. The authors report no conflict of interest to subjects.

Experimental Design

All athletes first performed a graded exercise test in thermoneutral conditions (21°C, 50% relative humidity [RH]) using an electronically braked cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands). The ergometer was equipped with standard 170 mm cranks and the athletes' own shoes. The positions of the handlebars and seat height were adjusted to align with those used by the athletes on their own bikes. The test was performed until complete exhaustion to determine maximal oxygen uptake and maximal aerobic power (MAP) (Table 1). The exercise protocol started with a 5-minute warm-up at a workload of 100 W and then increased by 20 W/min until voluntary exhaustion. Subjects wore a facemask covering their mouth and nose to collect all expired breath (Hans Rudolph, Kansas City, MO). Oxygen and carbon dioxide concentrations in the exhaled gas were continuously measured and monitored on a breath-by-breath basis (Quark, Cosmed, Rome, Italy). The gas analyzer and the flow meter of the spirometer used were calibrated before each test.

During the second and the third sessions, participants in the temperate group performed a 20-km TT at 21°C, 50% RH, whereas subjects in the heat group performed the 20-km TT in a climate chamber at 35°C, 50% RH (Thermo Training Room, Paris, France). There were 11 ± 4 days between each TT for each group.

Table 1 Participant Characteristics, Mean ± SD

Variable	Temperate	Heat
Age (y)	30.7 ± 4.4	31.6 ± 5.6
Height (cm)	178.8 ± 6.6	179.6 ± 5.4
Body mass (kg)	69 ± 7	72.7 ± 5.6
$\dot{V}O_2max$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	63.3 ± 2.1	62.2 ± 3.6
Maximal aerobic power (W)	378 ± 45	390 ± 38

Abbreviation: $\dot{V}O_2max$, maximal oxygen uptake.

To ensure that performance variations during the TTs were due to experimental procedures and not to the previous training load, subjects were required to respect a 24-hour rest period before each laboratory session. Sessions were scheduled at the same hour of the day.

To assure hydration status at the beginning of each session, participants were instructed to standardize the fluid consumed based on the absorption of 1 L of water distributed throughout the last 2 hours before the visit. At the commencement of each session, participants completed a questionnaire assessing perceived fatigue, motivation, and delayed onset muscle soreness (DOMS) as based on 5-point Likert scales and were instructed to complete the TT as fast as possible. Then, following a 10-minute seated period, a 15-minute warm-up was completed, including 10 minutes of cycling at 100 W and 5 minutes at 50% of the individual's MAP. Each participant performed both the warm-up and the TT on their own bike mounted on a braked Cyclus2 ergometer (RBM GmbH, Leipzig, Germany). During the TT, convective airflow from a fan set to a standard speed (750 mm, 1450 ± 5 rpm) facing the participant was used to mimic field conditions. To control for fluid intake between sessions, the participants were instructed during the second session that they could drink ad libitum during the passive phase, warm-up, and TT, with the volume of water ingested measured and then replicated during the ensuing TT.

The main measurements performed during the TT protocol were the time required to complete 20 km and the power output (PO) recorded by the Cyclus2 software at a sampling rate of 2 Hz. No feedback was provided to the subjects during TTs except for the distance remaining. The participants were not informed of their performance until the end of the study. PO values obtained for each TT were reported per kilometer of the TT and used to show the pacing strategy.

Training Load Monitoring

Participants continuously recorded their usual training program during the 2 experiments. For 3 weeks before the first visit, they were equipped for each training session with a global positioning system monitor (Garmin Forerunner 305 GPS, Garmin International, Inc, Kansas City, MO, USA) to measure training distance and speed. Details about the training duration, intensity, mode, and periodization of the typical training week were recorded (Table 2). To ensure that the training patterns applied before the 2 experimental sessions were similar, this training program was replicated in the 7 days preceding each test.

Data Analysis

Repeated-measures analyses of variance (ANOVAs) were performed on PO values with group (heat vs temperate, between-subjects), session (first vs second, within-subject), and kilometer (×20, within-subject) as factors. To estimate relative changes in intensity from the PO at TT onset, the intensity at each kilometer was reported relative to the starting intensity (which was set as 100%) and used as a within-subject factor. For the psychometric and training data, the factor session was used as a within-subject factor. TT durations were compared using independent- and paired-samples *t* tests for between- and within-group differences, respectively. All data were analyzed using SPSS software (IBM SPSS Statistics 20, IBM Corp, Armonk, NY). Planned comparisons were used in the general linear model for post hoc analyses when differences were significant ($P < .05$). Effect sizes are described in terms of partial eta-squared (η_p^2,

Table 2 Data From Training Monitoring and Performance Testing, Mean ± SD

		Temperate		Heat	
		Session 1	Session 2	Session 1	Session 2
Training data	Delayed-onset muscle sorness (Likert)	2.4 ± 0.9	2.2 ± 1	2.3 ± 0.8	2.5 ± 0.7
	Fatigue (Likert)	1.7 ± 0.5	1.8 ± 0.4	1.8 ± 0.6	1.8 ± 0.7
	Motivation (Likert)	4.2 ± 0.4	4.1 ± 0.6	4.1 ± 1	4.0 ± 0.9
	Power output (W)	247 ± 42	255 ± 40*	223 ± 20	234 ± 11*
	Time (min/s)	32.16 ± 2.01	31.52 ± 1.37*	33.22 ± 1.58	32.40 ± 1.23*
Testing data	Training volume (min)	812 ± 119	818 ± 87	812 ± 152	831 ± 148
	Distance (km)				
	cycling	259 ± 75	265 ± 73	271 ± 59	271 ± 64
	running	34 ± 18	32 ± 16	35 ± 19	32 ± 15
	swimming	8 ± 3	8 ± 3	8 ± 3	8 ± 2
	Frequency				
	cycling	5 ± 1	5 ± 2	5 ± 2	5 ± 2
	running	3 ± 1	3 ± 1	3 ± 1	3 ± 1
	swimming	2 ± 1	2 ± 1	2 ± 1	2 ± 1

Note: Likert is extracted from Likert scales.

*Significantly ($P < .05$) different from the first session in the heat.

with $\eta_p^2 \geq 0.06$ representing moderate difference and $\eta_p^2 \geq 0.14$, large difference). Values are presented as means ± SD.

Results

Training Loads and Perceived State

No effect was observed between groups for weekly training measures volume, distance, or frequency ($P > .10$) (Table 2). Further, no differences were evident for DOMS, fatigue, and motivation levels before TTs ($P > .10$).

PO and Pacing

A session effect ($P = .01$, $\eta_p^2 = 0.16$) showed that both the temperate and the heat groups improved their performance after the first session by 8 ± 14 W and 11 ± 24 W, respectively (Table 2), albeit without a group × session interaction ($P = .67$). However, a group × session × kilometer interaction ($P < .001$, $\eta_p^2 = 0.10$) revealed that in comparison with the first session, the heat group started the second TT at a lower intensity and performed the majority of the second TT bout at a higher PO ($P < .05$; Figure 1[B]). A group × session × kilometer × starting intensity interaction was also observed ($P < .001$, $\eta_p^2 = 0.08$), although there were no differences between sessions for the temperate group relative to the starting intensity (Figure 2[A], $P = .89$), with only the sprint finish differing from the starting intensity (Figure 2[A], $P = .03$). Conversely, in the heat group PO was reduced during the first session by 21% ± 19% of the initial PO (15–18 km, $P < .001$, $\eta_p^2 = 0.57$, Figure 2[B]). In turn, during the second session, the Heat group demonstrated temporary increases in PO during the TT relative to the starting intensity (Figure 2[B], $P < .05$).

Discussion

The aim of this study was to determine the effect of the initial heat familiarization as related to perceptual experience on the pacing profile during an unfamiliar free-paced endurance TT as compared with temperate conditions. Although the improvement in performance ($\eta_p^2 = 0.18$) appears comparable to those occurring in temperate conditions ($\eta_p^2 = 0.24$, large size effects), the specific changes in absolute and relative pacing profiles in the heat highlight an important role for heat familiarization. These findings suggest a specific "immediate" behavioral adaptation evident in the heat to allow improved endurance performance before any likely physiological acclimatization.

The use of a familiarization trial in research is important to reduce the influence of a repeated-bout effect (eg, learning) biasing the interpretation of the results. Given the standardization of training, such an outcome is likely observed in the 3.3% ± 1.2% improvement in temperate conditions, representing the TT variability due to task knowledge.[15] It was notable that despite the increased PO in the second TT, an almost identical even pacing profile existed between the initial and repeated trials in temperate conditions.[16] Similarly, the heat group also improved TT performance like the temperate group from the first to the second trial (5.6% ± 6.6%). However, in contrast to the temperate group, subjects initially exposed to 35°C specifically rearranged PO distribution during the second trial in the heat to prevent the ~20% reduction in PO relative to the starting intensity. Of interest, this reduction in PO during the TT in the heat is comparable to other recent evidence of similarly trained athletes and TTs.[4] However, between the 2 sessions, the heat group shifted from a positive pacing strategy to a less aggressive, more even pattern[15] characterized by a lower starting intensity

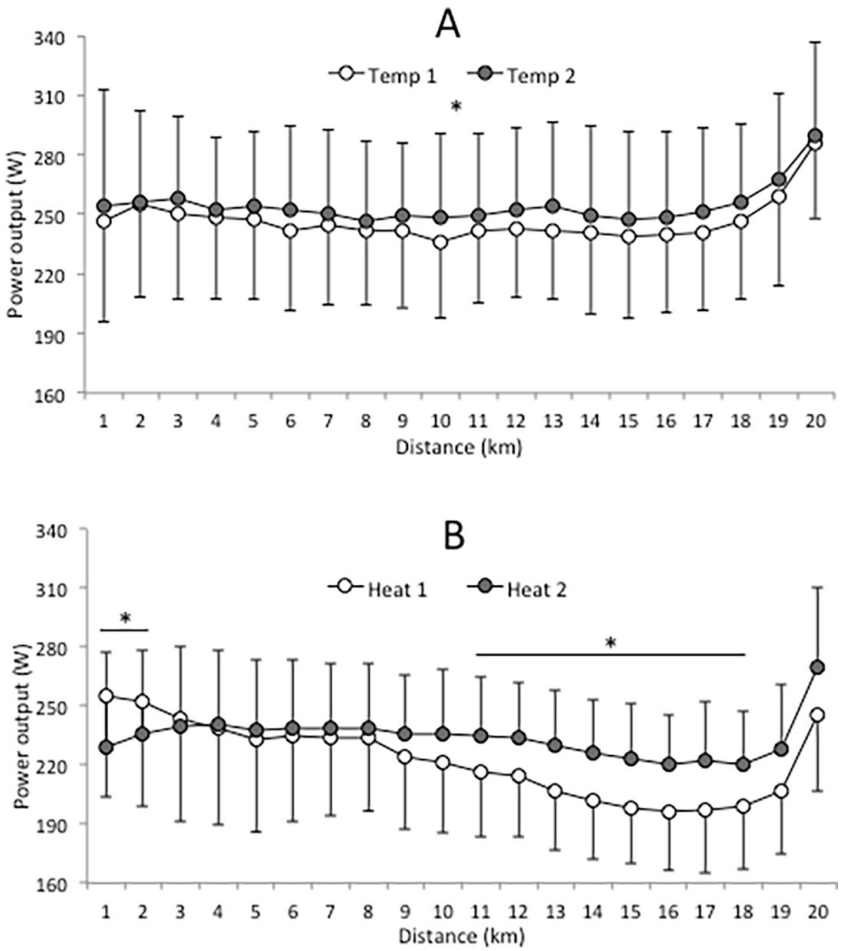


Figure 1 — Absolute changes in power output per kilometer from the first to the second trial in (A) temperate and in (B) hot conditions. Results are presented as the group mean ± SD. *Significant session effect ($P < .05$). Abbreviation: Temp, temperate.

(–26 ± 36 W, Figure 1[B]) and a steadier PO throughout the rest of the exercise bout (Figure 2[B]). Such reduction of the starting intensity has previously been noticed during repeated 20-km TTs in temperate conditions, although admittedly over more trials.[17] In addition, such shifts toward an equilibrate pattern of exercise intensity has previously been reported,[4] although this was from consecutive trials before and following a 2-week training camp in the heat. In part it is feasible that the initial trial was driven by a greater experience with temperate as opposed to the hot conditions. Hence, regardless of minimal familiarity with this explicit TT, a greater familiarity with the conditions may have existed. Regardless, the altered pacing strategies and performance improvement in the heat observed in the current study (likely without HA) would suggest that heat familiarization-based behavioral regulation assists partially compensate for the reduction in performance due to the environmental stress.

Accepted mechanisms as to why endurance performance decrement in the heat can be minimized following HA relate to physiological acclimatization, as driven by cardiovascular, thermoregulatory, and metabolic adaptations.[18,19] Complete HA has been reported to occur within 14 days of repeated exposure, though it has been shown that as little as 4 to 5 days can initiate 75% to 80% of HA adaptations.[12,20] Moreover, given that 1-week intervals between heat sessions curtail physiological adaptations,[20] it is likely that the time between the heat TTs in the current study (11 ± 4 d) was sufficient for the decay of any physiological adaptations that may have resulted from the initial TT. Indeed, it has been reported that 1 day of HA is lost for every 2 to 5 days without heat exposure.[21–23] However, we must acknowledge the lack of HA measures as a limitation of this study. Nonetheless, assuming a lack of physiological HA with the ~7 to 15 days separating heat TTs, it is logical that the initial improvement in TT performance and altered pacing strategy are due to changes in perceptions of the heat following the heat familiarization rather than physiological responses.

It is suggested that cognitive factors mainly account for changes in the pacing strategy. Accordingly, it is likely that the experience of the first trial in the heat provided the athletes with better information to anticipate the risks associated with an aggressive start during the second trial.[24] This greater awareness resulted in the adoption of a more even, and by virtue, potentially tolerable, pacing strategy during the second trial in the heat.[25] Such reduction in initial intensity would enable a lowered rate of heat storage, subsequently preventing the precipitated physiological strain expected under these conditions. Regardless, experience is widely reported to be a powerful regulator of energy expenditure[26,27] and may explain why the heat group demonstrated during the second trial an even strategy relative to the starting intensity (Figure 2[B]), while still undertaking a powerful end spurt. The down-regulation of PO during the TT noted in the current study contrasts with Racinais et al[4] and may be explained by the fact that the second TT of

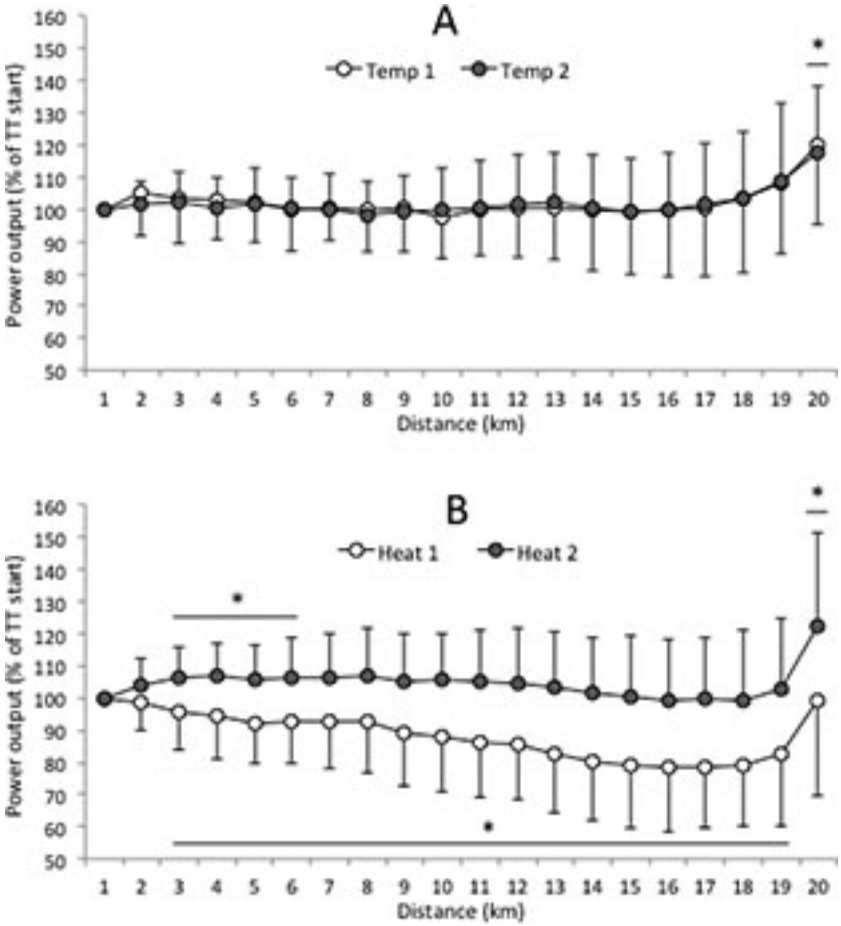


Figure 2 — Relative changes in power output from the first kilometer within the first and the second trial in (A) temperate and in (B) hot conditions. Results are presented as the group mean ± SD. *Significant differences from the first kilometer ($P < .05$). Abbreviations: Temp, temperate; TT, time trial.

their study occurred 6 days after the daily HA commenced. It is, therefore, possible that in this previous study, the adaptations to the repeated heat exposures were concurrent with a familiarization effect and, therefore, obscured any manifestation of heat-related perceptive adaptations on pacing adjustments during the second TT in the heat. Regardless, the current findings highlight the potential benefits of full familiarization with the environmental conditions and even perhaps regardless of achieving full acclimatization status if such time is not permitted.

Although the current study provides new information on the importance of heat familiarization before endurance performance in hot conditions, some limitations need to be recognized in the current findings. Based on previous reports of decay rates of HA, the 11 ± 4 days between the 2 sessions should have been sufficient to allow for the decay of any substantial HA following the first TT in the heat. However, the lack of physiological measures does not allow us to fully dismiss putative cardiovascular or thermoregulatory factors in performance improvement. Such measures would enable us to consider the role of both physiological and psychological changes in performance enhancement during HA. In this perspective, future studies should address to what extent primary changes in performance following an initial exposure to the heat are associated to perceptive (eg, rating of perceived exertion or thermal comfort) and/or cognitive (pacing template) parameters. Indeed, in regard to the highly individual variations in physiological and performance responses following HA,[7,10] the progressive development of mechanisms driving performance improvement in the heat remains to be elucidated. It must also be acknowledged that the even pacing pattern observed in the temperate group since the first TT may partly result from the knowledge of this specific environmental condition (ie, 21°C). Previous experience may, therefore, have constituted a more substantial basis than for the heat group; although this may be an important implication to prepare athletes who compete in unfamiliar hot conditions.

Practical Implications and Conclusions

This investigation highlights the role of heat familiarization during free-paced endurance TTs in the heat compared with temperate conditions. We observed changes in pacing profile following a single TT in the heat, which did not occur in temperate conditions, suggesting that heat-related improvements may occur independently to physiological changes (especially since there were 11 ± 4 d separating the trials). Although mechanistic explanation might relate to perceptual adaptations and prior experience, further research should continue to determine the independent contributions of perceptual versus physiological adaptations for performance improvement in the heat as part of the HA process. In this perspective, our results highlight the need for athletes without time for sufficient HA to undertake efforts to ensure familiarity with the conditions and

reduce the uncertainty from behavior-based outcomes that may impede performance.

Acknowledgments

The results of the current study do not constitute endorsement of the product by the authors of the journal. C.S. and R.D. contributed equally to the present work.

References

1. Guy JH, Deakin GB, Edwards AM, Miller CM, Pyne DB. Adaptation to hot environmental conditions: an exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. *Sports Med*. 2015;45(3):303–311. doi:10.1007/s40279-014-0277-4
2. Altareki N, Drust B, Atkinson G, Cable T, Gregson W. Effects of environmental heat stress (35 degrees C) with simulated air movement on the thermoregulatory responses during a 4km cycling time-trial. *Int J Sports Med*. 2009;30:9–15. PubMed doi:10.1055/s-2008-1038768
3. Tatterson AJ, Hahn AG, Martin DT, Febbraio MA. Effects of heat stress on physiological responses and exercise performance in elite cyclists. *J Sci Med Sport*. 2000;3:186–193. PubMed doi:10.1016/S1440-2440(00)80080-8
4. Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. *Med Sci Sports Exer*. 2015;47:601–606. PubMed doi:10.1249/MSS.0000000000000428
5. Tucker R, Marle T, Lambert EV, et al. The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *J Physiol*. 2006;574:905–915. PubMed doi:10.1113/jphysiol.2005.101733
6. Flouris AD, Schlader ZJ. Human behavioral thermoregulation during exercise in the heat. *Scand J Med Sci Sports*. 2015;25(suppl 1):52–64. PubMed doi:10.1111/sms.12349
7. Racinais S, Buccheit M, Bilsborough J, Bourdon PC, Cordy J, Coutts AJ. Physiological and performance responses to a training-camp in the heat in professional Australian football players. *Int J Sports Physiol Perform*. 2014;9(4):598–603. PubMed doi:10.1123/ijspp.2013-0284
8. Périard JD, Racinais S, Sawka MN. Adaptations and mechanisms of human heat acclimation: applications for competitive athletes and sports. *Scand J Med Sci Sports*. 2015;25(Suppl 1):20–38. PubMed doi:10.1111/sms.12408
9. Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol*. 1993;460(1):467–485. PubMed doi:10.1113/jphysiol.1993.sp019482
10. Keiser S, Flück D, Hüppin F, Stravs A, Hilty MP, Lundby C. Heat training increases exercise capacity in hot but not in temperate conditions: a mechanistic counter-balanced cross-over study. *Am J Physiol Heart Cir Physiol*. 2015;309(5):H750–H761. PubMed doi:10.1152/ajpheart.00138.2015
11. Gaoua N, Grantham J, Racinais S, El Massioui F. Sensory displeasure reduces complex cognitive performance in the heat. *J Environment Physiol*. 2012;32:158–163.
12. Garrett AT, Creasy R, Rehrer NJ, Patterson MJ, Cotter JD. Effectiveness of short-term heat acclimation for highly trained athletes. *Eur J Appl Physiol*. 2012;112:1827–1837. PubMed doi:10.1007/s00421-011-2153-3
13. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. *J Appl Physiol*. 2010;109:1140–1147. PubMed doi:10.1152/japplphysiol.00495.2010
14. Duffield R, Green R, Castle P, Maxwell N. Precooling can prevent the reduction of self-paced exercise intensity in the heat. *Med Sci Sports Exerc*. 2010;42(3):577–584. PubMed doi:10.1249/MSS.0b013e3181b675da
15. Abbiss CR, Laursen PB. Describing and understanding pacing strategies during athletic competition. *Sports Med*. 2008;38(3):239–252. PubMed doi:10.2165/00007256-200838030-00004
16. Abbiss CR, Levin G, McCuigan MR, Laursen PB. Reliability of power output during dynamic cycling. *Int J Sports Med*. 2008;29(7):574–578. PubMed doi:10.1055/s-2007-989263
17. Thomas K, Stone MR, Thompson KG, Gibson ASC, Ansley L. Reproducibility of pacing strategy during simulated 20-km cycling time trials in well-trained cyclists. *Eur J Appl Physiol*. 2012;112(1):223–229. PubMed doi:10.1007/s00421-011-1974-4
18. Nielsen B, Strange S, Christensen NJ, Warberg J, Saltin B. Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflugers Arch*. 1997;434:49–56. PubMed doi:10.1007/s004240050361
19. Shapiro Y, Moran D, Epstein Y. Acclimatization strategies – preparing for exercise in the heat. *Int J Sports Med*. 1998;19:S161–S163. PubMed doi:10.1055/s-2007-971986
20. Barnett A, Maughan RJ. Response of unacclimatized males to repeated weekly bouts of exercise in the heat. *Br J Sports Med*. 1993;27:39–44. PubMed doi:10.1136/bjsm.27.1.39
21. Givoni B, Goldman RF. Predicting rectal temperature response to work, environment, and clothing. *J Appl Physiol*. 1972;32:812–822. PubMed
22. Pandolf KB, Burse RL, Goldman RF. Role of physical fitness in heat acclimatisation, decay and reinduction. *Ergonomics*. 1977;20:399–408. PubMed doi:10.1080/00140137708931642
23. Taylor NAS. Principles and practices of heat adaptation. *J Hum Environ Syst*. 2000;4(1):11–22. doi:10.1618/jhes.4.11
24. Slovic P, Finucane ML, Peters E, MacGregor DG. Risk as analysis and risk as feelings: some thoughts about affect, reason, risk, and rationality. *Risk Anal*. 2004;24(2):311–322. PubMed doi:10.1111/j.0272-4332.2004.00433.x
25. Micklewright D, Parry D, Robinson T, et al. Risk perception influences athletic pacing strategy. *Med Sci Sports Exerc*. 2015;47(5):1026–1037. doi:10.1249/MSS.0000000000000500
26. Foster C, Hendrickson KJ, Peyer K, et al. Pattern of developing the performance template. *Br J Sports Med*. 2009;43(10):765–769. PubMed doi:10.1136/bjsm.2008.054841
27. Micklewright D, Papadopoulou E, Swart J, Noakes T. Previous experience influences pacing during 20-km time trial cycling. *Br J Sports Med*. 2010;44:952–960. PubMed doi:10.1136/bjsm.2009.057315

TRAVAIL DE THÈSE

Schmit, C., Le Meur, Y., Duffield, R., Robach, P., Oussedik, N., Coutts, A. J., & Hausswirth, C. (2015). Heat-acclimatization and pre-cooling: a further boost for endurance performance?. *Scandinavian journal of medicine & science in sports*.

Résumé en français

Afin de déterminer si l'usage bénéfique du pre-cooling (PC) reste ergogénique pour la performance en endurance en chaleur après une phase d'acclimatatisation (HA), 13 triathlètes ont réalisé deux contre-la-montre (CLM) de 20km à 35°C, 50% d'humidité, avant et à la suite d'un camp d'entraînement en chaleur de 8 jours, chaque fois avec et sans PC. Les stratégies d'allure, réponses physiologiques et perceptives ont été évaluées au cours de chaque CLM. L'usage du PC et de HA ont induits des augmentations *modérées* (+10 ± 18W ; ES 4,4 ± 4,6%) et *très larges* (+28 ± 19W ; ES 11,7 ± 4,1%) de la puissance de pédalage, respectivement. L'effet global de PC est resté *peu clair* après HA (+4 ± 14W ; ES 1,4 ± 3,0%). Cependant, les analyses de pacing ont révélé que PC restait transitoirement bénéfique post-HA *i.e.,* au cours de la première partie du CLM. PC et HA ont tous deux été caractérisés par une augmentation de la puissance de pédalage sans perturbations cardio-thermorégulatoires ou perceptives supplémentaires, tandis que PC post-HA induisait une amélioration du confort thermique. L'usage du PC améliore la performance en CLM de 20km chez des sujets non-acclimatés, mais une période HA de 8 jours atténue la magnitude de cet effet. Les incidences physiologiques respectives du PC et de HA peuvent expliquer cette inhibition de la réponse au PC. Cependant, les bénéfices perceptifs du PC pourraient rester présents post-HA et expliquer les légers ajustements de la stratégie d'allure observés.

Cette étude a permis d'apporter un éclairage sur l'intérêt et les mécanismes ergogéniques de la combinaison entre *cooling* et acclimatation pour la performance en endurance en chaleur. Associés avec le travail de thèse précédent, la question de la limite (*i.e.,* un plafonnement) de l'utilité de ce type de stratégie se pose alors.

Scand J Med Sci Sports 2015: ●●: ●●–●●
doi: 10.1111/sms.12629

SCANDINAVIAN JOURNAL OF
MEDICINE & SCIENCE
IN SPORTS

Heat-acclimatization and pre-cooling: a further boost for endurance performance?

C. Schmit[1], Y. Le Meur[1], R. Duffield[2], P. Robach[1,3], N. Oussedik[4], A. J. Coutts[2], C. Hausswirth[1]

[1]French National Institute of Sport, Expertise and Performance (INSEP), Research Department, Laboratory of Sport, Expertise and Performance (EA 7370), Paris, France, [2]Sport & Exercise Discipline Group, Faculty of Health, University of Technology Sydney (UTS), Moore Park, Australia, [3]Medical Department, National School for Mountain Sports, Site of the National School for Skiing and Mountaineering (ENSA), Chamonix, France, [4]Department of Biological Toxicology, University Hospital Lariboisière (AP-HP), Paris, France
Corresponding author: Christophe Hausswirth, French National Institute of Sport, Expertise and Performance, Laboratory of Sport, Expertise and Performance, 11, avenue du Tremblay, 75 012 Paris, France. Tel.: +33 (0)1 41 74 43 85, Fax: +33 1 41 74 45 35, E-mail: christophe.hausswirth@insep.fr

Accepted for publication 30 October 2015

To determine if pre-cooling (PC) following heat-acclimatization (HA) can further improve self-paced endurance performance in the heat, 13 male triathletes performed two 20-km cycling time-trials (TT) at 35 °C, 50% relative humidity, before and after an 8-day training camp, each time with (PC) or without (control) ice vest PC. Pacing strategies, physiological and perceptual responses were assessed during each TT. PC and HA induced *moderate* (+10 ± 18 W; effect size [ES] 4.4 ± 4.6%) and *very large* (+28 ± 19 W; ES 11.7 ± 4.1%) increases in power output (PO), respectively. The overall PC effect became *unclear* after HA (+4 ± 14 W; ES 1.4 ± 3.0%). However, pacing analysis revealed that PC remained transiently beneficial post-HA, i.e., during the first half of the TT. Both HA and PC pre-HA were characterized by an enhanced PO without increased cardio-thermoregulatory or perceptual disturbances, while post-HA PC only improved thermal comfort. PC improved 20-km TT performance in unacclimatized athletes, but an 8-day HA period attenuated the magnitude of this effect. The respective converging physiological responses to HA and PC may explain the blunting of PC effectiveness. However, perceptual benefits from PC can still account for the small alterations to pacing noted post-HA.

Self-paced endurance exercise is reported to be compromised in the heat, with important implications for competitive endurance events (e.g., World Triathlon Series, Athletics Championships). As evidence, the −0.3% to −0.9% performance decrement per 1 °C increase in ambient temperature above 10 °C is well noted (Racinais et al., 2015) and these negative effects on performance are increased with greater exercise duration (~2% for ~6.5 min, Altareki et al., 2009; ~7% for 30 min, Tatterson et al., 2000; ~16% for ~70 min, Racinais et al., 2015). To improve both performance and athlete health, various strategies have been developed to cope with exercise in the heat (Coris et al., 2004). In particular, heat-acclimatization (HA) and pre-cooling (PC) procedures have both been shown to increase work capacity in the heat (e.g., Garrett et al., 2011; Ross et al., 2013). However, it is currently unknown if when combined, these strategies provide additional physiological benefits or ergogenic effects on endurance performance.

Medium-term heat-acclimation, i.e., 7–14 training days in the heat, improves endurance performance at high ambient temperatures for both fixed- (e.g., Nielsen et al., 1993) and self-paced (e.g., Lorenzo et al., 2010; Racinais et al., 2015) cycling. Systemic protective adaptations against heat stress have been suggested to underpin these benefits. For example, decreases in sweat electrolyte (e.g., sodium), and increases in plasma volume (PV) and sweat rate have been reported to provide cardiovascular and thermoregulatory benefits (Nielsen et al., 1993). In addition to these physiological adaptations, lower perception of effort and reduced feelings of heat stress have also been reported (Daanen et al., 2011), although these have received less attention as to their ergogenic benefits. Given the role of passive exposure (i.e., free living) in addition to training in the heat during HA to maximize these adaptations (Shido et al., 1999), there has been increased interest in the efficacy of using heat training camps to prepare athlete for competition (e.g., Racinais et al., 2013, 2015).

Cooling the body prior to exercise in the heat has been shown to protect athletes from the negative effects of the heat through delaying the rise in endogenous temperature and limiting the decrement in endurance performance (Bongers et al., 2015). Reduced skin temperature (Tskin) following PC has been proposed an important factor determining aerobic performance during submaximal exercise in the heat, via improved core-to-skin temperature gradients and thermal comfort (TC) (Cuddy et al., 2014). A greater core-to-skin gradient is rationalized to be ergogenic by improving heat transfer from the core to the periphery, and heart rate by reducing skin perfusion (Cuddy et al., 2014). Given their low level of invasiveness on athletes preparation or competition routines, and avoidance of cooling active musculature, cooling vests have become a popular form of PC. Worn either at rest or during warm-up, ice vests enable improved steady- and self-paced endurance cycling tests in the heat (Johnson et al., 2008; Bogerd et al., 2010). In part, this improvement may occur as a result of augmented perception of TC (Schlader et al., 2011), though high variability between athletes in perceived thermal comfort may subsequently influence the magnitude of performance changes.

It is presently unknown if there is additional benefit of combining PC following HA to performance in hot conditions for endurance athletes. Indeed, it is possible that the HA-related thermoregulatory adjustments (e.g., lower increase in core temperature [Tcore] for sweat onset) could be further improved during exercise by the addition of a favorable core-to-skin gradient achieved with PC. The improved metabolic efficiency achieved with HA (Febbraio et al., 1994) might also add to the lowered energy expenditure reported with PC. In contrast, the PV expansion following HA may not allow PC to provide the same magnitude of benefit to cardiovascular function (i.e., preserved blood volume) to exercise in the heat that has been reported in unacclimatized athletes (Quod et al., 2008), and this may reduce the expected performance effects. Similarly, it is presently unknown whether perceptual improvements from HA interact with the PC-related increased TC of unacclimatized athletes.

To date, few studies have combined these strategies. Recently, after a 10-day heat-acclimation period, Castle et al. (2011) reported no further ergogenic effect of thigh PC in moderately trained subjects during an intermittent-sprint exercise protocol in the heat. However, this type of performance may be impaired by muscle PC due to loss in metabolic and neuromuscular efficiency (e.g., Sleivert et al., 2001), and thus not adequately reflect the HA–PC interaction for endurance performance. Brade et al. (2013) reported the same absence of cumulative effect in moderately trained participants using ice-slushie and cooling jackets after a five-session heat-acclimation exposure. However, in this study, no effect of PC on performance was reported pre-acclimation, making interpretation of the PC–HA interaction difficult. Accordingly, the present study investigated the effects of PC prior to, and following a period of HA on self-paced endurance performance in the heat. At the end of the training camp, we hypothesized ice vest PC would absorb excess body heat and increase TC prior to exercise, and HA would further reduce heat storage during exercise, resulting in greater performance improvement than each method separately.

Methods

Participants

Thirteen well-trained national-level males triathletes (age 31 ± 4 years, height 179.5 ± 4 cm, body mass 71.7 ± 5.6 kg, maximal oxygen consumption [VO_{2max}] 64.9 ± 6.9 mL/kg/min, maximal power output [MPO] 406 ± 34 W) volunteered to participate in this study. All subjects had competed at the national level for at least 3 years and trained ≥7 sessions/week. Prior to inclusion in the study, participants were examined by a cardiologist to obtain a medical clearance. The experimental design of the study was approved by the Ethics Committee of *Hôtel Dieu*, Paris (acceptance no. 2013-A00824-41) and the protocol was performed in accordance with the Declaration of Helsinki. After comprehensive verbal and written explanations of the study, all subjects gave their written informed consent for participation.

Experimental design

An outline of the full protocol is presented in Fig. 1. All participants performed repeated testing with and without PC in a randomized fashion, before and after a period of HA. Specifically, at the commencement of the study, all athletes resided in Paris (France) and had no heat exposure in the previous 6 months. Each participant attended five pre- and three post-HA sessions (all at the same time of day), either at INSEP (Paris, France) or at the Centre of Sports Resources, Expertise and Performance (CREPS) of Pointe-à-Pitre (Guadeloupe). Performance testing was conducted on two consecutive days before traveling (7 h) to Pointe-à-Pitre (departure at 10:00, west direction) or to Paris (departure at 17:00, east direction). Furthermore, on the day prior to departure for Guadeloupe and on the day of return to Paris, PV was determined and a heat tolerance test (HTT) was completed. At both testing locations, the research equipment and procedures were standardized and overseen by the same research team. To note, as pre-HA sessions were performed in a climate chamber, at least 7 days were allowed between the familiarization and the experimental sessions to avoid initiating heat adaptations prior to the training camp.

During the first pre-HA session, a graded exercise test was performed in normothermic conditions (21 °C, 40% RH) using an electronically braked cycle ergometer (Excalibur Sport, Lode®, Groningen, The Netherlands). Subjects wore a facemask covering their mouth and nose to collect all expired breath (Hans Rudolph, Kansas City, Missouri, USA). The exercise protocol started with a 5-min warm-up at a workload of 100 W, and then increased by 20 W/min until voluntary

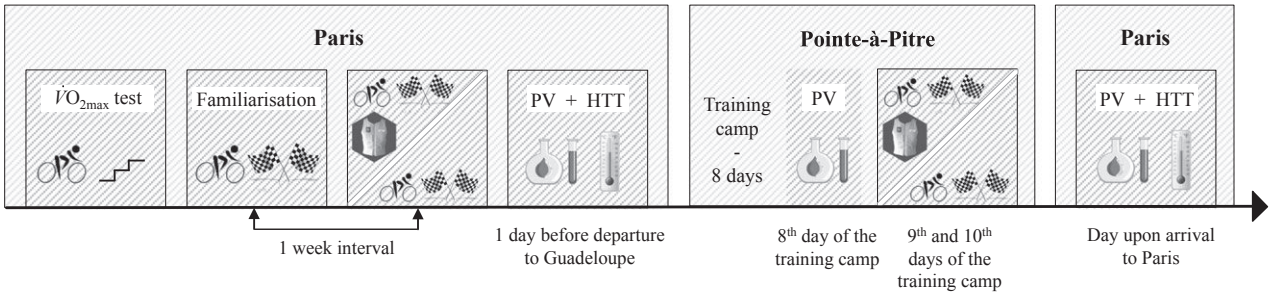


Fig. 1. Schematic representation of the full experimental protocol. VO_{2max}, maximal oxygen consumption; PV, estimated plasma volume; HTT, heat tolerance test.

exhaustion to estimate VO_{2max} (Quark, Cosmed®, Rome, Italy) and MPO.

To limit learning-induced changes in pacing strategy, all participants were familiarized with the exercise protocol (time-trial) in the heat during the second session. The protocol commenced with a 10-min passive seated period, followed by a 15-min warm-up involving 10 min at a workload of 100 W and 5 min at 50% of the individual's MPO in a climate chamber (Thermo Training Room, Paris, France) at 35 °C, 50% relative humidity (RH). After the warm-up, a 20-km time-trial (TT) was performed. Each participant performed both the warm-up and the TT on their own bike mounted on a braked Cyclus2 ergometer (RBM GmbH, Leipzig, Germany). To control for fluid intake between sessions, the participants were instructed that they could drink *ad libitum* during the passive phase, warm-up, and TT, with the volume of water ingested measured, and then replicated for the ensuing experimental sessions.

For the two experimental pre-HA sessions, participants completed the protocol in the above laboratory conditions, either with (PC) or without (NC) a PC intervention, in a randomized and counterbalanced order (seven participants completed PC session in the first session). During the NC condition, the participants performed the same protocol as the familiarization session. During the PC session, an ice vest (CryoVest®, CryoInnov, Saint Grégoire, France) was used that included four anterior and posterior pockets equipped with sealed packs of FirstIce® (150 × 150 mm, 120 g; EzyWrap, EzyWrap, CryoInnov, Saint Grégoire, France; body surface cooling = 0.18 m^2; total weight with the compresses ~1.9 kg). Ten minutes before the participant entered the room, the ice packs were removed from a −18 °C freezer and placed inside the pockets to allow cold transfer to the vest. The vest was worn during both the passive phase and the warm-up, though removed before the TT.

The two post-HA sessions were performed in a heat chamber at CREPS (Guadeloupe) at the end of the training camp and in the same order as the pre-HA sessions. To reproduce pre-HA conditions, participants were asked to rest in a temperate room at the same temperature as the pre-HA environment (~21 °C) for 20 min before entering the heat chamber. Ambient conditions (temperature and RH) from the heat chambers of the two laboratories were controlled (Kestrel 4500; Nielsen-Kellerman Co, Boothwyn, Pennsylvania, USA) every 5 min throughout experimental sessions.

To ensure that any variations in performance during the TTs were due to experimental procedures and not to the previous training load, subjects were required to avoid heavy training or fatiguing activities during the 20 h prior to each laboratory session. During the TT, convective airflow from a fan set to a standard speed (750 mm, 1450 ± 5 rpm, ~8.5 ms) facing the participant was used to mimic field conditions. The main measurements performed during the TT were the time required to complete the 20 km and the power output (PO) and speed (km/h) recorded by the Cyclus2 software at a sampling rate of 2 Hz. No feedback was provided to the subjects during TTs except for the distance remaining, and they were not informed of their performance until the end of the study. To show the participant's mean pacing strategy, PO values obtained for each TT were reported per km of the TT.

Experimental measurements for the time-trial protocol

Six and a half hours (Lee et al., 2010) before arriving at the laboratory, the participants were instructed to swallow an ingestible radio telemetry capsule (VitalSense; Mini Mitter, Bend, Oregon) to measure Tcore via an external sensor (HQI Inc, Coretemp®, Sarasota, Florida, USA). The participants were also instructed to consume 1 L of water in the 2 h prior to visiting the laboratory. Upon arrival at the laboratory, the subjects provided a urine sample as an indicator of hydration status based on urine specific gravity (USG) measured using a clinical refractometer (PAL-10S, Atago Co. Ltd, Tokyo, Japan). Following provision of a urine sample, participants then filled out a questionnaire assessing fatigue, motivation, and delayed onset muscle soreness (DOMS; Vaile et al., 2008).

Tskin was measured in temperate conditions (~21 °C) before the passive phase, immediately after the warm-up, and the TT (~20 s) using a Thermo Vision SC 640 Thermal imaging camera (Flir Systems, Danderyd, Sweden) with the corresponding software (Thermacam Researcher Pro 2.10, Flir Systems, Danderyd, Sweden). Thermograms of the body (torso, abdominal, right and left forearms, arms, thighs, and legs, and respective posterior regions all towel dried) were obtained with the camera placed 4 m from the participant.

Immediately after each Tskin measurement, towel dried nude body mass (BM) was measured using a digital platform scale (± 100 g, ED3300; Sauter Multi-Range, Ebingen, West Germany) to estimate sweat loss (pre – post BM + fluid ingested).

During the TT, RPE was assessed every 4 km from the start to the end of the TT (Borg, 1998). At the same time, Tcore values were recorded, and TC was assessed using a 10-point scale, with −5 as "very uncomfortable" and +5 as "very comfortable". Heart rate (HR) was continuously sampled every 5 s (Polar, Kempele, Finland) during the TT.

Heat-acclimatization measurements

Plasma volume

Plasma volume was determined before (on the day before departure to Guadeloupe), during (on the day before experi-

mental sessions), and after (upon arrival in Paris) the training camp. Before and after the camp, PV was derived from the measurement of total hemoglobin mass, performed with a carbon monoxide (CO) rebreathing technique, as previously described (Robach et al., 2014). Briefly, after 20 min of rest, the subject breathed 100% O_2 for 4 min, before rebreathing chemically pure CO (CO N47; Air Liquide, Paris, France) for 10 min. After rest and immediately at the end of the rebreathing period, 1.5 mL of blood was obtained for percent carboxyhemoglobin, hemoglobin concentration (hemoximeter ABL800; Radiometer, Copenhagen, Denmark), and hematocrit to derive PV (Robach et al., 2014). During the camp, hematocrit percentage (micromethod, 4 min at 13 500 rpm) and hemoglobin concentration (Dill & Costill, 1974) were analyzed in quadruplicate and used to estimate percent changes in PV. All tests were performed by the same operator.

Heat Tolerance Test

The HTT was performed to examine thermoregulatory responses to given thermal and exercise stresses. Participants were instructed to standardize their hydration prior to each TT, and were not allowed to consume fluid during the HTT. The test consisted of a 10-min rest period and 30 min of cycling exercise at 50% of MPO (same ergometer and adjustments as the MPO test) in a climate chamber set at 35 °C, 50% RH. Every 5 min during the cycling test, Tcore and perceived TC sensations were recorded.

Sweat concentration analysis

Before participants entered the heat chamber, dermal patches (5 × 9 cm, Tegaderm, HP, 3M®, Neuss, Germany) were applied inferiorly to the participants' *scapula* to collect sweat samples. At the end of the HTT, the absorbent tissue contained in the patch was carefully separated from the adhesive tape using sterile tweezers, before being inserted into the tube of a single-use syringe (Terumo syringe SS+10ES1 10 mL, Terumo Europe, Belgium) for sweat extraction. The sweat sample obtained was then stored frozen at −18 °C in aliquots (Eppendorf type, 2000 μL per sample) until analysis.

Sweat samples from the HTTs were analyzed for sodium concentration using Inductively Coupled Plasma Atomic Spectrometry (ICP-AES) on a ICAP® 6300 DV simultaneous spectrometer (Thermo Scientific, Les Ulis, France). Samples were diluted 1:10 in ultrapure water (MilliQ®, Millipore, Guyancourt, France). Calibration curves were made with NaCl 0.09% in place of sweat and spiked with 0.1 g/L multielements standard solution (CCS-4, Inorganic™ Ventures, distributed by Analab, Hœnheim, France). Final standard concentrations were: 0; 62.5; 125; 250 mg/L. Utak® urine normal and high ranges (Utak Laboratories Valencia, CA, USA) and Seronorm urine Level 2 (Sero) both distributed by Ingen-Biosciences (Chilli-Mazarin, France) were used as internal QC control.

Heat-acclimatization procedure

During the 8-day training camp in Guadeloupe, from breakfast to dinner, participants were instructed to remain outdoors and asked to return to their accommodation only to shower after training sessions. Meals, recovery periods, and social activities were completed outdoors. Running, cycling, and swimming sessions were all performed outdoors (30 ± 5 °C, 74 ± 15% RH), while strength and conditioning training was performed in a weight room (26 ± 3 °C, 43 ± 16% RH). The participants reproduced their habitual weekly training program so that training distribution, activities, and content were kept constant from Paris to the training camp (see next section and Table 1 for details).

Training monitoring

The participant's internal and external training loads were monitored throughout the study. Before the training camp, subjects continuously recorded their usual training program over a 3-week period. For each training session, they were equipped with a Global Positioning System (GPS) monitor (Garmin Forerunner 305 GPS®, Garmin International, Inc., Kansas, Missouri, USA) to measure training distance and speed. Based on these measures, a typical training week was calculated so that participants could reproduce it during the training camp (i.e., matched for weekly training distribution, activities, and content). To ensure that the training completed in Guadeloupe was similar to those applied in France, the external training loads were also monitored. Additionally, all training sessions were monitored using the session-RPE method (Foster et al., 2001) using Borg's category-ratio 15 scale (Borg, 1998).

Data analysis

As previously recommended by Hopkins et al. (2009a, b) for studies in sports medicine and exercise sciences, magnitude-based inference analyses were performed on each aforementioned dependent variable. Accordingly, we calculated the between-trial standardized differences or effect sizes (ES, 90% CI) using the pooled standard deviation (Cohen, 1988). Threshold values for ES statistics were 0.2, 0.6, 1.2, 2.0, and 4.0 of the within-athlete variation, as thresholds for *small*, *moderate*, *large*, *very large*, and *extremely large* differences in the changes observed between trials (Hopkins et al., 2009a , b). The smallest worthwhile change (SWC) was defined as (a) 0.2 × 1.3 for TT's performance (Paton & Hopkins, 2006), (b) 0.2 × 1.3 × 2.5 for PO values (Bonetti & Hopkins, 2009), and (c) a small standardized effect based on Cohen's effect size principle (0.2× between-athletes standard deviation [Hopkins et al., 2009a, b]) for other parameters. Accordingly, the SWC was determined to be 0.3% in performance time, and 0.7% in PO. Quantitative chances of higher or lower differences were qualitatively evaluated as follows: <1%, *almost certainly not*; 1–5%, *very unlikely*; 5–25%, *unlikely*; 25–75%, *possible*; 75–95%, *likely*; 95–99%, *very likely*; >99%, *almost certain*. If the chance of higher or lower differences was >5% when considering the proportion of positive vs negative effects of the intervention on the variable of interest (e.g., 50/25/25), the true difference was deemed *unclear*. Otherwise, we interpreted that change as the observed chance. All values are presented as means ± standard deviation (SD).

As the effects of PC progressively dissipate as exercise in the heat continues, we investigated whether the pattern of PC-related effects on performance at Post (Guadeloupe) differed from these at Pre (Paris). To examine this, a Pearson correlation was performed between PC-induced differences in PO before and after HA on a 5-km-block basis. In addition, as individual responses may be observed relative to PC-induced TC, and that this may affect performance, associations between individual perceptual responses to PC and PO were also explored.

Table 1. Mean environmental and individual characteristics, and data from training monitoring for Paris and Guadeloupe

	Variables	Pre-HA		Post-HA	
		NC	PC	NC	PC
Testing data	Env Temperature [°C]	35.2 ± 0.8	35.1 ± 0.7	35 ± 0.5	35.1 ± 0.5
	RH [%]	51.9 ± 4.5	49.9 ± 2.9	51.2 ± 3.5	50.3 ± 2.5
	DOMS [Likert]	2.2 ± 0.8	2.2 ± 0.9	2.6 ± 0.9	2.5 ± 0.9
	Fatigue [Likert]	3.7 ± 1	3.6 ± 0.9	0.7 ± 1.3	3.6 ± 1.4
	Motivation [Likert]	4.2 ± 0.4	4.2 ± 0.7	4 ± 1	4.2 ± 0.7
	USG	1.008 ± 0.006	1.009 ± 0.009	1.011 ± 0.007	1.011 ± 0.007
	Heart rate (bpm)	167 ± 10	165 ± 9	164 ± 8	165 ± 8
	Thermal comfort (AU)	−1.7 ± 1.3	−0.9 ± 1.4†	−1.7 ± 0.9	−1.2 ± 1.1‡
		Paris		Guadeloupe	
Training data	Training volume (min)	843 ± 216		842 ± 216	
	Training load	11245 ± 2305		11901 ± 2355*	
	Distance (km) Cycling	263 ± 84		261 ± 74	
	Running	35 ± 21		31 ± 17	
	Swimming	8 ± 3		9 ± 5	
	Frequency Cycling	5 ± 2		5 ± 1	
	Running	3 ± 1		3 ± 1	
	Swimming	3 ± 1		3 ± 1	

Results are presented as the group mean ± SD.

NC, non-cooling; PC, pre-cooling; env, environmental; RH, relative humidity; DOMS, delayed onset muscle soreness; USG, urine specific gravity upon arriving at the laboratory.

*_Likely_ increase compared to Paris (ES ± 90% CI, 0.33 ± 0.32).

†_Almost certain_ increase compared to NC Pre-HA (ES ± 90% CI, 0.52 ± 0.26).

‡_Likely_ increase compared to NC post-HA (ES ± 90% CI, 0.32 ± 0.28).

Results

Participant, environmental, and training characteristics

Characteristics of each parameter are shown in Table 1. Differences in DOMS, fatigue, motivation, USG levels, and thermal environment between the four conditions, as well as differences in the weekly training characteristics (volume, distance, or frequency) between Paris and Guadeloupe were *unclear*. In contrast, as compared to Paris, the training camp induced a *likely* (76/23/1, ES ± 90% CI, 0.33 ± 0.32) increase in internal training load.

Heat-acclimatization

During each day of the training camp, the athletes spent ~14 h in outdoor natural heat exposure and 121 ± 31 min per day training in the heat. Changes in PV were *almost certain* (100/0/0) both during (from 3603 ± 508 to 4190 ± 602 mL, 16.6 ± 8.5%, ES ± 90% CI, 0.98 ± 0.23) and after the camp (4068 ± 492 mL, 11.8 ± 6.9%, ES ± 90% CI, 0.71 ± 0.20). From pre- to post-training camp, smaller increases in Tcore during the HTT were *almost certain* (−0.2 ± 0.3 °C, 0/2/97, ES ± 90% CI, −0.85 ± 0.54) (Fig. 2a), although changes in TC were *unclear* (0.6 ± 1.3, 35/14/51, ES ± 90% CI, −0.23 ± 2.15) (Fig. 2b). Increases in sweat loss from the HTT were *almost certain* (0.26 ± 0.25 L, 99/1/0, ES ± 90% CI, 0.95 ± 0.49) (Fig. 2c), while the decrease in sweat sodium concentration was *likely* (221 ± 200 mg/L, 1/12/87, ES ± 90% CI, −0.50 ± 0.47) (Fig. 2d).

Time-trial performances

Power output

The temporal changes in PO for each TT are shown in Fig. 3. An overall range of individual effects of *likely* and *trivial* to *very large* beneficial effects of PC on PO was observed Pre-HA (249 ± 38 and 259 ± 35 W for the NC and PC conditions, respectively, 94/1/5, 4.4 ± 4.6%). Specifically, during each 5-km split of the TT, PC benefits were *likely* (0–5 km: 14 ± 29 W, 93/1/6, 6.5 ± 7.4%), *very likely* (5–10 km: 13 ± 23 W, 95/1/4, 6.1 ± 6.0%; 10–15 km: 11 ± 19 W, 95/1/4, 4.7 ± 4.6%), and *unclear* (15–20 km: 2 ± 22 W, 57/6/38, 0.6 ± 4.6%).

When comparing the pre- to post-HA NC trials, the training camp induced an *almost certain very large* to *extremely large* improvement in PO (28 ± 19 W, 100/0/0, 11.7 ± 4.1%).

At the end of the training camp, PC induced an overall *unclear* effect on PO (from 277 ± 37 to 281 ± 35 W, 76/7/17, 1.4 ± 3.0%). During each 5-km split of the TT, however, PC-related benefits were first *possible* (0–5 km: 5 ± 25 W, 70/5/25, 1.7 ± 5.0%) and *likely* (5–10 km: 5 ± 19 W, 80/4/16, 2.2 ± 4.9%), before becoming *unclear* (10–15 km, 3 ± 21 W, 65/6/29, 1.1 ± 4.2%; 15–20 km: 2 ± 16 W, 64/8/27, 0.8 ± 2.9%). As compared to pre-HA, wearing the ice vest post-HA induced a

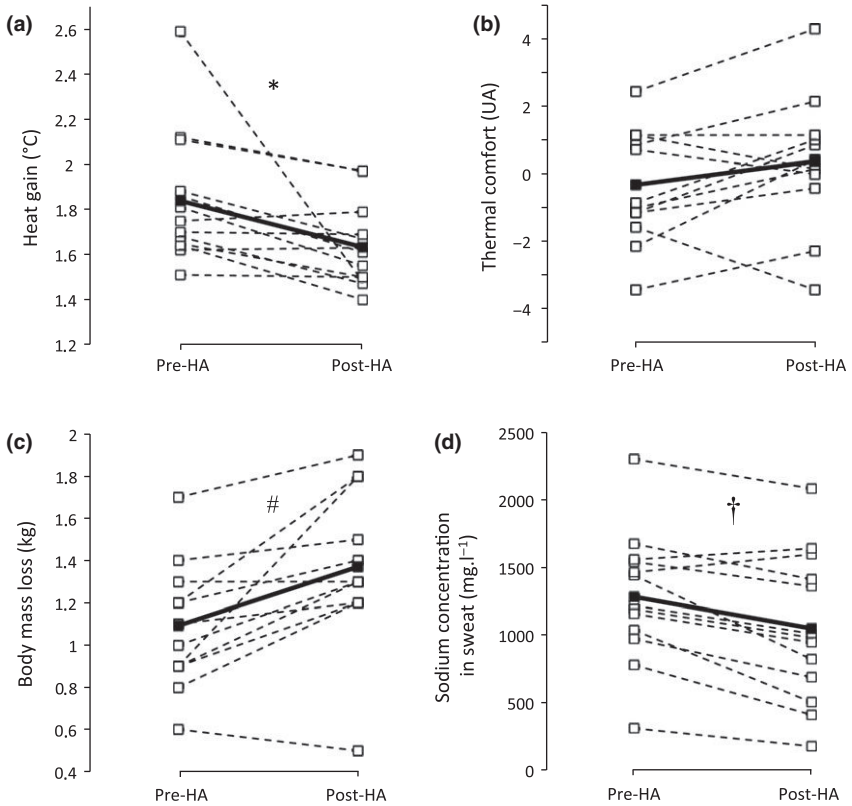


Fig. 2. Individual changes to heat-acclimatization-related HTT measurements pre- and post-training camp in (a) Tcore; (b) Thermal comfort; (c) Body mass loss; (d) Sodium concentration in sweat. *Almost certain decrease compared to pre (ES ± 90% CI, −0.85 ± 0.54); #Almost certain decrease compared to pre (ES ± 90% CI, 0.95 ± 0.49); †Likely decrease compared to pre (ES ± 90% CI, −0.50 ± 0.47).

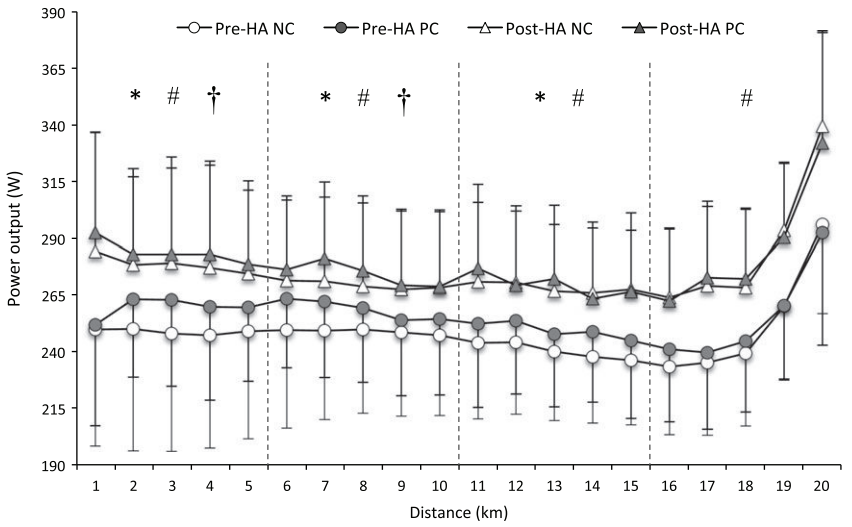


Fig. 3. Power output per kilometer for the four time-trials in the experimental protocol. Results are presented as the group mean ± SD. HA, heat-acclimatization; NC, non-cooling; PC, pre-cooling. *Changes at least *likely* between Pre-NC and Pre-PC; #Changes at least *almost certain* between Pre-NC and Post-NC; †Changes at least *possible* between Post-NC and Post-PC

likely (93/3/5) and *trivial* to *very large* (8.4 ± 9.3%) beneficial effect on PO.

Finally, correlational analyses revealed that PC-induced differences in PO before and after HA were positively correlated (r = 0.43 ± 20). Furthermore, individual differences in PC-related changes in TC between Post and Pre were related to individual changes in PC effects on PO between Post and Pre.

In particular, this was evident at 4 km ($r = 0.54 \pm 0.38$) and 8 km ($r = 0.48 \pm 0.39$), then reducing in association for the rest of the TT (12 km, $r = 0.26 \pm 39$; 16 km, $r = 0.23 \pm 35$; 20 km, $r = 0.09 \pm 32$).

TT duration

Changes in individual performance for each TT are shown in Fig. 4. Before HA, PC induced a *likely* benefit on TT duration (min:s; from 32:29 ± 01:39 to 32:04 ± 01:14 for NC and PC, respectively; 4/8/87, −1.3 ± 1.6%). The training camp had an *almost certain* positive effect on TT duration (−67 ± 58 s from the NC test at Pre to the NC test at Post; 100/0/0, −3.1 ± 1.7%). At the end of the training camp, the overall PC effect on TT duration was *unclear* (−6 ± 42 s, 14/30/56, −0.4 ± 1.1%). As compared to pre-HA, wearing the ice vest post-HA induced a *likely* (94/2/4) beneficial effect on TT duration (−1.7 ± 1.5%).

Physiological and perceptual measurements during the TT

TT core and skin temperatures

Time-trials resulted in *almost certain* increases in Tcore (from 37.6 ± 0.5 °C pre-TT to 39.5 ± 0.6 °C post-TT, 100/0/0, ES ± 90% CI, 4.61 ± 0.52). However, the changes in Tcore within and between pre- and post-HA TTs were *unclear*.

PC-induced decreases in whole body Tskin pre-TT were *almost certain* both pre- and post-HA (−1.5 ± 0.9 °C, 100/0/0, ES ± 90% CI, 3.23 ± 0.56 and −1.2 ± 0.7 °C, 100/0/0, ES ± 90% CI, 2.43 ± 0.55, respectively) (Fig. 5). Before the training camp, during the NC condition, the increase in Tskin was *likely* during the TT (0.9 ± 1.5 °C W, 90/9/1, ES ± 90% CI, 0.59 ± 0.51). In contrast, during the NC condition post-HA, changes in Tskin were *unclear* during the TT (0.2 ± 1.1 °C, 41/24/35, ES ± 90% CI, 0.06 ± 1.15).

Accordingly, the increase in the gradient of temperature during the TTs, post-HA was *likely* higher as compared to pre-HA (1.0 ± 0.3 °C, 82/15/4, ES ± 90% CI, 0.53 ± 0.65). Moreover, regardless of the HA status, changes in the core-to-skin gradient were *almost certain* during the TT in the NC condition (1.2 ± 0.3 °C, 100/0/0, ES ± 90% CI, 3.44 ± 0.47) though *unclear* using the ice vest (0.0 ± 0.3 °C, 31/28/41, ES ± 90% CI, −0.07 ± 0.99).

TT sweat loss and heart rate

Sweat losses during the TT were *likely* increased due to HA (0.28 ± 0.32 L, 93/6/1, ES ± 90% CI, −0.75 ± 0.60), and *likely* reduced by PC pre-HA (−0.05 ± 0.23 L, 3/22/75, ES ± 90% CI, −0.40 ± 0.52), but not post-HA (*unclear*, −0.03 ± 0.23 L, 8/52/39, CI ± 90%, −0.14 ± 0.41). Changes in HR due to HA (−2 ± 10 bpm, 17/35/48, ES ± 90% CI, −0.18 ± 0.69), PC pre-HA (−1 ± 5 bpm, 8/83/8,

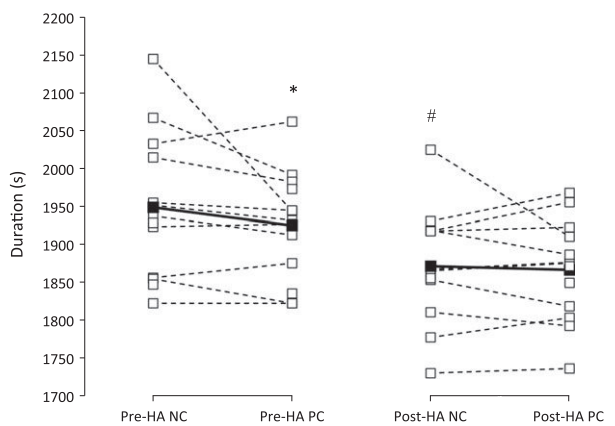


Fig. 4. Individual time-trial duration for the four conditions of the experimental protocol. HA, heat-acclimatization; NC, non-cooling; PC, pre-cooling. **Likely* decrease compared to Pre-HA NC (ES ± 90% CI, −1.3 ± 1.6%); #*Almost certain* decrease compared to Pre-HA NC (ES ± 90% CI, −3.1 ± 1.7%). Without the participant reporting drastic decreases in the two PC conditions (at the top of the graphs), results were as follow: *possible* between Pre-HA NC and Pre-HA PC (4/23/72; ES ± 90% CI, −0.6 ± 0.8%); *very likely* between Pre-HA NC and Post-HA NC (2/3/95; ES ± 90% CI, −2.5 ± 2.1%), and *unclear* between Post-HA NC and Post-HA PC (31/51/17; ES ± 90% CI, 0.1 ± 0.7%).

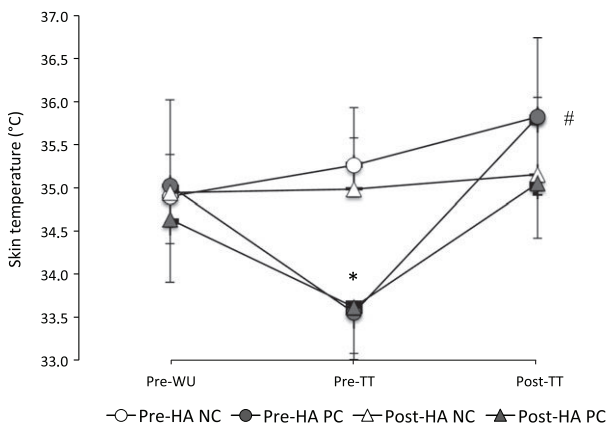


Fig. 5. Changes in skin temperature at different times during the experiment for each condition Pre- and Post-training camp. Results are presented as the group mean ± SD. HA, heat-acclimatization; NC, non-cooling; PC, pre-cooling; Pre-WU, before the passive phase; Pre-TT, immediately before the time-trial; Post-TT, immediately after the time-trial. **Almost certain* decrease compared to NC conditions (ES ± 90% CI, 3.23 ± 0.56 and ES ± 90% CI, 2.43 ± 0.55 for Pre-HA and Post-HA, respectively); #*Likely* increase compared to Pre-WU (ES ± 90% CI, 0.59 ± 0.51).

ES ± 90% CI, 0.00 ± 0.24), or PC post-HA (1 ± 7 bpm, 25/64/10, ES ± 90% CI, 0.06 ± 0.36) were *unclear* (Table 1).

Thermal comfort and RPE

Pre- and post-HA, TC was *almost certainly* (0.7 ± 0.8 UA, 98/2/, ES ± 90% CI, 0.52 ± 0.26) and *likely* (0.4 ± 0.8 UA, 77/22/0, ES ± 90% CI, 0.32 ± 0.28) improved due to PC, respectively. HA-related changes in TC were *unclear* (0.0 ± 1.2 UA, 20/58/22, ES ± 90% CI, −0.01 ± 0.43). Changes in RPE values due to HA (0.4 ± 0.8 UA, 49/44/8, ES ± 90% CI, 0.19 ± 0.46), PC pre-HA (−0.2 ± 1.1 UA, 6/36/58, ES ± 90% CI, −0.26 ± 0.48), or PC post-HA (0.2 ± 1.0 UA, 20/64/16, ES ± 90% CI, 0.01 ± 0.38) were *unclear*.

Discussion

The primary aim of this study was to test the hypothesis that HA and PC cumulate to improve self-paced endurance performance in the heat. In contrast to our hypothesis, the results showed that the combination of HA and PC using an ice vest did not further improve 20-km TT performance in the heat as compared to HA alone. Nonetheless, analysis of pre- and post-HA pacing strategies based on presence of PC revealed a small effect of PC on PO during the initial stages of the TT post-HA. Given that both PC and HA improved TT performance without exacerbated physiological responses, it is likely that the HA-induced cardiovascular and thermoregulatory adaptations reduced the ergogenic effects of PC. Despite the blunted effects on the overall performance, PC following HA improved perceptions of thermal tolerance, which appeared to be related to pacing.

Acute pre-cooling for endurance performance in the heat

In agreement with some (Johnson et al., 2008; Bogerd et al., 2010), but not all (Quod et al., 2008; Stannard et al., 2011) studies, the present results showed that ice vest PC improved 20-km TT performance in the heat. While the 4.4% increase in PO observed in the current study is similar to the 5.2% improvement during a 20-km TT reported by Johnson et al. (2008), Quod et al. (2008) only reported a 1.5% improvement in similar environmental and self-paced exercise conditions to the present study. However, in their study, Quod et al. (2008) removed the cooling vest before the 20-min warm-up, which may have reduced the efficacy of PC on the TT performance. Also, despite comparable durations of PC during the warm-up, our results differ from those of Stannard et al. (2011), who reported no effect of wearing a cooling vest before 40-min running exercise in warm conditions. The greater thermal stress imposed in our study (35 °C vs ~25 °C in Stannard et al. (2011)), as well as the ice vests' cooling efficiency may explain this difference (Ross et al., 2013). Notably, the Cryo-Vest® (body surface cooling = 0.18 m^2; weight <2 kg) enhanced TT performance despite fan-related airflow – which restricts PC benefits in laboratory settings (Morrison et al., 2014). This type of cooling might thus be relevant for outdoor competitions in the heat of ~30 min, as the effects of PC on PO progressively decreased with exercise duration.

As expected, PC reduced Tskin at the beginning of the TT, which likely enabled participants to adopt a higher PO (+10 ± 18 W) while maintaining similar cardiovascular and thermoregulatory responses to the NC condition. This absence of increases in HR, Tcore, and sweat loss despite the greater PO may be due to cooling-related reduced cutaneous vasodilatation and higher heat loss via tissue conduction (transfer from the core to the skin) and subsequent fan-based convection (transfer from the skin to the environment), respectively (Saunders et al., 2005). Tskin reached similar temperatures at the end of the TTs in both NC and PC conditions, suggesting the PC-induced benefits on PO paralleled the ephemeral PC effect on the core-to-skin gradient. Either due to, or alongside the reduced relative physiological loads, PC up-regulated the pacing pattern while maintaining the same perceptual exertion as in NC. This may result from the central integration of cutaneous afferences, as TC *per se* is able to influence PO without modifying RPE (Schlader et al., 2011, Flouris & Schlader 2015).

Heat-acclimatization and endurance performance in the heat

As determined from the HTT, the participant's exhibited symptoms of HA such as reduced heat gain (−0.2 ± 0.3 °C) and improved sweating rate (0.26 ± 0.25 L). PV also increased (16.6 ± 8.5% on day 7, 11.8 ± 6.9% on day 11) and sweat sodium concentrations were reduced during the TT (−17 ± 19%). Comparatively, these changes are similar to those observed in well-trained cyclists after a 2-week training camp (Karlsen et al., 2015), but greater than those previously reported using training camp models in professional football players (see Buchheit et al., 2011; Racinais et al., 2013). Such discrepancies may be as a result of the effective training volume of the present triathlete population and/or the large daily passive phases spent outdoors (Garrett et al., 2011).

The HA exhibited a *very large* effect on PO in the NC condition (ES ± 90% CI, 11.7 ± 4.1%). Such extent of performance improvement has not previ-

ously been reported on self-paced cycling exercise after heat-acclimation of similar duration (e.g., Lorenzo et al., 2010). Hence, it may be the combination of daily passive heat exposure with the heat training that amplified the performance improvement (Shido et al., 1999; Racinais et al., 2015). Moreover, the magnitude of physiological (i.e., cardiovascular and thermoregulatory) changes and perceptual adaptations may explain this improvement. Specifically, it is possible PV expansion and Tskin reduction enabled increased PO while limiting homeostasis disturbance (e.g., Tcore and cardiac strain, muscle metabolism). As evidence, PV expansion is suggested to offset increased HR responses by facilitating improved blood flow distribution during exercise (Nielsen et al., 1993). In parallel, as supported by our results, increased sweat losses provided greater evaporative cooling and core-to-skin gradient, especially in the presence of airflow (Saunders et al., 2005). Additionally, as a result of, or alongside these improved thermoregulatory adaptations, the athletes TC during the 20-km TT was similar to pre-HA despite the higher metabolic rate, and this may also allowed for increased PO (Schlader et al., 2011; Schulze et al., 2015).

Effect of pre-cooling after heat-acclimatization

In contrast to the initial hypothesis, PC did not provide further benefits on the overall performance once athletes were heat-acclimatized (4 ± 14 W). These findings extend previous studies (Castle et al., 2011; Brade et al., 2013) that examined the combined effects of PC with HA during repeated intermittent sprints performed in the heat. Accordingly, it is possible that the HA from the training camp led to a "ceiling effect" (i.e., saturation) of physiological adaptations (Castle et al., 2011), or blunted the PC-related physiological effects (i.e., converging effects), thus reducing the likelihood of further notable PC benefits on performance. Crucially, although our results do not enable to distinguish between these two hypotheses, both strategies demonstrated similar influence on the physiological responses to the TT. Specifically, while post-HA PC decreased Tskin to a similar extent as pre-HA, the sudomotor adjustments resulting from the heat exposure also promoted a lowering of Tskin during the TT. It is therefore possible that earlier and larger sweat losses resulting from HA (Shido et al., 1999) may have subsequently reduced the effectiveness of PC by inducing a larger core-to-skin temperature gradient than pre-HA. Similarly, PC improved cardiovascular efficiency during the unacclimatized TTs, which was also reflected in the post-HA responses in regard to PV expansion. However, the extent of PV increase (16.6 ± 8.5%) may have minimized the cardiovascular effect of PC observed Pre-HA by facilitating both cutaneous and muscle blood flow distribution (Nielsen et al., 1993). Together with the absence of any changes in psychometric variables pre-TTs, these results suggest that HA did not allow PC-related initial effects on PO to be as evident post-HA as pre-HA due to consubstantial physiological (i.e., cardiovascular and thermoregulatory) adaptations.

Although this study shows the advantage of PC is minimized by HA, the analysis of pacing suggests that PC still may have some role in the *acute* protection of exercise performance in the heat, regardless of acclimatization status. Indeed, at post-HA, PC temporarily induced *small* to *moderate* benefits in the TT, though only during the initial 10 km. This observation is consistent with the fact that PC- and post-HA PC-pacing profiles demonstrated comparable relationships (r = 0.43) for the trend of diminished benefit throughout the TTs (from 14 ± 29 to 2 ± 22 W at Pre, and from 5 ± 25 to 2 ± 16 W at Post). Such similarity in the temporal profiles, albeit with a smaller magnitude at Post-HA, suggests that the effectiveness of PC be related to individual athletes HA level – and it could further be speculated that the lesser the extent of HA the more effective PC would be.

Despite the smaller magnitude, the ephemeral beneficial effect of post-HA PC is likely related to perceptual improvements. For individual participants, an additional increase in TC from the vest at Post- compared to Pre-HA was associated with an additional increase in PO, notably during the first half of the TT (r = 0.54 and r = 0.48, at 4 and 8 km, respectively), i.e., when PC-induced benefits were the most remarkable. This suggests that when pre-cooled, athletes who perceived a greater increase in TC following HA were those continuing to gain performance benefit from wearing the vest. This relationship reduces with increased TT duration. Nonetheless, these observations highlight the purported role of improvements in TC following PC to improve endurance performance beyond cardiovascular or thermoregulatory implications (Schulze et al., 2015).

Perspectives

This study strengthens previous findings that PC (Johnson et al., 2008) and HA (Lorenzo et al., 2010) independently improve self-paced endurance performance in the heat (Castle et al., 2011). However, given each of these heat-combating strategies are likely to induce convergent physiological effects, the combination of HA and ice vest PC provided no additional overall ergogenic effect to 20-km TT performance in the heat. In spite of this, when considering pacing adjustments, PC-induced ergogenic effects were still persistent post-HA dur-

ing the first half of the TT, and thus remain of interest for HA athletes. Accordingly, we would still recommend that these combined strategies be encouraged when competing in the heat to ensure improved TC and assist performance benefits. Indeed, individual perceptual benefits from PC may potentially up-regulate pacing strategies – particularly if effective HA has already provided the athlete an improved physiological tolerance of the heat. In this perspective, the respective role of physiological (Bogerd et al., 2010; Bongers et al., 2015) vs perceptive (Schlader et al., 2011; Schulze et al., 2015) pathways inherent to PC strategies and leading to endurance performance improvement in the heat remains to be fully elucidated.

Key words: Heat-dissipating strategies, tropical climate, time-trial, pacing, cycling.

Acknowledgements

This study was made possible by technical support from the French Federation of Triathlon. The authors are grateful to Anaël Aubry, Bruno Coutant, and the Centre of Sports Resources, Expertise and Performance (CREPS) of Pointe-à-Pitre (Guadeloupe) for their help and cooperation. We receive funding for research on which this article is based from the French Ministry of Sport and the French National Institute of Sport, Expertise and Performance (Paris). The authors report no conflict of interest.

References

Altareki N, Drust B, Atkinson G, Cable T, Gregson W. Effects of environmental heat stress (35 degrees C) with simulated air movement on the thermoregulatory responses during a 4 km cycling time-trial. Int J Sports Med 2009: 30: 9–15.

Bogerd N, Perret C, Bogerd CP, Mossi RM, Daanen HAN. The effect of pre-cooling intensity on cooling efficiency and exercise performance. J Sports Sci 2010: 28 (7): 771–779.

Bonetti DL, Hopkins WS. Sea-level exercise performance following adaptation to hypoxia. Sports Med 2009: 39: 107–127.

Bongers CCWG, Thijssen DHJ, Veltmeijer MTW, Hopman MTE, Eijsvogels TMH. Precooling and percooling (cooling during exercise) both improve performance in the heat: a meta-analytical review. Br J Sports Med 2015: 49: 377–384.

Borg G. Borg's perceived exertion and pain scales. Champaign: Human Kinetics, 1998.

Brade C, Dawnson B, Wallman K. Effect of precooling and acclimation on repeat-sprint performance in the heat. J Sports Sci 2013: 31(7): 779–786.

Buchheit M, Voss SC, Nybo L, Mohr M, Racinais S. Physiological and performance adaptations to an in-season soccer camp in the heat: associations with heart rate and heart rate variability. Scand J Med Sci Sports 2011: 21: 477–485.

Castle P, Mackenzie RW, Maxwell N, Webborn ADJ, Watt PW. Heat acclimation improves intermittent sprinting in the heat but additional pre-cooling offers no further ergogenic effect. J Sports Sci 2011: 29(11): 1125–1134.

Cohen J. Statistical power analysis for behavorial sciences. Hillsdale, NJ: Lawrence Erlbaum, 1988: 567.

Coris EE, Ramirez AM, Van Durme DJ. Heat illness in athletes. Sports Med 2004: 34(1): 9–16.

Cuddy JS, Hailes WS, Ruby BC. A reduced core to skin temperature gradient, not a critical core temperature, affects aerobic capacity in the heat. J Thermal Biol 2014: 43: 7–12.

Daanen HAM, Jonkman AG, Layden JD, Linnane DM, Weller AS. Optimising the acquisition and retention of heat acclimation. Int J Sports Med 2011: 32(11): 822–828.

Dill DB, Costill DL. Calculation of percentage changes in volumes of blood, plasma and red cells in dehydration. J Appl Physiol 1974: 37: 247–248.

Febbraio MA, Snow RJ, Hargreaves M, Stathis CG, Martin IK, Carey MF. Muscle metabolism during exercise and heat stress in trained men: effect of acclimation. J Appl Physiol 1994: 76: 589–597.

Flouris AD, Schlader ZJ. Human behavioral thermoregulation during exercise in the heat. Scand J Med Sci Sports 2015: 25(Suppl. 1): 52–64.

Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, Doleshal P, Dodge C. A new approach to monitoring exercise training. J Strength Cond Research 2001: 15(1): 109–115.

Garrett AT, Rehrer NJ, Patterson MJ. Induction and decay of short-term heat acclimation in moderately and highly trained athletes. Sports Med 2011: 41(9): 757–771.

Hopkins WG, Batterham AM, Marshall SW, Hanin J. Progressive statistics. Sportscience 2009a: 13: 55–70.

Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc 2009b: 41: 3–13.

Johnson E, Sporer B, Sleivert GG, Pethick W, editors. The effect of precooling and ambient temperature on 20 km time-trial performance in trained cyclist. CSEP Annual Scientific Conference; 15–18 October. Banff, Alberta, Canada: Applied Physiology, Nutrition and Metabolism, 2008.

Karlsen A, Nybo L, Nørgaard SJ, Jensen MV, Bonne T, Racinais S. Time course of natural heat acclimatization in well-trained cyclists during a 2-week training camp in the heat. Scand J Med Sci Sports 2015: 25(Suppl. 1): 240–249.

Lee JKW, Nio AQX, Lim CL, Teo EYN, Byrne C. Thermoregulation, pacing and fluid balance during mass participation distance running in a warm and humid environment. Eur J Appl Physiol 2010: 109: 887–898.

Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. J Appl Physiol 2010: 109: 1140–1147.

Morrison SA, Cheung S, Cotter JD. Importance of airflow for physiologic and ergogenic effects of precooling. J Athl Train 2014: 49(5): 632–639.

Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. J Physiol 1993: 460(1): 467–485.

Paton CD, Hopkins WG. Variations in performance of elite cyclists from

race to race. Eur J Sport Sci 2006: 6 (1): 25–31.
Quod MJ, Martin DT, Laursen PB, Gardner AS, Halson SL, Marino FE, Tate MP, Mainwaring DE, Gore CJ, Hahn AG. Practical precooling: effect on cycling time trial performance in warm conditions. J Sports Sci 2008: 23: 1–11.
Racinais S, Buccheit M, Bilsborough J, Bourdon PC, Cordy J, Coutts AJ. Physiological and performance responses to a training-camp in the heat in professional Australian football players. Int J Sports Physiol Perf 2013: 9(4): 598–603.
Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. Med Sports Sci Exer 2015: 47: 601–606.
Robach P, Bonne T, Flück D, Bürgi S, Toigo M, Jacobs RA, Lundby C. Hypoxiv training: effect on mitochondrial function and aerobic performance in Hypoxia. Med Sci Sports Exer 2014: 46(10): 1936–1945.
Ross M, Abbiss C, Laursen P, Martin D, Burke L. Precooling methods and their effects on athletic performance. Sports Med 2013: 43: 207–225.
Saunders AG, Dugas JP, Tucker R, Lambert MI, Noakes TD. The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. Acta Physiol Scand 2005: 183: 241–255.
Schlader ZJ, Simmons SE, Stannard SR, Mündel T. The independent role of temperature and thermal perception in the control of human thermoregulatory behavior. Physiol Behav 2011: 103(2): 217–224.
Schulze E, Daanen HAM, Levels K, Kilding A, Skleryk J, Plews D, Siegel R, Laursen P. Effect of cooling strategy on cycling performance in the heat: thermal state vs. thermal comfort. Int J Sports Physiol Perf 2015: 10: 655–663.
Shido O, Sugimoto N, Tanabe M, Sakurada S. Core temperature and sweating onset in humans acclimated to heat given at a fixed daily time. Am J Phyiol 1999: 276: 1095–1101.
Sleivert GG, Cotter JD, Roberts WS, Febbraio MA. The influence of whole-body vs. torso pre-cooling on physiological strain and performance of high-intensity exercise in the heat. Comp Biochem Physiol Part A 2001: 128: 657–666.
Stannard AB, Brandenburg JP, Pitney WA, et al. Effects of wearing a cooling vest during the warm-up on 10-km run performance. J Strength Cond Res 2011: 25(7): 2018–2024.
Tatterson AJ, Hahn AG, Martin DT, Febbraio MA. Effects of heat stress on physiological responses and exercise performance in elite cyclists. J Sci Med Sports 2000: 3: 186–193.
Vaile J, Halson S, Gill N, Dawson B. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. J Sports Sci 2008: 26: 431–440.

Schmit, C., Duffield, R., Hausswirth, C., Brisswalter, J., & Le Meur, Y. Optimizing heat-acclimation for performance peaking: high- vs low-intensity training. *Submitted* (in *International journal of sports physiology and performance*)

Résumé en français

Dans cette étude, nous avons étudié les effets d'entraînements à haute vs. basse intensité en chaleur, et d'une période d'affûtage consécutive, sur la performance en endurance. Dix-neuf triathlètes bien-entraînés ont réalisé 5 jours d'entraînement normal puis 1 semaine d'affûtage, chaque période incluant des entraînements à basse (HA-L, n=10) ou haute intensité (HA-H, n=9) en chaleur (30°C, 50% d'humidité). Un groupe contrôle (n=10) a reproduit le même protocole en conditions tempérées. Des épreuves de contre-la-montre à vélo de 20km (35°C, 50% d'humidité) ont été réalisées avant (Pre) et après la période d'acclimatation (Mid), et suite à la période d'affûtage (Post). Des mesures physiologiques et perceptives additionnelles au CLM ont été effectuées. La puissance de pédalage est restée stable dans le groupe contrôle entre Pre et Mid (ES: -0.10 ± 0.26) et a augmenté entre Mid et Post (0.18 ± 0.22). Le groupe HA-L a démontré une amélioration progressive de sa performance entre Pre et Mid (0.62 ± 0.33) et entre Mid et Post (0.53 ± 0.30), en parallèle de signes typiques d'acclimatation à la chaleur (réductions de la température centrale et de la fréquence cardiaque, plus grande perte de poids de corps). Alors que le groupe HA-H présentait des adaptations similaires, une augmentation de la fatigue perçue et une baisse du niveau de performance à Mid (-0.35 ± 0.26) ont aussi été observées, et rétablies à Post (0.50 ± 0.20). Aucune différence de puissance de pédalage n'a été reportée entre le groupe contrôle et le groupe HA-H en Post. Ainsi, un protocole d'entraînement HA-L semble plus à-même de promouvoir une acclimatation à la chaleur tout en limitant l'accumulation de fatigue chez des athlètes d'endurance avant une compétition en chaleur. En contraste, un protocole HA-H augmente les chances de développement d'un état de surmenage fonctionnel.

Les 3 travaux présentés jusqu'ici ont permis d'appréhender les relations entre 'exercice en conditions chaudes' et 'physiologie de l'organisme'. Dans ce contexte, pour autant, les incidences cognitives de la chaleur restent mal connues.

Optimizing heat acclimation for performance peaking: High- vs low-intensity training

Schmit C[1], Duffield R[2], Hausswirth C[1], Brisswalter J[3], Le Meur Y[1,3,4].

[1] French Institute of Sport, Expertise and Performance (INSEP), Research Department, Laboratory of Sport, Expertise and Performance (EA 7370), Paris, France

[2] Sport & Exercise Discipline Group, Faculty of Health, University of Technology Sydney (UTS), Moore Park, Australia

[3] Laboratory LAMHESS (EA6312), University of Nice Sophia-Antipolis, Nice, France

[4] Medical Department, AS Monaco Football Club, Monaco

Corresponding author:

Cyril Schmit

French Institute of Sport, Expertise and Performance

Laboratory of Sport, Expertise and Performance

11, avenue du Tremblay

75 012 Paris, France

Tel: +33 (0)1 41 74 42 95

Fax: +33 1 41 74 45 35

✉ Email: cyril.schmit@gmail.com

Short title: Heat-acclimation and ecological training

Abstract

Purpose: Current heat-acclimation (HA) models are not representative of the ecological training program of endurance athletes. We sought to determine the effect of high- vs. low-intensity training in the heat and ensuing taper period used prior on endurance performance.

Methods: Twenty-nine well-trained triathletes undertook five consecutive days of either normal training (Control, n=10), or of low- (HA-L, n=10) or high- (HA-H, n=9) intensity training sessions in the heat (30°C, 50% of relative humidity) before a one-week taper phase in the heat. Twenty-kilometre time-trials (TT; 35°C, 50% RH) were performed before the protocol (Pre) and the taper (Mid) and after the taper (Post). Additional physiological (core temperature, heart rate, body mass loss) and perceptual (rating of perceived exertion) responses to the TT were measured.

Results: The control group displayed no changes in power output (PO) from Pre to Mid (ES: -1.3 ± 1.8). The HA-L group demonstrated a progressive increase in performance from Pre to Mid (ES: 6.7 ± 4.6) and from Mid to Post (ES: 6.4 ± 3.9), alongside typical physiological signs of HA (reduced core temperature and heart rate, increased body mass loss). While the HA-H group also presented these signs, they further demonstrated symptoms of overreaching (increased perceived fatigue and decreased performance at Mid, -4.9 ± 3.5), which was reversed at Post (ES: 7.6 ± 3.1). No difference was reported at Post between the HA-H and the control groups.

Conclusion: A real-life HA-H scenario is not recommended to endurance athletes as it increases the risk of overreaching and does not result in performance supercompensation after the taper period. On the contrary, HA-L minimises fatigue development and maximises performance improvement.

Introduction

Heat-acclimation (HA) has the potential to improve performance in the heat, effectively countering heat-induced decrements and returning performance to that achieved in cool conditions (Racinais et al., 2015). In general, HA is accomplished via regular exercise at submaximal intensities to environments of sufficient temperature (Taylor, 2014; Tyler et al., 2016). In addition, due to the seemingly dose-response relationship of HA, current evidence suggest >15 days of HA to best improve performance in the heat (Guy et al., 2014). Whilst current HA models, including >15 days of low-moderate intensities, are purported to optimise HA, they are not representative of the ecological training program of endurance athletes. More specifically, these protocols sit in contrast with the high-intensity training and ensuing taper period used prior to competition. Therefore, HA protocols fitting the dual requirements of providing HA whilst conforming to ecological training needs of endurance athletes remain to specify.

The perspective that high-intensity training in the heat offers a more valid training context than low-intensity HA has recently been proposed (Tyler et al., 2016). However, only two studies to date investigated adaptations following HA of distinctly different exercise intensities. Following seven days of exercising at a low (50% of the maximal oxygen consumption [$\dot{V}O_{2max}$] for 60 min·d^{-1}) or a moderate (75% of $\dot{V}O_{2max}$ for 30-35 min·d^{-1}) intensity, Houmard et al. (1990) reported equally reduced heart rate [HR], T_{core}, and caloric expenditure (no performance measurement). More, recently, Wingfield et al. (2016) reported larger decreases in HR and T_{core} and greater performance benefits (-5.9 ± 7.0% vs. -0.18 ± 3.9% in a 20-km time-trial duration, at ~33°C, ~60% RH) following a 5-day low- (cycling at 40% of peak power output [PPO] for 90 min) vs. high-intensity (30 min of alternating 40%-70% of PPO) HA protocol. However, the "high-intensity" exercises used in these protocols are not representative of training intensities typically undertaken by athletes. Moreover, the lack of a control group, of training volume of subjects, or of sufficient daily heat exposure

(recommended to be ≥60 min, Chalmers et al., 2014; Tyler et al., 2016) cloud the interpretation of the results. This is regrettable since high-intensity training during HA (and the related endogenous heat load) could compensate for the fact that endurance athletes behave as if they were already partially heat-acclimatised, which makes them prone to insufficient thermal impulse during typical submaximal HA (Taylor, 2006). However, training at high-intensity in the heat is also likely to increase the internal training load and to augment the risk of functional overreaching. Since functional overreaching is associated with alterations in cardiovascular (reduced submaximal and maximal HR) and perceptive responses to exercise, while not incurring subsequent performance supercompensation (Decroix et al., 2016), these maladaptations are likely to counterbalance the effects of HA. To our knowledge, no performance impairment has been reported to date following HA; however athlete's tolerance to the training regimen remains crucial concerns for coaches especially when a training camp in the heat is scheduled before a major competition.

In this perspective, endurance athletes' typical precompetitive program systematically includes a taper phase between the overload (e.g., HA regiment) and the competitive periods. Although this issue appears of further relevance high-intensity training is scheduled in the heat, taper-based performance rebound has not been characterised to date consecutive to HA. Within this context, the present study investigated the respective effects of either high- (HA-H) or low-intensity (HA-L) training sessions in the heat on self-paced endurance performance in well-trained athletes before and after a taper period. We hypothesized greater psycho-physiological and performance improvements in the high-intensity HA group following the HA and the taper phases.

METHODS

Participants

Twenty-nine well-trained males triathletes (age 32 ± 4 y, height 177 ± 8 cm, body mass 70.3 ± 6.1 kg, [$\dot{V}O_{2max}$] 62.1 ± 5.4 ml·kg^{-1}·min^{-1}, maximal power output 347 ± 37W) volunteered to participate in this study (De Pauw et al., 2013). All participants competed at the national level for at least 3 y, trained ≥7 sessions per week and had no heat exposure in the previous three months. Prior to inclusion, participants were examined by a cardiologist to obtain a medical clearance. The experimental design of the study was approved by the Ethics Committee of *Hôtel Dieu*, Paris (acceptance no. 2015-A01220-49) and was performed in accordance with the Declaration of Helsinki. After comprehensive verbal and written explanations of the study, all participants gave their written informed consent for participation.

Experimental design

Participants' recruitment resulted in a homogenous profile, and thus participants were randomly ascribed to the control, HA-L or the HA-H group.

An outline of the four phases of the experimental protocol is presented in Figure 1. Simply, the initial two phases were standardised for all participants, and consisted of 2 weeks of 'normal' training regimen (phase I, no monitoring) and 2 weeks of individualised training load (phase II, training monitored). During the 1-week third period (phase III), one group of participants continued the individualised training in temperate conditions (Control, n=10), while two groups undertook respective HA procedures either in the form of low- (HA-L, n=10) or high- (HA-H, n=9) intensity training sessions, as based on phase II training. All participants then completed a final 1-wk taper (phase IV).

Prior familiarisation of all protocols, measures and environments was ensured to limit learning-induced changes, particularly for time trial pacing strategies (Foster et al., 2009). More specifically, all athletes were familiarised twice with the exercise performance protocol, including once in temperate (21°C, 50% RH) and once in hot conditions (35°C, 50% RH)

(Thermo Training Room®, Paris, France,). Performance testing, consisting of a 20km time trial (TT), was completed at the end of each week (phase II: Pre; phase III: Mid; phase IV: Post) on 3 consecutive days (one group per day). A 1-wk interval between TTs in the heat was chosen as it is sufficient i) to curtail physiological adaptations to heat from Familiarisation session 2 to Pre (Barnett and Maughan, 1993), ii) to induce HA between Pre and Mid since sufficient daily training is scheduled in the heat (i.e., ≥60min; Tyler et al., 2016), and iii) to promote taper-related recovery processes between Mid and Post for performance peaking (Bosquet et al., 2007).

Heat-acclimatisation procedure

No variable except the intensity of training sessions performed in the heat differed between the two HA groups during phases III and IV (i.e., same ambient conditions, heat exposure duration, time of the day). Specifically, over phase III, each participant of the HA-L group reproduced 1h per day during 5 consecutive days of the lowest intensity sessions of his personal training week at 30°C and 50% RH (e.g., 60min of an initial 90min running session at 11km·h^{-1}), while athletes from the HA-H group underwent 1h per day of their personal highest intensity sessions in similar conditions (e.g., eight repetitions of 400 m at 19km·h^{-1}, with warm-up and recovery phases). In the case that a session was longer than 60min, the remaining training duration was performed out of the heat chamber in temperate conditions (21°C, 50% RH). A similar split between training duration in temperate and hot conditions was maintained and adjusted for the tapering phase (i.e., 30min of training in the heat and the remaining training duration performed out of the heat chamber). For each participant, the selection of the training sessions performed in the heat was determined according to his typical training week set up from phase I, and relative to session's absolute intensity (km·h^{-1}

or Watt [W]) and duration (≥ 60min). Only running, cycling and rowing sessions (and no swimming, strength and conditioning sessions) were scheduled in the climate chamber.

During phases III and IV, outdoor environmental conditions from 6.00 am to 10 pm were 6.5 ± 4.1°C and 65.0 ± 15.3% RH (www.infloclimat.fr). To minimize intervention-based management differences between groups during these two weeks, participants of the control group undertook some training sessions in the lab (at 21°C, 50% RH).

Exercise performance

Maximal Power Output test

During the first session, a graded exercise test was performed in temperate conditions (21°C, 50% RH) using an electronically-braked cycle ergometer (Excalibur Sport, Lode®, Groningen, The Netherlands). Participants wore a facemask covering their mouth and nose to collect all expired breath (Hans Rudolph, Kansas City, MO). The exercise protocol started with a 5-min warm-up at a workload of 100 W, and then increased by 25 W per 2 minutes until voluntary exhaustion to estimate $\dot{V}O_{2max}$ (Quark, Cosmed®, Rome, Italy) and Maximal Power Output (MPO).

Time-trial protocol

An outline of the TT protocol is presented in Figure 1. The protocol was performed on participants' own bike mounted on a Cyclus2 ergometer (RBM GmbH, Leipzig, Germany). Participants were required to avoid heavy training or fatiguing activities during the 24 h prior to each laboratory session. To control for fluid intake between sessions, the participants were instructed during Familiarisation session 2 that they could drink *ad libitum* during the warm-up and TT, with the volume of water ingested measured, and then replicated for the ensuing experimental sessions. During the warm-up and the TT, convective airflow from a fan was

used to mimic field conditions (750 mm, ~6 $m \cdot s^{-1}$ and ~8.5 $m \cdot s^{-1}$, respectively). During the TT, duration (min) to complete the 20 km TT and the power output (PO) were recorded by the Cyclus2 software at a sampling rate of 2 Hz. No feedback was provided to the participants during TT's except for the distance remaining. Pacing analysis was performed over a 5 km-block basis, since it appears effective in discriminating heat-related alterations in pacing strategies during a series of 20-km TTs (Schmit et al., 2015).

Experimental measurements

Six and a half hours before arriving at the laboratory (Lee et al., 2010), the participants were instructed to swallow an ingestible radio telemetry capsule (e-Celsius performance, BodyCap, Caen, France) to measure T_{core} via an external sensor (e-Viewer performance, BodyCap, Caen, France). The participants were also instructed to consume 1L of water in the 2 hours prior to visiting the laboratory. Upon arrival at the laboratory, the participants provided a urine sample as an indicator of hydration status based on urine specific gravity (USG) measured using a clinical refractometer (PAL-10S, Atago Co. Ltd, Tokyo, Japan).

Participants then completed a questionnaire assessing fatigue, motivation and delayed onset muscle soreness (DOMS; Vaile et al., 2008) via respective 100mm visual analogic scales, before performing one training block of a cognitive task (80 trials, <3min) (see *Cognitive task* section). Before and immediately after the warm-up and the TT, towel dried nude body mass (BM) was measured using a digital platform scale (ED3300; Sauter Multi-Range; Ebingen; West Germany, ± 100g) to estimate sweat loss (pre – post BM + fluid ingested). During the TT, participant's rating of perceived exertion (RPE) was assessed every 5 km using Borg's category-ratio 15 scale (Borg, 1998). T_{core} values were recorded at the beginning and the end of the warm-up, and every 5km during the TT. HR was continuously sampled every 5 s (Polar, Kempele, Finland) during the warm-up and the TT.

Training monitoring

For each training session during phase I, the participants were equipped with a HR monitor (Polar, Kempele, Finland) and provided with a personal diary to measure training duration, calculate training speeds, and report a session-RPE (Foster et al., 2001) using Borg's category-ratio 15 scale (Borg, 1998) for the purpose of calculating training load. Based on these measures, a typical training week was established so that participants could reproduce it during phases II and III (i.e., matched for weekly training distribution, activities and content; see Table 1 for details). The phase IV taper was also calculated according to guidelines for optimal tapering in endurance sports (Bosquet et al., 2007). For example, a 1-h run including 8 x 400 m at the maximal aerobic running speed was converted into an approximately 30-min run including 4 x 400 m at the same speed. To ensure that training patterns applied during phases III and IV were coherent with the individualised training, participants remained equipped with the monitor and the HA sessions were supervised by a researcher.

Statistical analysis

Data were analysed using a magnitude-based inference approach for all parameters (Hopkins et al., 2009). All data were log transformed before analysis to reduce bias arising from non-uniformity of error. For clarity, the values presented in the text, tables and figures are not transformed. The magnitude of the within-group changes, between-group differences in the changes, and differences in the changes of group mean, were interpreted by using values of 0.2, 0.6, 1.2, 2.0 and 4.0 of the variation as thresholds for *small*, *moderate*, *large*, *very large*, and *extremely large* differences in the change between the trials (Hopkins et al., 2009). The smallest worthwhile change (SWC) was defined as 1) 0.2 x 1.3 for TT's performance (Paton & Hopkins, 2006), 2) 0.2 x 1.3 x 2.5 for PO values (Bonetti & Hopkins,

2009), and 3) a small standardized effect based on Cohen's effect size (ES) principle (0.2 x between-athletes standard deviation [Hopkins et al., 2009]) for other parameters. Accordingly, the SWC was determined to be 0.3% in performance time, and 0.7% in PO. Quantitative chances of higher or lower values than the smallest worthwhile change were evaluated qualitatively as follows: <1%, almost certainly not; 1%–5%, very unlikely; 5%–25%, unlikely; 25%–75%, possible; 75%–95%, likely; 95%–99%, very likely; and >99%, almost certain. If the chance of higher or lower values was >5%, the true difference was assessed as *unclear*. Otherwise, we interpreted that change as the observed chance (Hopkins et al., 2009). The practical interpretation of an effect was deemed *unclear* when the 90% confidence interval (CI) of standardized change/difference included zero (Hopkins et al., 2009). All values are presented as means ± standard deviation (SD).

RESULTS

Training and Control data

Changes in training data are shown in Table 1.

Training parameters. At baseline, between-group differences in training volume (TV) were unclear (Table 1). All three groups displayed trivial changes phases II to III in the overall TV. During the taper, the three groups showed an almost certain very large decrease in the overall TV (ES: control, -3.62 ± 0.41; HA-L, -3.23 ± 0.31; HA-H, -2.93 ± 0.38).

Training load. During phase III, the HA-L and HA-H groups showed likely small and almost certain moderate increases in TL (ES: 0.28 ± 0.13 and 0.73 ± 0.19, respectively), with a likely small between-group difference in changes (ES: 0.35 ± 0.22). During phase IV, all three groups displayed almost certain very large decreases in TL (ES: control, -2.43 ± 0.20; HA-L, -3.18 ± 0.33; HA-H, -4.18 ± 0.69), with likely greater decreases in TL for the HA-L

and HA-H groups as compared to the control group (ES: -0.65 ± 0.39 and -0.78 ± 0.63, respectively).

Delayed onset muscle soreness. DOMS scores showed a possible small decrease after the tapering period for the three groups (ES: control, -0.33 ± 0.31; HA-L, -0.43 ± 0.48; HA-H, -0.29 ± 0.25), with unclear between-group differences for all phases.

Perceived Fatigue. Within-group differences in fatigue scores showed a likely moderate increase after phase III for the HA-H group (ES: 0.61 ± 0.45), but unclear changes for the control and HA-L groups (ES: 0.00 ± 0.45 and 0.14 ± 0.85, respectively). After the taper, the HA-H group displayed a likely moderate reduction in fatigue scores (ES: -0.93 ± 1.03).

Time-trial data

Changes in average PO and TT duration from Pre to Post are shown in Figure 2.

Average power output. Between-group differences were unclear at baseline. From Pre to Mid, the control group displayed unclear changes in PO (ES: -1.3 ± 1.8). In contrast, the HA-L group showed a very likely very large increase in PO (ES: 6.7 ± 4.6), while the HA-H group demonstrated a very likely large decrease in PO (ES: -4.9 ± 3.5). The difference in PO at Mid between the HA-H and the HA-L groups was very likely very large.

From Mid to Post, all three groups displayed a greater PO, which was likely small for the control group (ES: 1.8 ± 1.3), very likely large for the HA-L group (ES: 6.4 ± 3.9) and very likely very large for the HA-H group (ES: 7.6 ± 3.1). These increases were very likely larger for the HA-L and HA-H groups compared to the control group with unclear differences against each other.

From Pre to Post, the control and the HA-H groups demonstrated unclear within-group changes (ES: 1.3 ± 3.8 and 2.3 ± 6.0, respectively) and unclear between-group difference in

changes (ES: 1.6 ± 6.9) in PO, while the HA-L group showed an almost certain extremely large increase in PO (ES: 14.5 ± 4.4).

TT duration. Between-group differences were unclear at baseline. From Pre to Mid, the control group displayed unclear changes in performance (ES: -0.2 ± 1.5). In contrast, the HA-L group showed an almost certain large decrease in TT duration (ES: -2.2 ± 1.1), while the HA-H group demonstrated a very likely large increase in TT duration (ES: 1.7 ± 1.3). From Mid to Post, decreases in TT duration were very likely large for the HA-L group (ES: -2.3 ± 1.5), and almost certainly large for the HA-H group (ES: -2.5 ± 1.0), with unclear between-group differences in changes. From Pre to Post, only the HA-L group showed a reduction in TT duration, which was almost certainly very large (ES: -4.4 ± 1.3).

Pacing. The temporal changes in PO for each TT are shown in Figure 3. For reading clarity, only within-group differences in PO per 5-km split across the protocol are highlighted in the figure. Within-trial differences during Pre, Mid and Post systematically showed for the three groups to increase PO from 10-15 to 15-20 km. In addition, the HA-L group displayed moderate decreases in PO at Pre and Mid from 0-5 to 5-10 km and from 5-10 to 10-15 km, which were noticeable at Post in all three groups. Between-group differences in PO per 5-km split highlighted reduced PO for the HA-H group at Mid from 0-5 to 10-15 km as compared to the control and HA-L groups. At post, the HA-L group showed greater PO from 0-5 to 10-15 km as compared to the other groups.

Heart rate. Warm-up: both the HA-L and the HA-H groups showed possible small decreases in HR from Pre to Mid (-2 ± 5 bpm, ES: -0.24 ± 0.26, and -5 ± 5 bpm, ES: -0.26 ± 0.16, respectively), with a possible small between-group difference in changes (ES: 0.20 ±

0.26). The HA-H group also presented a possible small increase in HR values from Mid to Post (3 ± 3bpm, ES: 0.20 ± 0.11).

Time-trial: within-group difference in mean HR values showed trivial changes for all three groups from Pre to Mid, and very likely moderate increases from Mid to Post for the HA-L (6 ± 5 bpm, ES: 0.63 ± 0.28) and the HA-H (8 ± 6 bpm, ES: 0.84 ± 0.37) groups, with a possible small between-group difference in changes (ES: 0.23 ± 0.43). Within-group difference in HRmax revealed a very likely small reduction in the HA-H group from Pre to Mid (-4 ± 2 bpm, ES: -0.43 ± 0.14) and increases from Mid to Post both in the HA-H (7 ± 5 bpm, ES: 0.72 ± 0.31) and the HA-L (5 ± 3 bpm, ES: 0.43 ± 0.63) groups, with a possible small between-group difference in changes (ES: 0.27 ± 0.39).

Body mass loss. At least possible small increases in the post-TT change in BML were observed both for the HA-L and HA-H groups from Pre to Mid (ES for the warm-up: HA-L, 0.51 ± 0.83; HA-H, 0.51 ± 0.67; TT: HA-L, 0.20 ± 0.23; HA-H, 0.54 ± 0.26) and from Mid to Post (ES for the warm-up: HA-L, 0.95 ± 0.69; HA-H, 0.34 ± 0.57; TT: HA-L, 0.24 ± 0.17; HA-H, 0.20 ± 0.29). Between-group difference in changes revealed likely greater changes in BML during the TT from Pre to Mid in the HA-H group (ES: 0.40 ± 0.42).

Core temperature. Between-group differences in T_{core} were systematically unclear upon arriving at each testing session. Within-group difference showed likely small reduction in T_{core} elevation from Mid to Post during the warm-up phases both for the HA-L and HA-H groups (ES: -0.50 ± 0.72 and -0.44 ± 0.70, respectively) with unclear between-group difference in changes. For each group, unclear differences were found in T_{core} elevation between the TTs.

RPE during the time-trial. A possible small increase in RPE was found for the HA-H group from Pre to Mid (0.4 ± 1.1 on the 6-20 scale, ES: 0.24 ± 0.44). Further within-group differences in RPE were unclear.

DISCUSSION

This study investigated real life scenario of HA by simulating either individualised high- or low-intensity HA training sessions and a taper phase to improve endurance performance in the heat. The main finding was that the performance response was maximized in the low-intensity HA protocol (i.e., improvement both before and after the taper) while the high-intensity HA protocol resulted in symptoms often noted in a functional overreaching (F-OR) state i.e., reduced performance and weak supercompensation during tapering. Despite similar physiological responses between respective HA protocols, only the HA-L resulted in overt time trial improvements. Accordingly, the beneficial psychophysiological responses from HA were seemingly counterbalanced by the training FOR-like maladaptations following high-intensity training in the heat.

Heat-acclimation and endurance performance

A key outcome of any HA protocol is to ensure appropriate psycho-physiological adaptations to the heat (Périard et al., 2015). In the present study, physiological adjustments of the both HA groups were in agreement with classic HA-induced exercise responses in the heat i.e., reduced sub-maximal HR (from plasma volume expansion) and T_{core} (from enhanced blood flow distribution and evaporative cooling rate), and an increased BML (from earlier and increased sweat loss) following exercise (Nielsen et al., 1993; Sawka et al., 2011). Whilst both groups showed congruent heat adaptations – similar to Houmard et al., (1990) but divergent from the control group which displayed no changes – no between-group differences

existed in BML or T_{core}. In explanation, could be the fact that heat exposures during HA were of similar duration and/or that the intensity of the warm-up during testing was not high enough to elicit discrepancies in response to heat stress (Taylor et al., 2006).

In contrast, incongruent performance responses between HA groups were observed. Specifically, training then tapering in the heat appeared ergogenic (cumulative gains in TT duration) for the HA-L group, which is coherent with the beneficial effects of both submaximal HA and tapering on endurance performance (Bosquet et al., 2007; Tyler et al., 2016). In particular, tapering allows the athlete to recover from prior heavy training load (i.e., increased heat load-related fatigue in the present study) while avoiding detraining (Bosquet et al., 2007). Of note, the magnitude of improvement at Mid (i.e., after a 5-day HA) in the HA-L group was not as pronounced as that reported by Wingfield et al. (2016) (-2.2 ± 1.1 vs. -5.9 ± 7.1%), possibly due to heat exposure durations (60 $min \cdot d^{-1}$ vs. 90 $min \cdot d^{-1}$, respectively) and/or participants' fitness level (well-trained vs. recreational participants, respectively). On the contrary, a novel and unexpected observation is the large increase in TT duration at Mid following HA-H. More precisely, the present study shows that when giving priority to high-intensity exercise, HA has the potential to impair performance in the heat to such an extent that it is not overcompensated following a one-week taper phase, and ultimately reaches the same performance level as a control group.

For the HA-L group, the corresponding increase in PO at Mid displayed within an up-regulated pacing strategy throughout the TT. The greater PO noticed during the second part of the TT is a well-known observation attributed to improved heat loss mechanisms (Racinais et al., 2015; Sawka et al., 2011), however the increased PO from TT start may speculatively relate to a higher confidence following HA. That is, experience is widely reported to be a powerful regulator of energy expenditure (Micklewright et al., 2010) and, relative to multiple training sessions in the heat, may provide participants with important cognitive substrates to

optimizing pacing. However, the progressive perceptual influence of heat exposures remains to clarify since the control group did not report pacing improvement after two anterior TT (Fam 2 + Pre). Contrastingly, the HA-H group showed a ~5% decrease in PO at Mid which was effective throughout the TT and until the endspurt, as was the pacing alteration recently reported in subjects temporarily led to overreaching using a 6-day training overload (Skorski et al., 2015). In particular, in the present study eight of the nine HA-H participants revealed performance decline, a finding that highlights the consistent harmful effect of an acute moderate increase of the internal TL. Since such homogenous response is anecdotal in studies reporting athletes led to F-OR (e.g., Aubry et al., 2014), this observation opens the door to protocols differentiating internal vs. external TL manipulation to investigate performance changes and optimize training regimen. Consequently, despite notion of HA-H being more ecologically valid than HA-L, it seems that high-intensity training in the heat in well-trained athletes was not beneficial to performance.

Performance decrements in HA-H: a role for F-OR?

Given the limited literature on performance outcomes following HA-H, mechanistic explanations of the slower TT outcomes may borrow from F-OR literature; especially given the HA-H group concomitantly revealed typical signs of F-OR at Mid that dissipated during the taper phase. As context, F-OR athletes are often characterized by altered perceptual and cardiovascular responses to all exercise intensities (Decroix et al., 2016; Le Meur et al., 2013). The present results show that HA-H was associated to a moderate increase in TL and leads to an inability to reach HRmax during the TT (at Mid), two observations that were not observed in the HA-L group. In addition, greater decreases in HR values were found during the steady exercise in the HA-H group at Mid compared to the HA-L group, a delta that eclipsed consecutive to the taper. Such descriptions have previously been evidenced in athletes

temporarily led to an overreached state from excessive increases in training volume (Le Meur et al., 2013, 2014), and the increased internal loads reported here during HA-H may provide indirect support of these hypotheses.

Practical recommendations for athlete's monitoring during HA

Even when programing a taper phase prior to the competition to dissipate the accumulated fatigue, coaches should remain aware that HA-H increases the risk of F-OR and should therefore be scheduled away from preliminary heat exposures. Accordingly, low-intensity training sessions in the heat are more appropriate to maximise performance improvement when a one week HA procedure is scheduled prior to a taper phase. However, high-intensity training session could remain of interest after initial adaptations i.e., following 4-5 days of HA-L (Périard et al., 2015). In this perspective, the effectiveness of progressive intensity HA i.e., prioritizing low- prior to high-intensity training sessions, remains to investigate.

CONCLUSION

It is currently admitted that performance improvement in the heat is an indices of HA (Sawka et al., 2011; Tyler et al., 2016). This study demonstrates that the phenotype of HA is not systematically synonymous of performance improvement. In particular, in the specific ecological context of high-level athletes, the present results show that high-intensity HA scheduled on the basis of athlete's usual training program is effective at inducing physiological HA (i.e., improved HR and BML responses to exercise) but does *not* represent an ergogenic intervention for endurance performance in the heat even if a 1-week taper phase is programmed to dissipate the accumulated fatigue. In fact, such protocol applied to non-HA athletes induced signs of overreaching without consecutive performance supercompensation. Within this context, combining athlete's monitoring during training sessions in temperate

conditions to HA-related psychophysiological responses (e.g., wellness scores, TL, HR, sweat loss, T_{core}) could be relevant to adjust training load during HA without leading athletes to overreaching. In this perspective, high-intensity training sessions would take their full meaning within longer HA protocols, consecutive to more tolerable, submaximal exercise intensities.

ACKNOWLEDGMENTS

We receive funding for research on which this article is based from the French National Institute of Sport, Expertise and Performance (Paris). The authors report no conflict of interest, and would like to thank Guillaume Renard for his support in driving the study.

REFERENCES

Aubry, A., Hausswirth, C., Louis, J., Coutts, A. J., & Le Meur, Y. (2014). Functional overreaching: the key to peak performance during the taper?. *Medicine and science in sports and exercise*, *46*(9), 1769-1777.

Barnett, A., & Maughan, R. J. (1993). Response of unacclimatized males to repeated weekly bouts of exercise in the heat. *British journal of sports medicine*, *27*(1), 39-44.

Bosquet, L., Montpetit, J., Arvisais, D., & Mujika, I. (2007). Effects of tapering on performance: a meta-analysis. *Medicine and science in sports and exercise*, *39*(8), 1358.

Chalmers, S., Esterman, A., Eston, R., Bowering, K. J., & Norton, K. (2014). Short-term heat acclimation training improves physical performance: a systematic review, and exploration of physiological adaptations and application for team sports. *Sports Medicine*, *44*(7), 971-988.

Decroix, L., Piacentini, M. F., Rietjens, G., & Meeusen, R. (2016). Monitoring Physical and Cognitive Overload During a Training Camp in Professional Female Cyclists. *International journal of sports physiology and performance*.

De Pauw, K., Roelands, B., Cheung, S. S., De Geus, B., Rietjens, G., & Meeusen, R. (2013). Guidelines to classify subject groups in sport-science research. *Int J Sports Physiol Perform*, *8*(2), 111-122.

Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, Doleshal P, Dodge C. A new approach to monitoring exercise training. *J Strength Cond Research*. 2001; 15(1): 109-115.

Foster C, Hendrickson KJ, Peyer K, et al. Pattern of developing the performance template. *Br J Sports Med*. 2009; 43(10): 765–9.

Gaoua, N., Periard, J.D., Wilson, M.G., Racinais, S. Hyperthermia-induced alteration in complex cognitive task performance is reversed by 11 days of heat acclimation. Abstract ECSS 2015.

Guy, J. H., Deakin, G. B., Edwards, A. M., Miller, C. M., & Pyne, D. B. (2015). Adaptation to hot environmental conditions: an exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. *Sports Medicine*, *45*(3), 303-311.

Helton, W. S., Head, J., & Russell, P. N. (2011). Reliable-and unreliable-warning cues in the Sustained Attention to Response Task. *Experimental brain research*, *209*(3), 401-407.

Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc*. 2009; 41: 3–13.

Houmard, J. A., Costill, D. L., Davis, J. A., Mitchell, J. B., Pascoe, D. D., & Robergs, R. A. (1990). The influence of exercise intensity on heat acclimation in trained subjects. *Medicine and Science in Sports and Exercise*, *22*(5), 615-620.

Lee, J. K., Nio, A. Q., Lim, C. L., Teo, E. Y., & Byrne, C. (2010). Thermoregulation, pacing and fluid balance during mass participation distance running in a warm and humid environment. *European journal of applied physiology*, *109*(5), 887-898.

Le Meur, Y., Louis, J., Aubry, A., Guéneron, J., Pichon, A., Schaal, K., ... & Hausswirth, C. (2014). Maximal exercise limitation in functionally overreached triathletes: role of cardiac adrenergic stimulation. *Journal of applied physiology*, *117*(3), 214-222.

McVay, J. C., & Kane, M. J. (2010). Does mind wandering reflect executive function or executive failure? Comment on Smallwood and Schooler (2006) and Watkins (2008).

Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol*. 1993; 460(1): 467-85.

Nybo, L. (2012). Endurance training and competition in the heat. In *Endurance Training*. Iñigo Mujika.

Paton CD, Hopkins WG. Variations in performance of elite cyclists from race to race. *Eur J Sport Sci*. 2006; 6(1): 25-31.

Périard, J. D., Racinais, S., & Sawka, M. N. (2015). Adaptations and mechanisms of human heat acclimation: applications for competitive athletes and sports. *Scandinavian journal of medicine & science in sports*, *25*(S1), 20-38.

Petersen, C. J., Portus, M. R., Pyne, D. B., Dawson, B. T., Cramer, M. N., & Kellett, A. D. (2010). Partial heat acclimation in cricketers using a 4-day high intensity cycling protocol. *Int J Sports Physiol Perform*, *5*(4), 535-45.

Piper BJ, Li V, Eiwaz MA, Kobel YV, Benice TS, Chu AM, Olson R, Rice D, Gray H, Mueller ST, Raber J. Executive function on the psychology experiment building language tests. Behavior Research Methods. 2012; 44(1):110–123. [PubMed: 21534005]

Racinais, S., Buchheit, M., Bilsborough, J., Bourdon, P. C., Cordy, J., & Coutts, A. J. (2014). Physiological and performance responses to a training camp in the heat in professional Australian football players. *International Journal of Sports Physiology & Performance*, *9*(4).

Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. *Med Sports Sci Exer*. 2015;47: 601-606.

Sawka, M. N., Leon, L. R., Montain, S. J., & Sonna, L. A. (2011). Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. *Comprehensive Physiology*.

Schmit, C., Le Meur, Y., Duffield, R., Robach, P., Oussedik, N., Coutts, A. J., & Hausswirth, C. (2015). Heat‐acclimatization and pre‐cooling: a further boost for endurance performance?. *Scandinavian journal of medicine & science in sports*.

Schmit, C., Duffield, R., Hausswirth, C., Coutts, A. J., & Le Meur, Y. (2015). Pacing Adjustments Associated With Familiarisation: Heat vs. Temperate Environments. *International journal of sports physiology and performance*.

Skorski, S., Hammes, D., Schwindling, S., Veith, S., Pfeiffer, M., Ferrauti, A., ... & Meyer, T. (2015). Effects of training-induced fatigue on pacing patterns in 40-km cycling time trials. *Medicine and science in sports and exercise*, *47*(3), 593-600.

Taylor, N. A. (2014). Human heat adaptation. *Comprehensive Physiology*.

Taylor, N. A., & Cotter, J. D. (2006). Heat adaptation: Guidelines for the optimisation of human performance. *International SportMed Journal*, *7*(1).

Tyler, C. J., Reeve, T., Hodges, G. J., & Cheung, S. S. (2016). The Effects of Heat Adaptation on Physiology, Perception and Exercise Performance in the Heat: A Meta-Analysis. *Sports Medicine*, 1-26.

Vaile J, Halson S, Gill N, Dawson B. Effect of cold water immersion on repeat cycling performance and thermoregulation in the heat. *J Sports Sci*. 2008; 26: 431–40.

Wingfield, G. L., Gale, R., Minett, G. M., Marino, F. E., & Skein, M. (2016). The effect of high versus low intensity heat acclimation on performance and neuromuscular responses. *Journal of thermal biology*, *58*, 50-59.

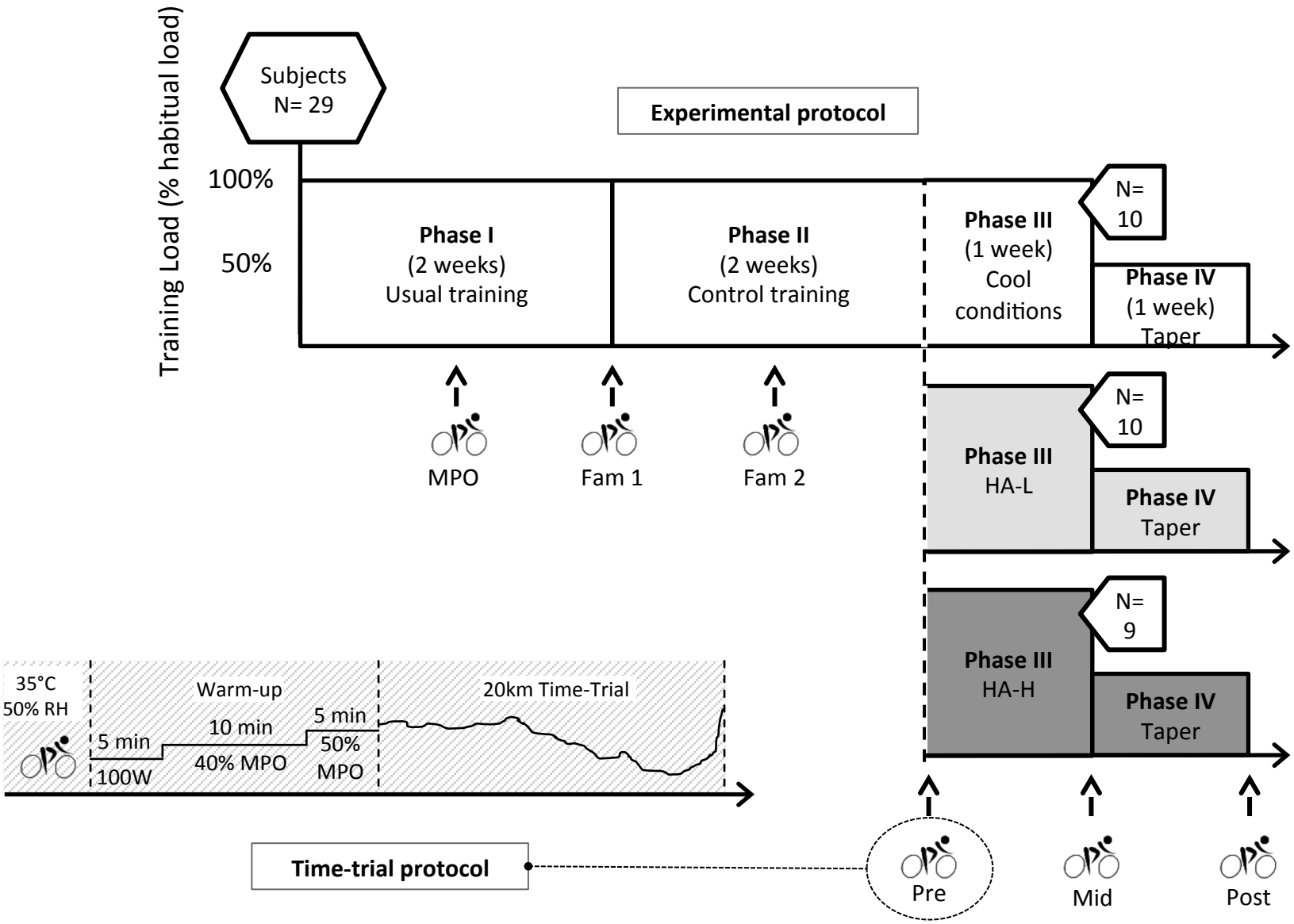


Figure 1. Schematic representation of the experimental and time-trial protocols.
Notes. MPO = maximal power output; Fam = familiarisation session; HA-L = heat-acclimation at low exercise intensity; HA-H = heat-acclimation at high exercise intensity.

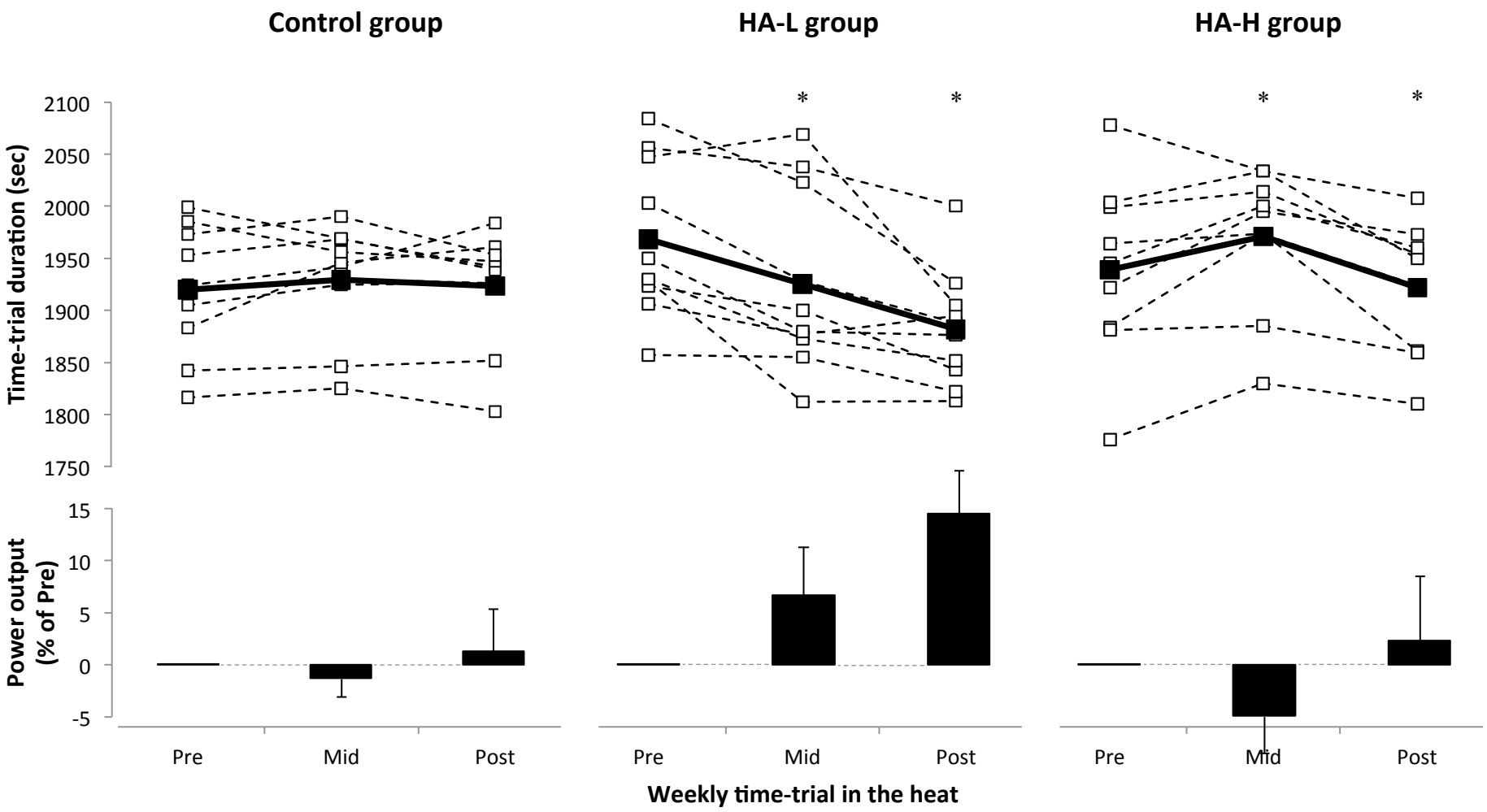


Figure 2. Individual and average time-trial duration, and corresponding relative changes in average power output for each group and for the three trials of the experimental protocol.
Notes. HA-L = heat-acclimation at low exercise intensity; HA-H = heat-acclimation at high exercise intensity. The thick line represents the group average.
* changes at least very likely large from the precedent trial.

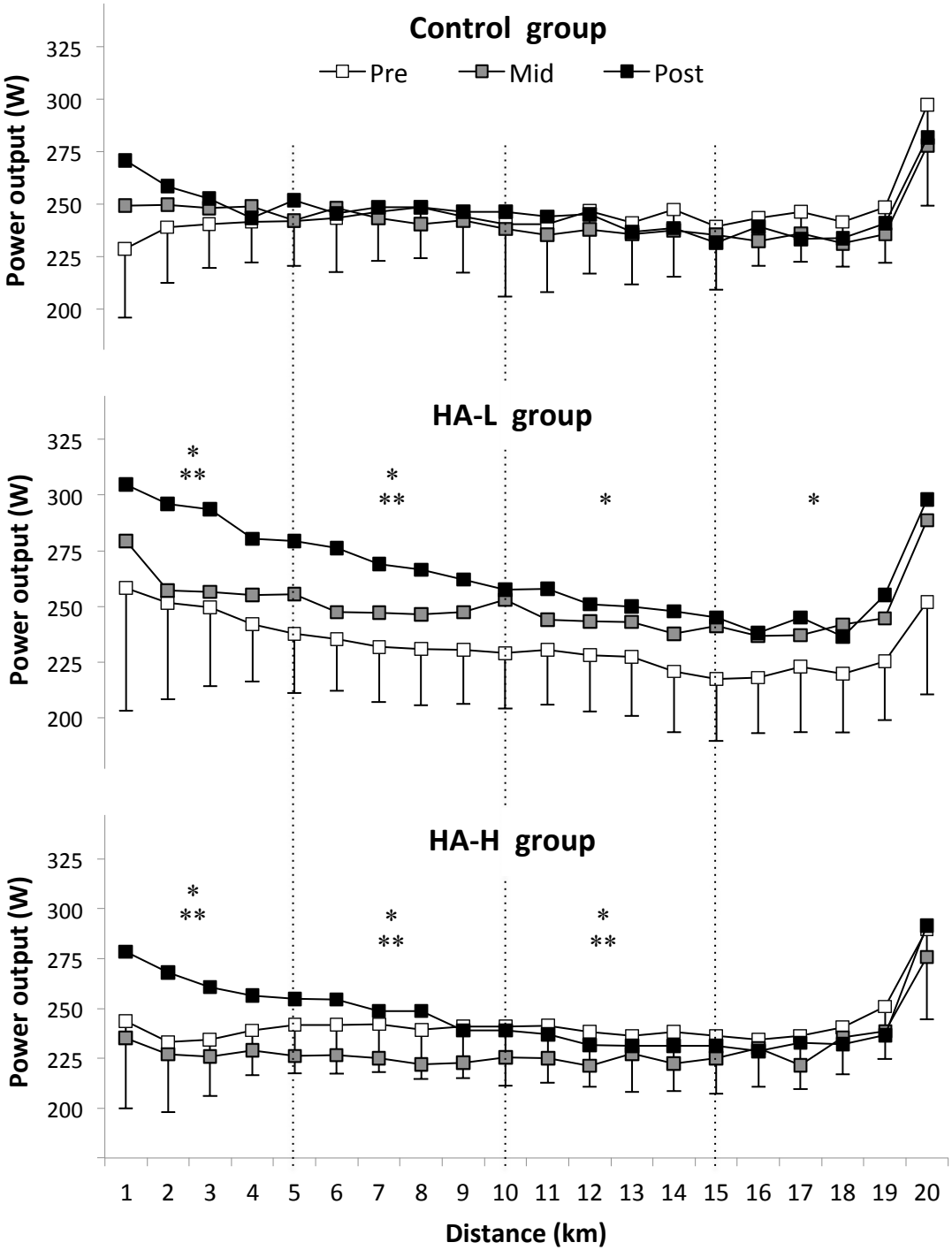


Figure 3. Power output per kilometre for each group and for the four time-trials in the heat.
Results are presented as the group mean ± SD. For clarity, only error bars of Pre are presented. Errors bars of Mid and Post were within the same range.
Notes. HA-L = heat-acclimation at low exercise intensity; HA-H = heat-acclimation at high exercise intensity.
* changes at least likely large between Pre and Mid; ** changes at least likely moderate between Mid and Post.
Between-groups differences in changes and within-trials changes are presented in the Results (*Testing data* section).

TRAVAIL DE THÈSE

Schmit, C., Hausswirth, C., Le Meur, Y., & Duffield, R. (2016). Cognitive functioning and heat strain: performance responses and protective strategies. *Sports Medicine*.

Résumé en français

En dépit de recherches prédominantes sur la performance physique en conditions chaudes, de nombreuses activités requièrent un fonctionnement cognitif optimal dans une optique de performance (*i.e.,* prise de décision) et/ou de santé (*i.e.,* risque de blessure). Les périodes prolongées d'activités cognitives sollicitantes, ou de fatigue induite par l'exercice, induisent une détérioration du fonctionnement cognitif. L'ajout de conditions environnementales chaudes exacerbe cette détérioration et impactent donc négativement la performance globale. Le présent document tente d'extraire de la littérature croisant 'chaleur' et 'cognition' des éléments consistants permettant d'explorer la nature de la performance cognitive dépendamment du niveau de stress thermique rencontré. Plus spécifiquement, les études investiguant la performance cognitive en conditions d'hyperthermie, souvent via la réalisation de tests sur ordinateur (tâches cognitives), sont utilisés pour mieux cerner la relation entre charge thermique interne et performance cognitive. L'existence d'une relation en U-inversé entre le développement d'un état d'hyperthermie et la performance cognitive est suggéré, et met en lumière qu'une température centrale proche de 38,5°C pourrait potentiellement représenter une 'seuil' discriminant les incidences cognitives positives vs. négatives de la chaleur.

Ce travail de thèse a permis de compléter la cartographie des effets de la chaleur sur les performances humaines appréhendée jusqu'ici, en caractérisant spécifiquement les incidences cognitives d'une élévation de la température centrale. Ce travail ouvre la voie à de futures études qui permettront d'investiguer plus-avant les interactions psycho-physiologiques.

Sports Med
DOI 10.1007/s40279-016-0657-z

REVIEW ARTICLE

Cognitive Functioning and Heat Strain: Performance Responses and Protective Strategies

Cyril Schmit[1] · Christophe Hausswirth[1] · Yann Le Meur[1,2,3] · Rob Duffield[4]

Abstract Despite the predominance of research on physical performance in the heat, many activities require high cognitive functioning for optimal performance (i.e. decision making) and/or health purposes (i.e. injury risk). Prolonged periods of demanding cognitive activity or exercise-induced fatigue will incur altered cognitive functioning. The addition of hot environmental conditions will exacerbate poor cognitive functioning and negatively affect performance outcomes. The present paper attempts to extract consistent themes from the heat–cognition literature to explore cognitive performance as a function of the level of heat stress encountered. More specifically, experimental studies investigating cognitive performance in conditions of hyperthermia, often via the completion of computerised tasks (i.e. cognitive tests), are used to better understand the relationship between endogenous thermal load and cognitive performance. The existence of an inverted U-shaped relationship between hyperthermia development and cognitive performance is suggested, and highlights core temperatures of ~38.5 °C as the potential 'threshold' for hyperthermia-induced negative cognitive performance. From this perspective, interventions to slow or blunt thermal loads and protect both task- and hyperthermia-related changes in task performances (e.g. cooling strategies) could be used to great benefit and potentially preserve cognitive performance during heat strain.

✉ Cyril Schmit
cyril.schmit@gmail.com

1 Laboratory of Sport, Expertise and Performance (EA 7370), Research Department, French National Institute of Sport, Expertise and Performance (INSEP), 11, Avenue du Tremblay, 75 012 Paris, France

2 Medical Department, AS Monaco Football Club, Monaco, Monaco

3 Laboratory LAMHESS (EA6312), University of Nice Sophia-Antipolis, Nice, France

4 Sport and Exercise Discipline Group, Faculty of Health, University of Technology Sydney (UTS), Moore Park, NSW, Australia

Key Points

- An inverted U-shaped response exists for cognitive performance as related to increasing hyperthermia.
- Initial increases in core temperature up to ~38.5 °C improve cognitive functioning, but after ~39.0 °C impairments start to appear as a function of task complexity.
- The heat–cognition interaction may be explained as an inability to deal with concomitant heat- and task-related constraints.
- Nascent reports suggest this inverted U-shaped pattern is modifiable using typical strategies (e.g. cooling, hydration) so as to promote heat-related cognitive enhancement and reduce the debilitative effect of heat strain on cognitive performance.

1 Introduction: Cognitive Performance in the Heat

The interaction between exercise in the heat and performance decrement (compared with temperate conditions) is often presented in a dose-response fashion, with greater heat exposure further limiting exercise duration [1–4]. This relationship is classically explained by the progressive

Published online: 17 December 2016

exacerbation of cardiovascular, thermoregulatory, metabolic and perceptual functioning [5]. Briefly, the augmented skin blood flow accompanying elevated skin (T_{skin}) and core (T_{core}) temperatures poses additional challenges to cardiac strain and working muscles while amplifying perceived exertion [6]. Increased ventilation accompanying T_{core} elevation may also decrease cerebral blood flow (CBF) and incur inadequate substrate delivery to, and heat removal from, the brain [7]. Alongside these exacerbated perceptual and physiological responses from exercising in hot conditions, increased cognitive strain (i.e. the challenge of operating cognitive operations) is present [8].

Despite predominant research focussing on physical performance in the heat, many prolonged exercise events require maintenance of high cognitive functioning for performance (i.e. decision making [9]) and/or health purposes (i.e. injury risk [10, 11]). Indeed, in circumstances of goal-directed actions, outcome-based consequences rely on the ability to repeatedly and accurately regulate behaviour according to this goal. For example, athletes may be required to initiate immediate behavioural adjustments in response to a direct opponent (e.g. in dual sports), alter a pacing strategy (e.g. in prolonged efforts), encode and attend to a steady target (e.g. in shooting actions), interpret specific proprioceptive information (e.g. in natural outdoor settings), or distinguish between unfair and fair moves (e.g. referee decision making). Such a collection of circumstances shows the direct need for optimal cognitive functioning during any athletic endeavour. Prolonged periods of demanding cognitive activity (i.e. mental fatigue) or exercise-induced fatigue will result in impaired cognitive functioning [12, 13]. The addition of hot environmental conditions (i.e. heat stress) will exacerbate poor cognitive functioning and negatively affect performance outcomes [14]. Accordingly, a further understanding of the effect of environmental-induced heat strain (i.e. the heat-related personal constraint) on cognitive functioning is of importance to discriminate and potentially protect the ability to adopt optimal goal-directed behaviour during exercise.

Cognitive performance reflects the accuracy and efficiency of subcortical and cortical processes, allowing external stimuli detection and identification, response selection, preparation and activation [15]. Therefore, specific to sports performance, cognitive functioning refers to the ability to remain lucid during the strain of exercise. Behavioural response speed (i.e. reaction time) and accuracy (i.e. error rate) from test batteries are most commonly used as measures of information processing efficiency, whilst test specificity then allows inferences about specific cognitive operations (e.g. working memory—keep information in a quickly retrievable state; selective inhibition—deliberately inhibit an impulsive response when necessary). Of the cognitive operations assumed to be tested, working memory and vigilance tasks are considered the most demanding (i.e. relative to effort and glucose and oxygen consumption), more so than dual tasks or tracking, and perceptual-motor and simple reaction time tests, respectively [16, 17]. Thus, task selectivity and measures enable access to behavioural outcomes whose changes in performance level then allow inferences about changes in cognitive functioning (e.g. improved cognitive control, increased impulsivity, impaired attentional focus). Accordingly, the use of behavioural outcomes during cognitive tasks is useful to (i) provide an inexpensive, easy-to-use outcome to complement neuroimagery techniques (e.g. electroencephalography [EEG]); (ii) allow distinction to be made between, and within, motor and upstream stages of response processes when combined with electromyography (EMG); and (iii) allow observations of *functional* brain responses (as opposed to resting cognitive activity). The abovementioned outcomes make cognitive performance particularly pertinent to enable further consideration of processes, leading to greater cognitive fatigue and impaired goal-directed behaviour in the heat.

In hyperthermic conditions i.e. when T_{core} rises, a number of responses are evident, including (i) cognitive functions slow before physiological variables reach critical limits; and (ii) the most effortful/complex cognitive computations are impaired earlier [18]. Furthermore, it has been suggested that both the magnitude of heat strain and cognitive task complexity interact to predict cognitive efficiency; such that the more stressful the combination of hyperthermia *and* cognitive demands, the larger the cognitive impairment [19, 20]. Although it is intuitive that this relationship (heat strain and cognitive function) parallels the aforementioned dose-dependent effect of heat stress on endurance performance decrement, it is yet to be conclusively shown [21]. For example, some studies have reported no effect or positive cognitive performance outcomes in varying conditions of hyperthermia [8, 22]. If this is the case, it potentially makes the exacerbated thermal load during exercise in the heat relevant not just to physical performance but also to cognitive performance for athletes, and precautionary behavioural applications become even more important.

To date, due to its multifaceted dimension (dynamic conception of cognitive and physiological strains in the heat), the maximum adaptability model [23] represents the most established rationale to explain the heat–cognition response relationship. Based on empirical reports, the model suggests cognitive improvement with initiation of hyperthermia, resulting from adaptive strategies to heat strain, such as increased attentional focus. However, as hyperthermia develops, heat strain-induced depletion of attentional resources (expressed in terms of dynamic body T_{core} increase) leads to cognitive performance decrement as

a function of cognitive task complexity [23]. Despite growing interest in understanding hyperthermia-related physiological changes [24], this model has not been updated to date [25]; however, the recent development of innovative technologies that enables better tracking of cognitive processes (e.g. functional magnetic resonance imaging [fMRI]; EMG applied to stimulus-response tasks) could challenge initial empirical reports. For instance, in temperate conditions, Ando et al. [26] and Grego et al. [27] highlighted discrepancies between behavioural and near-infrared spectroscopy and EEG indices of cognitive performance, respectively. Therefore, such observation suggests that the recent deeper monitoring of brain activity could refine empirical reports from cognitive tasks in the understanding of brain activity under increased heat stress.

Within this context, the present paper aims at extracting consistent themes from the recent heat–cognition literature to understand, and ultimately provide guidance for, cognitive performance as a function of the level of heat stress encountered. A three-step rationale for this possible relationship (together with potential explanatory mechanisms) is presented, a summary model of cognitive efficiency under heat stress is then proposed, and, finally, the potential practical implications are outlined.

2 Cognitive Responses During Hyperthermia

2.1 Initial Increases in Core Temperature and Related Cognitive Benefits

Irrespective of task complexity, cognitive functions initially improve when body heat accumulation elevates T_{core}. Indeed, simple reaction times are reported as faster once T_{core} begins to increase in situations of heat stress, highlighting a speeding effect of increased heat load on low-demanding tasks [28]. Greater performances are also reported during complex computations performed when passive heating (heat exposure in a resting state) induces a small increase in T_{core} ($\sim$38.2 °C) [29]. Specifically, Simmons et al. [29] found faster responses on both simple reaction time, choice reaction time and complex (vigilance) cognitive tests, which suggest that a rise in body temperature induces a *systematic* improvement in the capacity to intensively, but punctually, focus and respond to a stimulus. The possibility of an initial cognitive improvement has more recently been reinforced by reports of small increases in T_{core} ($\sim$38.0 °C), resulting in improved working memory task and psychomotor vigilance performances [8, 30]. Interestingly, in the study by Bandelow et al. [8], block-tapping test performance improved when T_{core} was elevated to $\sim$38.2 °C, a temperature beyond which the heat-induced beneficial effect disappeared (see Sect. 2.2 for further discussion). Schlader et al. [31] provided further evidence for this initial heat-related acceleration of central processing for both attention and executive control tasks when T_{core} was passively elevated in the range of $\sim$1 °C. Thus, it is evident that small increases in endogenous heat accumulation result in an improvement in a variety of cognitive task performances.

The mechanisms responsible for this cognitive improvement may be plural and intertwined. First, they could relate to a decrease in thermal comfort (i.e. the sensation of pleasantness related to heat), leading individuals to perceive greater difficulty in interpreting and responding to a given demand, compared with colder conditions [5]. As a consequence, the athlete may deliberately invest a greater amount of cognitive effort into task completion to compensate (or even overcompensate) for the increased perception of difficulty induced by environmental conditions [32]. In addition, the heat-related cognitive benefits could relate to the arousal effect derived from endogenous thermoregulatory control mechanisms occurring during heat exposure [33, 34]. In particular, the massive projections from the dopaminergic pathways to the limbic system and the prefrontal cortex (PFC) could benefit from changes in neurotransmitters and hormonal activity and accelerate the cognitive computations [35]. Of note, this arousing effect is also reported to account for exercise-related enhanced cognitive performance in colder conditions [15] and could therefore be argued to mediate hyperthermia-related improved performance during heat- and exercise-induced hyperthermia (i.e. active heating) [8]. Cognitive improvement during modest passively-induced hyperthermia seems a consistent finding, while post-exercise-related cognitive responses differ depending on the type of cognitive task undertaken [36]. Thus, when moderate hyperthermia is induced through exercise, exercise-related arousal may only partially contribute to cognitive performance improvement. An additional explanation for the observed enhancement of cognitive processing following small increases in T_{core} relates to the Van't Hoff Q_{10} effect. Similar to its metabolic effect at the muscular level, the Q_{10} effect may account for the increased cerebral metabolic rate of oxygen ($CMRO_2$, an index of the rate of brain oxygen consumption) [37] observed with T_{core} elevation [24], and thus mediate cognitive functioning efficiency. This proposal is supported both by age- and exercise-cognition studies reporting parallel variations in $CMRO_2$ and cognitive performances [38, 39]. Other potential candidates that could support the positive cognitive effect of modest hyperthermia include changes in regional CBF and substrate availability (i.e. an increase in CBF augmenting brain glucose delivery [40]), as well as blood–brain barrier permeability and circulating brain-derived neurotrophic factor [30].

Regardless of the multiple, although speculative, explanatory mechanisms, it seems a slight increase in T_{core} (~38.2 °C) improves cognitive performance. Importantly, both simple and complex cognitive processes are enhanced with initial increases in T_{core}, regardless of whether passive or active in nature, suggesting no harmful cognitive consequences for athletes under limited heat stress or from normal pre-exercise behaviours, i.e. warm-up.

2.2 A Heat-Induced Threshold for the Reduction in Cognitive Performance?

Noticing this initial positive effect on cognitive function induced by a passive mild T_{core} elevation, Gaoua et al. [22] remarked that cognitive performance enhancement ceased once T_{core} reached ~38.7 °C. Indeed, this 'threshold' denoted an absence of further benefit in attention-based performance (i.e. reaction time and accuracy of rapid visual information processing). The existence of this 'threshold' has previously been highlighted by Bandelow et al. [8] for a range of psychomotor tasks, implying a T_{core} of ~38.2 °C represented a plateau in heat-induced cognitive improvements. Consistent with the same trend, performance of numerous functions (verbal learning, attention and concentration) has been reported as unchanged at a T_{core} of ~38.5 °C, whereas the most demanding performances tended to start to deteriorate [41]. Together, these observations suggest the existence of a threshold following which the initial cognitive benefits of heat stress start to plateau, and task complexity determines whether cognitive performance is further impaired.

Intuitively, interpretations about the absence of changes in cognitive task performance when T_{core} is ~38.5 °C may rely on whether a thermal stress is sufficient to appropriately disturb homeostasis, i.e. normal cognitive functioning. However, at these levels of hyperthermia, changes in brain functioning have been shown and, in contrast to the proposal of a 'normal' cognitive functioning, led to discussions about the presence of a heat-related cognitive 'ceiling' effect. More precisely, it has been proposed that the contribution of the two components of reaction time, i.e. the central component (the time interval between the onset of the response signal and the onset of the EMG activity of the muscles involved in the response) versus the peripheral component (the time interval from the onset of EMG activity to the onset of the required motor response), is altered in response to increased heat strain (~38.5 ± 0.2 °C), despite overall performance remaining stable [22, 42]. In particular, when T_{core} values approach ~38.7 °C, Gaoua et al. [22] suggested that the central component slows to such an extent that it compensates the heat-induced accelerated peripheral component of reaction time. This phenomenon has also been suggested by Hocking et al. [41] within the same range of heat stress (~38.7 ± 0.2 °C), with observations that alterations within steady-state visually evoked potentials (SSVEP) remained noticeable, even though no clear changes in task performances were evident. In this study, greater brain activity, as measured via increased SSVEP magnitude and decreased latency, was indicative of a greater utilization of neural resources in order to preserve performance [41]. However, at higher T_{core}, the authors speculated that heat strain would overload cognitive capacities (i.e. the ability to deal with both heat and the task), making any trade-off and performance maintenance difficult. More recently, Liu et al. [43] reported findings consistent with this speculation when performing fMRI analysis during alerting and orienting cognitive tasks. The authors suggested such compensating processes might result from a greater activity of variant regional brain areas (i.e. a compensation from additional brain structures, including the frontal and temporal areas), rather than greater specific cortical involvement (i.e. hyperactivation of the brain area initially activated).

These findings, obtained using different tools, interestingly point towards a common phenomenon, i.e. that a heat-induced 'threshold' exists for cognitive performance. More specifically, as T_{core} reaches ~38.5 °C, a plateau in cognitive functioning appears evident, made explicit through the greater cognitive activity observed while task performance remains stable. However, beyond this threshold, the greater activation of frontal and temporal cortices does not compensate for heat-related constraints and leads to concomitant increased cortical activity and decreased cognitive performance [44].

2.3 Cognitive Impairment as a Function of Hyperthermia

As T_{core} values progressively exceed ~38.7 °C, the extent of hyperthermia and task complexity seemingly interact to determine the type and magnitude of cognitive performance impairment [25, 45–47]. For instance, at a T_{core} of ~39.0 °C, both Racinais et al. [48] and Gaoua et al. [22] reported negative effects of passive hyperthermia on complex tasks, i.e. working memory capacity and recognition. Concomitantly, the delay before subjects started to be more impulsive became shorter compared with colder conditions, i.e. from 20 to 7 min [22], which is considered an indicator of the depletion of ego resources [49]. In contrast, at this level of hyperthermia, simple tests (choice reaction time, visual processing) remained unaffected, demonstrating a task-dependent response to T_{core} drift. A similar complexity-based discrimination between tasks has more recently been reported when assessing the three components of the attention network [43]. In particular, when T_{core} was passively increased to ~38.8 °C,

impairment of the efficiency of executive component of self-control was evident (i.e. resolving conflicting situations), while the orienting and alerting components (which are critically less-demanding tasks) remained unaffected. This led the authors to suggest that "tasks demanding lower attention are less vulnerable to the effect of hyperthermia than those requiring more attention" [43]. However, when T_{core} continues to increase towards higher values (greater than ~39 °C), this sparing effect of 'less vulnerable' tasks seems to dissipate and hyperthermia-induced cognitive impairment spreads to simple tasks. Indeed, in addition to impaired high-demand performances (complex levels of visual processing, working memory) when T_{core} reached up to ~40 °C, Bandelow et al. [8] noticed a global, non-specific slowing effect of hyperthermia that appeared consistent on psychomotor response speed, regardless of task complexity. Such extended cognitive deterioration suggests the existence of a subject's maximal capacity to tolerate an *overall* cognitive strain (both heat- and task-mediated), beyond which performance level could not be maintained.

Reasons for these observed decrements may vary. In part, the heating of neurons, together with temperature-related endogenous feedback from baro- and chemoreceptors, and type III and IV afferents, may exert inhibitory influence on cognitive functioning [50]. This inhibition may operate through basal ganglia, which, in response to thalamic and insular reaction to hyperthermia, may act to preserve perceived effort by inhibiting output intensity towards the PFC [51–53]. Evidence in support of this basal forebrain-dependent hypothesis has previously been obtained in subjects undergoing physical effort [54] and is further corroborated by heat-related changes in EEG. In particular, a generalized reduction of cortical activity in the heat is reported after exercising participants reach T_{core} between ~39.5 °C and ~40.0 °C [55, 56]. The decreased β activity observed in conditions of hyperthermia (from the occipital to the frontal lobe) is classically considered as symptomatic of a lower arousal [55], changes in brain connectivity [57] and slowing computations [58]. Of note, this reduction in cortical activity does not seem to be metabolism-based, i.e. during hyperthermia (greater than ~39.5 °C) neither a serotonin-fatigue hypothesis in humans nor the reduced CBF (and the related reduction in substrates delivery) were found to support such cortical downregulation [59, 60]. In contrast, a hormonal perspective has been proposed to account for the reduction in cognitive performance. Specifically, impaired performances on high-loading working memory tasks were found to correlate with high cortisol values [61]. As a potential mechanistic explanation, it has then been proposed that cerebral resources could be redistributed from the frontal to emotional centres of the brain in response to heat stress-induced cortisol [34].

Given the above responses, it is presumed that hyperthermia-induced cognitive impairment is not systematic, even at T_{core} above ~38.5 °C. Rather, task complexity plays a key role in cognitive outcomes at this level of heat stress and suggests practical implications for athletes before specific cognitive processes become altered.

2.4 Summary: A U-Shaped Interpretation of Cognitive Responses?

Collectively, the aforementioned findings suggest cognitive performance to be a function of the extent of hyperthermia

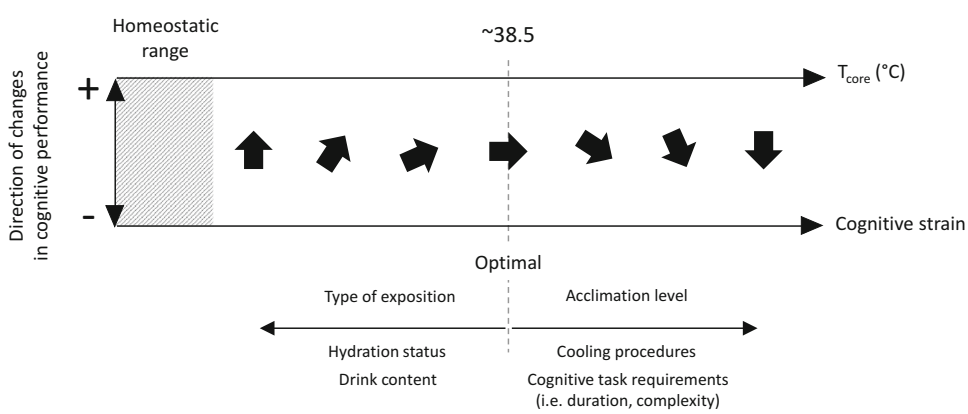


Fig. 1 Schematic representation of heat-related cognitive performance. The direction of the arrows indicates the nature of the effect of T_{core} on cognitive performance. This effect can be positive (*arrow pointing up*), neutral (*arrow pointing horizontally*) or negative (*arrow pointing down*). The *vertical broken line* corresponds to the cognitive 'threshold', i.e. the maximal cognitive load (100%) the subject can sustain before hyperthermia-induced cognitive benefits cease. This load could be attained at a T_{core} of ~38.5 ± 0.2 °C, and modulated by factors potentially regulating T_{core} and listed under the curve. As an example, an individual whose cognitive reserve reaches ~80% as a result of T_{core} increase would lie on the *left side of the vertical broken line* (i.e. *arrows pointing up*) and would thus cognitively benefit from heat strain. T_{core} core temperature

and task complexity, and, more specifically, propose the existence of an inverted U-shaped response of cognitive performances as T_{core} increases (Fig. 1). In particular, as hyperthermia develops, a T_{core} of $\sim 38.5 \pm 0.2$ °C could constitute the 'threshold' where cognitive improvement ceases and beyond which heat-related detrimental cognitive effects accumulate depending on cognitive task complexity [22, 41, 43]; however, the timing of this decrement remains unclear and a plateau of steady performance seems to persist before performance declines (Table 1). Therefore, from an ecological perspective, this plateau needs to be considered since (i) the onset of performance decrement along the dynamics of increasing T_{core} seems dependent on how complex the task is for the subject [62] and could therefore vary with expertise level; (ii) cognitive task performance results from decontextualized, laboratory measures of cognitive functioning that only partly reflect field characteristics; and (iii) interindividual variations in the response to heat stress will influence hyperthermia-related cognitive outcomes (e.g. sex difference [63]).

Interestingly, these results are coherent with the maximum adaptability model proposed earlier by Hancock and Warm [23]. Specifically, regardless of the underpinning mechanisms at play within this inverted U-shaped model, recent reports from the heat–cognition literature highlight an initial increase in cognitive performance before performance impairment becomes noticeable with hyperthermia development. Adding to the proposal by Hancock and Warm [23], recent studies using innovative technologies provide further explanations of the evolving effects of heat strain on cognitive functioning. Notably, the fact that modest hyperthermia could induce cognitive benefit seems systematic rather than situational, and, furthermore, could happen regardless of participants' willingness to increase attentional focus (e.g. improvement via hyperthermia-related arousal). In addition, before cognitive performance declines, the existence of a 'plateau' of performance seems to emerge and could actually relate to compensating activity from additional brain areas to those already involved in the task [43]. Some recent experimental findings also demonstrate that despite T_{core} values exceeding ~ 39.0 °C, cognitive performance decrement could be slowed, thus suggesting a possible uncoupling between heat strain and cognitive functioning (discussed in Sect. 3). With this in mind, future investigations should provide insights regarding some important questions in the context of sport performance e.g. whether the 'plateau' of cognitive performance could be anticipated and/or maintained before performance decline, or how long this plateau lasts depending on task complexity or exercise mode (passive vs. active)-related arousal. Indeed, the exercise–cognition relationship appears to be a dynamical pattern (with beneficial then/or detrimental effects depending on exercise intensity and duration, and participants' fitness level [13, 15]), fluctuations of which could overlap and thus moderate the heat–cognition relationship.

Recently, the debilitative responses to heat stress (i.e. the downward part of the inverted U-shaped curve of the present model) have been theorized to exist within an overload paradigm related to human cognitive capacity [22, 64]. Accordingly, heat stress (considered as a stressor equivalent to task complexity) would place additional attentional demand on the limited cognitive workspace, thereby reducing the amount of resources remaining available for concurrent cognitive tasks [65]. Either induced passively or through exercise, hyperthermia development would progressively lead the cognitive strain to become overloaded, making parallel computational demands subservient to spontaneous impulses [22]. If this switch from a control to an automatic mode of cognitive response during progressive hyperthermia is confirmed, then the downward part of the inverted U-shaped curve could potentially be underlined by mental fatigue paradigms [49, 66]. These paradigms propose the hypothesis that self-control resources would empty as cognitive processes are implemented—somewhat akin to muscle strength decreases during exercise. In the present case, the emptying of the cognitive 'reserve' would be both task- and heat-dependent; however, this proposition remains speculative and requires confirmation by concomitant behavioural and neuroimagery studies.

Regardless, a practical viewpoint of the possible inverted U-shaped relationship of hyperthermia and cognitive performance highlights the need to track T_{core} during exercise to identify situations that may impair cognitive performance and to consider interventions that could moderate cognitive performance. Specifically, these moderators could promote heat-related improvements (i.e. the upward part of the inverted U-shaped curve) (Fig. 1) while delaying or preventing the drift towards excessive hyperthermia (i.e. greater than ~ 39.0 °C) to maintain optimal cognitive processing alongside physical performance.

3 Behavioural Implications of Cognitive Performances Under Heat Stress

3.1 Cooling Manoeuvres

Exercise- and/or environment-induced increased T_{core} is associated with a greater physiological strain, e.g. sweating responses and hypovolemia, cardiac strain and perceived effort, thermal discomfort, and sensations of faintness [24]. Regardless of the pre- and/or post-exercise cooling methods used, the aim is to slow endogenous heat accumulation and thus limit the increase in T_{core}. Cooling methods are

Table 1 Summary of the investigations, reviewed in the present analysis, that integrated cognitive performance assessment during exposure to heat stress

References	Experimental design	Type of heat exposure and environmental conditions	T_{core} control/exp. (mean °C Δ)	T_{skin} control/exp. (mean °C Δ)	Type of cognitive task	Changes in cognitive performance vs. control	Supplementary indices of cognitive functioning and changes in signal	Potential strategy and effect on cognitive performance vs. hot only	
Bandelow et al. [8]	20 collegiate football players; between-group design; pre-, mid- and post-football match assessment	Active (no testing) then passive (testing)	~37.5 °C/ +2.5 °C	–			–	Cooling tent (~25 °C) and buckets with icy water	Rehydration with glucose
		~34 °C, 64% RH			Visual sensitivity	↘ (complex level)		↗	↗
					Visual/auditory working memory	↘		→	↗
					Visuospatial working memory	↘		→	↘
					Finger-tapping test	↘		↗	↗
Gaoua et al. [22]	16 subjects; counterbalanced design	Passive (60 min resting + 33 min of cognitive testing)	~37.1 °C/ +1.6 °C	~34.4 °C/ +5.0 °C			–	Head/neck cooling: cold packs (−14 °C)	
Racinais et al. [48]					Visual search	→		→	
					Choice reaction time	→		→	
		20 °C, 40% RH			Information processing speed	↘		↗	
		50 °C, 50% RH			Working memory	↘		↗	
					Pattern recognition memory	↘		→	
Hocking et al. [41]	11 subjects; 'partially' counterbalanced design	Active (1.8 km h^{-1})	~37.3 °C/ +1.4 °C	–	Verbal learning	→	Steady state probe topography: ↗ amplitude and ↘ latency	–	
					Attention	→			
		25 °C, 65% RH			Concentration	↘			
		35 °C, 65% RH			Information processing speed	↘			
					Working memory	↘			

Table 1 continued

References	Experimental design	Type of heat exposure and environmental conditions	T_{core} control/exp. (mean °C Δ)	T_{skin} control/exp. (mean °C Δ)	Type of cognitive task	Changes in cognitive performance vs. control	Supplementary indices of cognitive functioning and changes in signal	Potential strategy and effect on cognitive performance vs. hot only
Lee et al. [30]	12 subjects; counterbalanced design	Active (no testing) then passive (testing) ~30 °C, ~71% RH	~36.7 °C/ +2.8 °C to + 0.6 °C	~32.7 °C/ +2.0 °C to −0.3 °C	Working memory Executive control Sustained attention Vigilance	↗ → ↘ ↘	BDNF: ↗ blood concentration	Neck-cooling collar → ↗ → →
McMorris et al. [34]	8 subjects; counterbalanced design	Active (no testing) then passive (testing) 20 °C, 40% RH 36 °C, 75% RH	–/ + 1.10 °C	–	Working memory Short-term memory Choice reaction time	↘ → →	Plasma value of: cortisol, ↗ adrenaline, ↗	–
Simmons et al. [29]	10 subjects; counterbalanced design	Passive 25 °C, 50% RH, rising to 45 °C, 50% RH	~37.1 °C/ +1.2 °C	~32.6 °C/ +3.5 °C	Sustained attention	↘	–	Head/neck cooling: perfused balaclava (water at ~3 to ~8 °C) →
Gaoua et al. [70]	18 subjects; counterbalanced design	Passive 24 °C, 30% RH 50 °C, 30% RH	~37.3 °C/− 0.1 °C	~33.2 °C/ +2.8 °C	Attention Planning	→ ↘	Motor cortical excitability: →	–
Liu et al. [43]	23 subjects; between-group design	Passive 20 °C, 40% RH 50 °C, 40% RH	~37.7 °C/ +0.7 °C	–	Attention (alerting) Attention (orienting) Attention (executive network)	→ → ↘	fMRI: ↗ front. ↘ occ., par. ↗ temp. ↘ front., par., occ. ↗ front., temp. ↘ postcentral	–
Qian et al. [14]	20 subjects; counterbalanced design	Passive 25 °C, 60% RH 50 °C, 60% RH	~37.2 °C/ +1.2 °C	–	Vigilance	↘	Arterial spin labelling (for CBF): ↗ BS, thalamus ↘ front., par., cing.	–

Table 1 continued

References	Experimental design	Type of heat exposure and environmental conditions	T_{core} control/exp. (mean °C Δ)	T_{skin} control/exp. (mean °C Δ)	Type of cognitive task	Changes in cognitive performance vs. control	Supplementary indices of cognitive functioning and changes in signal	Potential strategy and effect on cognitive performance vs. hot only
Schlader et al. [31]	29 subjects; between-group design	Passive	~37.0 °C/ +1.0 °C	~34.0 °C/ +4.4 °C	Attention	↗	–	–
		Water-perfused suit: 34 °C			Choice reaction time	→		
		48 °C			Executive control	↗		
					Visual memory	→		
Sun et al. [58]	36 subjects; between-group design	Passive	~37.4 °C/–	–	Attention (alerting)	→	fMRI (for brain functional connectivity network):	–
		25 °C, 50% RH			Attention (orienting)	→		
		50 °C, 50% RH			Attention (executive network)	↘	65 disturbed connectivities	
							(50 ↘, 15 ↗)	
Schlader et al. [60]	21 subjects; single session	Passive	~36.9 °C/ +1.4 °C	~34.4 °C/ +5.0 °C	Working memory	→	Cerebral perfusion: →	–
		Water-perfused suit: 34 °C, rising to 49 °C						
Clarke et al. [73]	8 subjects; counterbalanced design	Active (no testing) with passive testing periods	~36.8 °C/ +2.0 °C	~31.0 °C/ +4.0 °C			–	Cold bath pre-exercise cooling (60 min at ~20 °C):
		~32 °C, ~47% RH			Visual discrimination (sensitivity)	→		→
					Visual discrimination (accuracy)	→		↗

BDNF brain-derived neurotrophic factors, *CBF* cerebral blood flow, *exp* experimental condition, *fMRI* functional magnetic resonance imaging, *RH* relative humidity, T_{core} core temperature, T_{skin} skin temperature; Brain regions: *front.* frontal, *occ.* occipital, *par.* parietal, *temp.* temporal, *BS* brainstem, *cing.* cingulate, → indicates no change in cognitive performance/indices, ↗ indicates improvements in cognitive performance, ↘ indicates impairments in cognitive performance

commonly classified as external or internal strategies [67]. Internal cooling strategies (e.g. ice slushy, slurry) and some external cooling manoeuvres (e.g. cold-water immersion, combined methods) have been shown to be effective in reducing T_{core} [68]. While this augments the core-to-skin gradient of temperature and delays thermal homeostasis disruption [69], some other external cooling methods modulate this gradient only by decreasing T_{skin} (e.g. cooling vest, ice towels, cool showers) [68]. Regardless of the specific intervention, an ergogenic effect of precooling for endurance performance in the heat is commonly reported [68]. However, in relation to the proposed model of cognitive functioning during hyperthermia, evidence for the effect of cooling strategies on cognitive performance remains limited, but seems appropriate depending on the timing of implementation.

Mechanisms by which cooling interventions could be ergogenic for cognitive performance are probably plural in nature, and we refer the reader to the study by Ross et al. [67] for detailed mechanisms of the effect of cooling on the reduction of physiological strain. In brief, by decreasing T_{skin}, external cooling will reduce skin blood flow and both cardiac and brain strain associated with cutaneous perfusion. Complementarily, by decreasing T_{core} and/or T_{skin}, cooling will reduce afferent information arising via endogenous thermo-, chemo- and baroreceptors, and thereby reduce thermoregulatory responses (e.g. sweating leading to dehydration). Overall, these cooling-based physiological changes could be of interest in the theory of a limited cognitive workspace [65], whereby reducing heat strain would increase attentional resource availability that would subsequently be used for coping with the cognitive demand.

The aforementioned U-shaped model suggests that a modest increase in T_{core} benefits cognitive functioning. However, in environmental hot conditions, an increase in T_{skin} per se is always reported before any increase in T_{core}, and, of note, this has been found to induce detrimental cognitive performance through sensory discomfort and associated altered cortical activity [5, 70]. Therefore, increasing T_{core} while concomitantly restricting the rise in T_{skin} may have use for athletes required to perform cognitively demanding tasks in hot conditions. To this end, an ice vest and towels, or cool showers, could promote skin cooling while not hindering T_{core} elevation [67]. In contrast, an internal cooling manoeuvre may be less advisable since it would inhibit hyperthermia initiation and therefore maintain cognitive performance at a non-optimal level.

Once T_{core} increases beyond ~38.5 °C, strategies delaying the rate of body heat accumulation and/or heat-related perceived cognitive loads could benefit cognitive performances in the heat. Indeed, according to an inverted U-shaped representation of cognitive functioning during the development of hyperthermia, these strategies may determine how long cognitive performance could plateau before declining. Accordingly, if practically appropriate, cooling methods could protect cognitive functioning against excessive hyperthermia (i.e. greater than ~39.0 °C). This protective effect may especially be effective when performance requires heavy, complex mental computations (e.g. planning, updating memory, switching from one rule to another). For instance, ice slurries and cold-water ingestion or immersion are known to decrease T_{core} [71, 72] and could therefore be implemented during resting periods of competitions, with specific doses and pretesting by athletes. Similarly, mixing an ice vest with cold packs has been found effective in decreasing T_{core} [68], and could therefore temporarily protect athletes performing for prolonged periods in the heat from dizziness and detrimental cognitive performance. However, such a protective effect for field-based cognitive performance (e.g. decision making for referees) has yet to be tested experimentally and thus remains speculative in real-world scenarios.

In support of the strategies described in this section, Clarke et al. [73] reported improved visual discrimination during hyperthermia (~39.0 ± 0.5 °C) induced while exercising in the heat when using cold-water immersion at 20.3 ± 0.3 °C for 60 min as a precooling intervention, in comparison to without precooling. Furthermore, cooling the head and neck during hyperthermic conditions was found to eclipse the hyperthermia-related debilitative effect on short-term memory [22], as well as search and memory functions [30], such that performances returned to baseline levels. Although such a cooling method does not appear to reduce T_{core} [29, 74–79], it appears effective in decreasing perceived exertion during exercise [5], which may help athletes push themselves to greater mental effort. In particular, among different head surfaces cooled, forehead (front) cooling could provide the most beneficial effect to reduce thermoregulatory responses in hot environments [80]. When it is considered that brain temperature may become higher than T_{core} in hyperthermic conditions [59], and that even moderate hyperthermia (i.e. ~38.4 °C) becomes increasingly debilitative as soon as cognitive effort has to be sustained over time [14, 21], these manoeuvres could feasibly become of primary importance for sport performance or medical assistance. However, do date, no experimental evidence has reported the effects of head/neck cooling in these practical contexts.

3.2 Hydration

Hydration usually refers to overall water intake over the course of the day; however, hydration is more precisely addressed in sports and laboratory settings, i.e. in terms of

drink quantity, content, temperature (or form: liquid or solid) and timing, each of which is of particular interest in the prevention of water deficit [81]. Indeed, water deficit and consumption are known to play a crucial role in both physical and mental performance [82]; however, body water loss (and the resultant dehydration) is markedly exacerbated in hot conditions compared with temperate conditions [83]. Therefore, besides its direct debilitative effect on cognitive performance, body water loss could also indirectly modulate cognitive functioning through its impact on thermoregulatory mechanisms and hyperthermia.

The debilitative psychological effect of dehydration has been demonstrated using both passive (i.e. hyperthermia) and active (i.e. exercise) methods of induction [84]. Specifically, while body water loss systematically impairs mood states, in contrast, cognitive functioning and sport skills appear less sensitive to dehydration, with evidence indicating that they are less sensitive in real-life tasks than in laboratory tasks [85]. Interestingly, when cognitive impairment is not reported following dehydration, this could relate to higher levels of frontal and temporal neuronal activities implemented to compensate for the water-deficit-related stress and enabling maintenance of performance level [86], an effect similar to that observed during hyperthermia [41, 43]. In explanation, dehydration-induced cognitive performance decline could originate in increased levels of cortisol [87], monoamine-related changes in blood–brain barrier permeability and associated perturbations of central nervous system functioning [88]. Alternatively, subjective feelings of 'difficulty in concentrating' and 'fatigue' that reduce the will to invest mental effort are also proposed to explain these declines [89, 90]. With this in mind, hydration status appears an important component of hyperthermia that could potentially exacerbate the decline in cognitive performance in hot conditions.

Accordingly, the general guideline is to initiate drinking before attaining 1–2% of body mass loss, i.e. before heat- and/or exercise-induced dehydration starts to markedly deteriorate cognition [91]. Beyond these values (i.e. near 3–5% of body weight loss), it is possible that the robustness of even simple processes may be weakened [65]. Therefore, when there is a need to maintain an ability to perform complex cognitive computations in the heat, ingestion of fluids at a temperature of ~4 to 5° C, or ice slushies ~20 to 45 min prior to the event, could be of particular utility for preventing excessive dehydration [92–94]. Specifically, providing the athlete simultaneously with water intake *and* cooling will promote (i) cooling-related reduction in T_{core} (direct effect via conduction); (ii) drink-related reduction in T_{core} (indirect effect via plasma volume restoration, i.e. convection); (iii) cooling-related reduction in sweating responses and skin blood flow; and (iv) improved thermal comfort and thirst [95, 96]. In so far as each of these mechanisms act to reduce heat strain, a cold drink may thus have the potential to promote cognitive performance in the heat. However, to our knowledge, the effect of internal cooling strategies on cognitive performance in the heat has not been tested yet.

As a complement to cool drink ingestion, additional cooling methods (e.g. ice vest, ice packs) should be employed as this combination may augment the reduction in T_{core}. Indeed, the cooling-based magnitude of changes in T_{core} appears amplified when in a euhydrated state compared with when dehydrated [97], thus providing support for the use of mixed cooling methods to improve cognitive functioning. Butts et al. [97] assumed this amplified effect relates to rehydration-based increased blood volume, which could improve the heat-carrying capacity of the blood and thus the ability to remove heat from the core. However, no cognitive performance measurements were reported from this study. An additional hydration strategy that could limit the effect of hyperthermia on cognitive functioning takes into consideration the components of the fluid consumed. In particular, supplementing the cold drink with electrolytes and carbohydrate (e.g. using sport drinks) will promote fluid absorption (via sodium) while meeting the heat-related higher cognitive load (via glucose) [98, 99]. For instance, Bandelow et al. [8] reported that the ingestion of drinks containing carbohydrates accelerated complex levels of visual processing and performance on memory tasks. In addition, the correlation between T_{core} and plasma glucose level (Pearson's $r = 50\%$) found in this study suggested that glucose supplementation enabled participants to partly counteract the negative cognitive effect of hyperthermia [8]. Therefore, in the case of combined interventions, it is speculated that these strategies act to shift the level of T_{core} from excessive hyperthermia back to 'threshold' levels and minimize the magnitude of hyperthermia-related cognitive performance decline. Of note, Lee et al. [30] reported that exercising in the heat while in a euhydrated state, and despite a T_{core} value superior to ~39.5 °C, improved memory performances. Such anecdotal observations could relate to interactions between body water maintenance and exercise-induced cognitive arousal [100]. This specific observation highlights the necessity for future research to discriminate the interacting effects of heat- versus exercise-related arousal from those of heat- versus exercise-related dehydration in cognitive performance changes.

4 Conclusions

Cognitive functioning, as determined from consideration of performance on respective cognitive tasks, is altered with increasing extent of hyperthermia. However, this heat–cognition relationship is initially ergogenic for cognitive

performance, with accumulating heat stress consistently improving both simple and complex cognitive processes when T_{core} is less than ~38.5 °C. However, beyond this threshold, cognitive performance plateaus, before declining at a T_{core} greater than ~39 °C, with the more effortful cognitive computations being the first impaired. Thus, an inverted U-shaped relationship emerges between the level of hyperthermia and cognitive performance, and may reflect the difficulty for the brain to deal with accumulating stressors (i.e. cognitive demand and increasing heat strain). While the upwards part of the curve may be explained through heat-related arousal, the downwards component has been suggested to reflect the inability of the 'cognitive reserve' to process such accumulation of constraints [22]. Since it mainly relies on T_{core}, this dynamical conceptualization also suggests cognitive performances are modifiable, i.e. performance could be protected in so far as heat-related physiological strains can be counteracted.

Compliance with Ethical Standards

Funding No sources of funding were used to assist in the preparation of this article.

Conflict of interest Cyril Schmit, Christophe Hausswirth, Yann Le Meur and Rob Duffield declare that they have no conflicts of interest relevant to the content of this review.

References

1. Altareki N, Drust B, Atkinson G, et al. Effects of environmental heat stress (35 °C) with simulated air movement on the thermoregulatory responses during a 4 km cycling time-trial. Int J Sports Med. 2009;30:9–15.
2. Guy JH, Deakin GB, Edwards AM, et al. Adaptation to hot environmental conditions: an exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. Sports Med. 2015;45(3):303–11.
3. Tatterson AJ, Hahn AG, Martin DT, et al. Effects of heat stress on physiological responses and exercise performance in elite cyclists. J Sci Med Sports. 2000;3:186–93.
4. Racinais S, Périard JD, Karlsen A, et al. Effect of heat and heat acclimatization on cycling time trial performance and pacing. Med Sports Sci Exerc. 2015;47:601–6.
5. Flouris AD, Schlader ZJ. Human behavioral thermoregulation during exercise in the heat. Scand J Med Sci Sports. 2015;25(Suppl. 1):52–64.
6. Périard JD, Cramer MN, Chapman PG, et al. Cardiovascular strain impairs prolonged self-paced exercise in the heat. Exp Physiol. 2011;96(2):134–44.
7. Rasmussen P, Dawson EA, Nybo L, et al. Capillary-oxygenation-level-dependent near-infrared spectrometry in frontal lobe of humans. J Cereb Blood Flow Metab. 2007;27(5):1082–93.
8. Bandelow S, Maughan R, Shirreffs S, et al. The effect of exercise, heat, cooling and rehydration strategies on cognitive function in football players. Scand J Med Sci Sports. 2010;20(3):148–60.
9. Smits BL, Pepping GJ, Hettinga FJ. Pacing and decision making in sport and exercise: the roles of perception and action in the regulation of exercise intensity. Sports Med. 2014;44(6):763–75.
10. Armstrong LE, Casa DJ, Millard-Stafford M, et al. American College of Sports Medicine. American College of Sports Medicine position stand. Exertional heat illness during training and competition. Med Sci Sports Exerc. 2007;39:556–72.
11. Zazulak BT, Hewett TE, Reeves NP, et al. The effects of core proprioception on knee injury a prospective biomechanical-epidemiological study. Am J Sports Med. 2007;35(3):368–73.
12. Lorist MM, Boksem MA, Ridderinkhof KR. Impaired cognitive control and reduced cingulate activity during mental fatigue. Cogn Brain Res. 2005;24(2):199–205.
13. Schmit C, Davranche K, Easthope CS, et al. Pushing to the limits: the dynamics of cognitive control during exhausting exercise. Neuropsychologia. 2015;68:71–81.
14. Qian S, Li M, Li G, et al. Environmental heat stress enhances mental fatigue during sustained attention task performing: evidence from an ASL perfusion study. Behav Brain Res. 2015;280:6–15.
15. McMorris T. Last word on viewpoint: reappraisal of the acute, moderate intensity exercise-catecholamines interaction effect on speed of cognition: role of the vagal/NTS afferent pathway. J Appl Physiol. 2016;120(6):661.
16. Barbas M. Connections underlying the synthesis of cognition, memory, and emotion in primate prefrontal cortices. Brain Res Bull. 2000;52:319–30.
17. Scholey AB, Harper S, Kennedy DO. Cognitive demand and blood glucose. Physiol Behav. 2001;73(4):585–92.
18. Ramsey JD, Kwon G. Recommended alert limits for perceptual motor loss in hot environments. Int J Ind Ergon. 1992;9:245–57.
19. Hancock PA. Sustained attention under thermal stress. Psychol Bull. 1986;99:263–81.
20. Pilcher JJ, Nadler E, Busch C. Effects of hot and cold temperature exposure on performance: a meta-analytic review. Ergonomics. 2002;45:682–98.
21. Liu K, Jiang Q, Li L, et al. Impact of elevated core body temperature on attention networks. Cogn Behav Neurol. 2015;28(4):198–206.
22. Gaoua N, Racinais S, Grantham J, et al. Alterations in cognitive performance during passive hyperthermia are task-dependent. Int J Hyperthermia. 2011;27(1):1–9.
23. Hancock PA, Warm JS. A dynamic model of stress and sustained attention. Hum Factors. 1989;31:519–37.
24. Nybo L, Rasmussen P, Sawka MN. Performance in the heat: physiological factors of importance for hyperthermia-induced fatigue. Compr Physiol. 2014;4:657–89.
25. Hancock PA, Vasmatzidis I. Effects of heat stress on cognitive performance: the current state of knowledge. Int J Hyperth. 2003;19:355–72.
26. Ando S, Kokubu M, Yamada Y, et al. Does cerebral oxygenation affect cognitive function during exercise? Eur J Appl Physiol. 2011;111:1973–82.
27. Grego F, Vallier J-M, Collardeau M, et al. Effects of long duration exercise on cognitive functions, blood glucose, and counterregulatory hormones in males athletes. Neurosci Lett. 2004;364(2):76–80.
28. Parsons KC. Human thermal environments: the effects of hot, moderate and cold environments on human health, comfort and performance. London: Taylor & Francis; 1993.
29. Simmons SE, Mündel T, Jones DA, et al. The effects of passive heating and head cooling on perception of exercise in the heat. Eur J Appl Physiol. 2008;104:281–8.
30. Lee JKW, Koh ACH, Koh SXT, et al. Neck cooling and cognitive performance following exercise-induced hyperthermia. Eur J Appl Physiol. 2014;114(2):375–84.
31. Schlader ZJ, Gagnon D, Adams A, et al. Cognitive and perceptual responses during passive heat stress in younger and older adults. Am J Physiol Regul Integr Comp Physiol. 2015;308(10):R847–54.

32. Razmjou S. Mental workload in heat: toward a framework for analyses of stress state. Aviat Space Environ Med. 1996;67:530–8.
33. Johnson RF, Kobrick JL. Psychological aspects of military performance in hot environments. In: Pandolf KB, Burr RE, Wenger CB, Pozos RS, editors. Medical aspects of harsh environments, vol. 1. Fort Detrick: U.S. Army Medical Research and Materiel Command; 2001.
34. McMorris T, Swain J, Smith M, et al. Heat stress, plasma concentrations of adrenaline, noradrenaline, 5-hydroxytryptamine and cortisol, mood state and cognitive performance. Int J Psychophysiol. 2006;61:204–15.
35. Robbins TW, Everitt BJ. A role for mesencephalic dopamine in activation: commentary on Berridge (2006). Psychopharmacology. 2007;191:433–7.
36. Lambourne K, Tomporowski P. The effect of exercise-induced arousal on cognitive task performance: a meta-regression analysis. Brain Res. 2010;1341:12–24.
37. Boas DA, Strangman G, Culver JP, et al. Can the cerebral metabolic rate of oxygen be estimated with near-infrared spectroscopy? Phys Med Biol. 2003;48(15):2405.
38. Mohtasib RS, Lumley G, Goodwin JA, et al. Calibrated fMRI during a cognitive Stroop task reveals reduced metabolic response with increasing age. Neuroimage. 2012;59(2):1143–51.
39. Rooks CR, Thom NJ, McCully KK, et al. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy: a systematic review. Prog Neurobiol. 2010;92(2):134–50.
40. Nybo L, Secher NH. Cerebral perturbations provoked by prolonged exercise. Prog Neurobiol. 2004;72(4):223–61.
41. Hocking C, Silberstein RB, Lau WM, et al. Evaluation of cognitive performance in the heat by functional brain imaging and psychometric testing. Comp Biochem Physiol. 2001;128:719–34.
42. Aird JW, Webb RD, Hoare J. Heat exposure-induced changes in motor outflow component of reaction time. Percept Mot Skills. 1983;56:699–706.
43. Liu K, Sun G, Li B, et al. The impact of passive hyperthermia on human attention networks: an fMRI study. Behav Brain Res. 2013;243:220–30.
44. Jiang Q, Yang X, Liu K, et al. Hyperthermia impaired human visual short-term memory: an fMRI study. Int J Hyperthermia. 2013;29(3):219–24.
45. Amos D. Physiological and cognitive performance of soldiers conducting routine patrol and reconnaissance operations in the tropics. Mil Med. 2000;165(12):961.
46. Hancock PA, Vasmatzidis I. Human occupational and performance limits under stress: the thermal environment as a prototypical example. Ergonomics. 1998;4:1169–91.
47. Ramsey JD, Kwon YG. Recommended alert limits for perceptual motor loss in hot environments. Int J Ind Ergonom. 1992;9(3):245–57.
48. Racinais S, Gaoua N, Grantham J. Hyperthermia impairs short-term memory and peripheral motor drive transmission. J Physiol. 2008;586:4751–62.
49. Muraven M, Baumeister RF. Self regulation and depletion of limited resources: does self control resemble a muscle? Psychol Bull. 2000;126:247–59.
50. Taylor JL, Todd G, Gandevia SC. Evidence for a supraspinale contribution to human muscle fatigue. Clin Exp Pharmacol Physiol. 2006;33:400–5.
51. Olausson H, Charron J, Marchand S, et al. Feelings of warmth correlate with neural activity in right anterior insular cortex. Neurosci Lett. 2005;389(1):1–5.
52. Schmidt L, Lebreton M, Cléry-Melin M-L, et al. Neural mechanisms underlying motivation of mental versus physical effort. PLoS Biol. 2012;10(2):1–13.
53. Sridharan D, Levitin DJ, Menon V. A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. Proc Natl Acad Sci. 2008;105(34):12569–74.
54. Schmidt L, Cléry-Melin M-L, Lafargue G, et al. Get aroused and be stronger: emotional facilitation of physical effort in the human brain. J Neurosci. 2009;29(30):9450–7.
55. De Pauw K, Roelands B, Marusic U, et al. Brain mapping after prolonged cycling and during recovery in the heat. J Appl Physiol. 2013;115:1324–31.
56. Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. J Appl Physiol. 2001;91:1055–60.
57. Liu K, Li B, Qian S, et al. Altered interhemispheric resting state functional connectivity during passive hyperthermia. Int J Hyperthermia. 2015;31(8):840–9.
58. Sun G, Qian S, Jiang Q, et al. Hyperthermia-induced disruption of functional connectivity in the human brain network. PLoS One. 2013;8(4):e61157.
59. Nybo L, Moller K, Volianitis S, et al. Effects of hyperthermia on cerebral blood flow and metabolism during prolonged exercise in humans. J Appl Physiol. 2002;93:58–64.
60. Schlader ZJ, Lucas RA, Pearson J, et al. Hyperthermia does not alter the increase in cerebral perfusion during cognitive activation. Exp Physiol. 2013;98(11):1597–607.
61. Kirschbaum C, Wolf OT, May M, et al. Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. Life Sci. 1996;58:1475–83.
62. Hancock PA. Task categorizations and the limits of human performance in extreme heat. Aviat Space Environ Med. 1982;53:778–84.
63. Ježova D, Kvetňanský R, Vigaš M. Sex differences in endocrine response to hyperthermia in sauna. Acta Physiol Scand. 1994;150(3):293–8.
64. Baars BJ. How does a serial, integrated and very limited stream of consciousness emerge from a nervous system that is mostly unconscious, distributed, parallel and of enormous capacity? Ciba Found Symp. 1993;174:282–90.
65. Gaoua N. Cognitive function in hot environments: a question of methodology. Scand J Med Sci Sports. 2010;20(3):60–70.
66. Baumeister RF, Bratslavsky E, Muraven M, et al. Ego depletion: is the active self a limited resource? J Pers Soc Psychol. 1998;74(5):1252.
67. Ross M, Abbiss C, Laursen P, et al. Precooling methods and their effects on athletic performance. Sports Med. 2013;43(3):207–25.
68. Bongers CC, Thijssen DH, Veltmeijer MT, et al. Precooling and percooling (cooling during exercise) both improve performance in the heat: a meta-analytical review. Br J Sports Med. 2014;19:2013–092928.
69. Cuddy JS, Hailes WS, Ruby BC. A reduced core to skin temperature gradient, not a critical core temperature, affects aerobic capacity in the heat. J Thermal Biol. 2014;43:7–12.
70. Gaoua N, Grantham J, Racinais S, et al. Sensory displeasure reduces complex cognitive performance in the heat. J Exp Psychol. 2012;32(2):158–63.
71. Byrne C, Owen C, Cosnefroy A, et al. Self-paced exercise performance in the heat after pre-exercise cold-fluid ingestion. J Athl Train. 2011;46:592–9.
72. Stevens CJ, Dascombe B, Boyko A, et al. Ice slurry ingestion during cycling improves Olympic distance triathlon performance in the heat. J Sports Sci. 2013;31:1271–9.
73. Clarke ND, Duncan MJ, Smith M, et al. Pre-cooling moderately enhances visual discrimination during exercise in the heat. J Sport Sci. 2016. doi:10.1080/02640414.2016.1164885

74. Armada-da-Silva PAS, Woods J, Jones DA. The effect of passive heating and face cooling on perceived exertion during exercise in the heat. Eur J Appl Physiol. 2004;91:563–71.
75. Desruelle AV, Candas V. Thermoregulatory effects of three different types of head cooling in humans during a mild hyperthermia. Eur J Appl Physiol. 2000;81:33–9.
76. Minett MG, Duffield R, Kellett A, et al. Mixed-method precooling reduces physiological demands without improving performance of medium-fast bowling in the heat. J Sport Sci. 2012;30(9):907–15.
77. Palmer CD, Sleivert GG, Cotter JD. The effects of head and neck cooling on thermoregulation, pace selection and performance. In: Proceedings of the international thermal physiology symposium, vol. 32. Australian Physiological and Pharmacological Society. Wollongong (NSW); 2001. p. 122.
78. Tyler CJ, Wild P, Sunderland C. Practical neck cooling and time-trial running performance in a hot environment. Eur J Appl Physiol. 2010;110:1063–74.
79. Tyler CJ, Sunderland C. Neck cooling and running performance in the heat: single versus repeated application. Med Sci Sports Exerc. 2011;43(12):2388–95.
80. Katsuura T, Tomioka K, Harada H, et al. Effects of cooling portions of the head on human thermoregulatory response. Appl Human Sci. 1996;15:67–74.
81. Masento NA, Golightly M, Field DT, et al. Effects of hydration status on cognitive performance and mood. Br J Nutr. 2014;111(10):1841–52.
82. Gómez-Pinilla F. Brain foods: the effects of nutrients on brain function. Nat Rev Neurosci. 2008;9(7):568–78.
83. Sawka MN, Pandolf KB. Effects of body water loss on physiological function and exercise performance. Perspectives in exercise science and sports medicine. In: Gisolfi CV, Lamb DR, editors. Fluid homeostasis during exercise. vol. 3. Carmel: Brown and Benchmark; 1990. p. 1–38.
84. Cian C, Barraud PA, Melin B, et al. Effects of fluid ingestion on cognitive function after heat stress or exercise-induced dehydration. Int J Psychophysiol. 2001;42:243–51.
85. Vázquez CC, Sánchez MV, Sánchez JC, et al. Dehydration, cognitive and skill performance in sport. Systematic review. Nutr Hosp. 2015;32(Suppl 2):24.
86. Kempton MJ, Ettinger U, Foster R, et al. Dehydration affects brain structure and function in healthy adolescents. Hum Brain Map. 2011;32(1):71–9.
87. Comijs HC, Gerritsen L, Penninx BW, et al. The association between serum cortisol and cognitive decline in older persons. Am J Geriatr Psychiatry. 2010;18(1):42–50.
88. Maughan RJ, Shirreffs SM, Watson P. Exercise, heat, hydration and the brain. J Am Coll Nutr. 2007;26(Suppl 5):604S–12S.
89. Armstrong LE, Ganio MS, Casa DJ, et al. Mild dehydration affects mood in healthy young women. J Nutr. 2012;142(2):382–8.
90. Gorby HE, Brownawell AM, Falk MC. Do specific dietary constituents and supplements affect mental energy? Review of the evidence. Nutr Rev. 2010;68(12):697–718.
91. Lieberman HR. Hydration and cognition: a critical review and recommendations for future research. J Am Coll Nutr. 2007;26(5):555–61.
92. Edmonds CJ, Crombie R, Ballieux H, et al. Water consumption, not expectancies about water consumption, affects cognitive performance in adults. Appetite. 2013;60:148–53.
93. Rogers PJ, Kainth A, Smit HJ. A drink of water can improve or impair mental performance depending on small differences in thirst. Appetite. 2001;36(1):57–8.
94. Siegel R, Mate J, Brearley MB, et al. Ice slurry ingestion increases core temperature capacity and running time in the heat. Med Sci Sports Exerc. 2010;42(4):717–25.
95. Coyle EF, Gonzalez-Alonso J. Cardiovascular drift during prolonged exercise: new perspectives. Exerc Sport Sci Rev. 2001;29(2):88–92.
96. Sawka MN, Montain SJ. Fluid and electrolyte supplementation for exercise heat stress. Am J Clin Nutr. 2000;72(2):564s–72s.
97. Butts CL, Luhring KE, Smith CR, et al. Effects of mild hypohydration on cooling during cold-water immersion following exertional hyperthermia. Eur J Appl Physiol. 2016;116(4):687–95.
98. Gold PE. Role of glucose in regulating the brain and cognition. Am J Clin Nutr. 1995;61(4):987S–95S.
99. Shirreffs SM, Taylor AJ, Leiper JB, et al. Post-exercise rehydration in man: effects of volume consumed and drink sodium content. Med Sci Sports Exerc. 1996;28(10):1260–71.
100. Brisswalter J, Collardeau M, Arcellin R. Effects of acute physical exercise characteristics on cognitive performance. Sports Med. 2002;32:555–66.

TRAVAIL DE THÈSE

Schmit, C., Davranche, K., Easthope, C. S., Colson, S. S., Brisswalter, J., & Radel, R. (2015). Pushing to the limits: the dynamics of cognitive control during exhausting exercise. *Neuropsychologia*, 68, 71-81.

Résumé en français

Cette étude a eu pour objet d'étudier les changements parallèles du contrôle cognitif et de l'oxygénation cérébrale (Cox) au cours d'un exercice intense et fixe conduit à l'épuisement volontaire. Quinze participants ont été monitorés via une NIRS (spectroscopie par infrarouges) placée sur leur cortex préfrontal, et via des mesures EMG (électromyographie) de leur pouce, au cours de la réalisation d'une tâche Eriksen réalisée au repos (condition contrôle) ou à l'exercice intense jusqu'à épuisement (condition exercice). Deux fenêtres temporelles d'intérêt ont été ciblées (au début puis en fin d'exercice) et comparées entre les deux conditions, afin d'étudier un potentiel effet cognitif évolutif lié à l'exercice. Au cours de la période initiale, la Cox est restée inchangée et, contrairement aux prédictions théoriques, l'exercice intense n'a pas induit de dégradation de la performance cognitive. Au contraire, les courbes delta obtenues suggèrent une plus grande efficience des processus cognitifs en première partie d'exercice. Peu avant l'épuisement, le niveau de Cox a drastiquement chuté, sans pour autant s'avérer caractéristique d'un état d'hypofrontalité. Aucun signe de déficit du contrôle inhibitoire n'a été enregistré. Malgré cette observation, une plus grande tendance à commettre des erreurs impulsives, et une perte d'efficience de la capacité de correction d'erreur, ont été relevées en fin d'exercice. Une corrélation négative entre les valeurs de Cox et le pourcentage d'erreurs a été notée et est discutée en termes de redistribution des ressources cérébrales.

Ce travail a permis de spécifier la relation dynamique entretenue entre 'cognition' et 'exercice', tout en questionnant les fondements mécanistiques de cette relation. Il ouvre la perspective d'un rationnel neuronal qui demande à être modélisé pour en proposer l'intérêt.

Neuropsychologia 68 (2015) 71–81

Contents lists available at ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

Pushing to the limits: The dynamics of cognitive control during exhausting exercise

Cyril Schmit [a,b,*], Karen Davranche [d], Christopher S. Easthope [a,c], Serge S. Colson [a], Jeanick Brisswalter [a], Rémi Radel [a]

[a] Laboratoire LAMHESS (EA6309), Université de Nice Sophia-Antipolis, France
[b] Institut National du Sport, de l'Expertise et de la Performance (INSEP), Département de la Recherche, Paris, France
[c] Spinal Cord Injury Centre, Balgrist University Hospital, Zurich, Switzerland
[d] Aix Marseille Université, CNRS, LPC UMR 7290, FR 3C FR 3512, 13331 Marseille cedex 3, France

ARTICLE INFO

Article history:
Received 26 May 2014
Received in revised form
25 November 2014
Accepted 7 January 2015
Available online 8 January 2015

Keywords:
Cognitive functions
Response inhibition
Physical exercise
Cerebral oxygenation
Exhaustion

ABSTRACT

This study aimed at investigating concurrent changes in cognitive control and cerebral oxygenation (Cox) during steady intense exercise to volitional exhaustion. Fifteen participants were monitored using prefrontal near-infrared spectroscopy and electromyography of the thumb muscles during the completion of an Eriksen flanker task completed either at rest (control condition) or while cycling at a strenuous intensity until exhaustion (exercise condition). Two time windows were matched between the conditions to distinguish a potential exercise-induced evolutive cognitive effect: an initial period and a terminal period. In the initial period, Cox remained unaltered and, contrary to theoretical predictions, exercise did not induce any deficit in selective response inhibition. Rather, the drop-off of the delta curve as reaction time lengthened suggested enhanced efficiency of cognitive processes in the first part of the exercise bout. Shortly before exhaustion, Cox values were severely reduced – though not characteristic of a hypofrontality state – while no sign of deficit in selective response inhibition was observed. Despite this, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed near exhaustion. A negative correlation between Cox values and error rate was observed and is discussed in terms of cerebral resources redistribution.

1. Introduction

It is now well-accepted that physical exercise has a positive effect on basic cognitive functions (Tomporowski, 2003; Lambourne and Tomporowski, 2010), however its impact on higher cognitive processes (*e.g.* selective inhibition) is less clear. According to the different meta-analyzes on exercise and cognition, it seems that exercise would lead to a rather small positive effect on cognitive functioning due to large variations of the reported results (Chang et al., 2012; McMorris et al., 2011). It has been proposed that exercise intensity would be the most important factor to explain this variability. Specifically, an inverted-*U* function has been suggested in such a form that exercise above a certain intensity is no longer beneficial to cognitive functioning (McMorris and Hale, 2012). In other words, while moderate exercise is associated with a positive effect, intense exercise (*i.e.* above the second ventilatory threshold, VT2) is associated to a null or negative effect on cognitive functioning. This view has been supported by the main theoretical models. According to the arousal-cognitive performance (Yerkes and Dodson, 1908), the catecholamine (Cooper, 1973; McMorris et al., 2008) and the reticular-activating hypofrontality (Dietrich, 2003, 2009; Dietrich and Audiffren, 2011) theories, intense exercise, by inducing high levels of arousal, increasing neural noise, or down-regulating prefrontal cortex activity, respectively, is predicted to impede higher cognitive processes.

Evidence of the detrimental effect of intense exercise mostly come from studies using incremental protocols in which the effects of exercise, probed at the end of the exercise, are confounded with the effect of exhaustion (*e.g.* Ando et al., 2005; Chmura and Nazar, 2010; McMorris et al., 2009). The present study aimed to dissociate the effect of the intensity from the state of exhaustion using a steady state intense exercise performed until exhaustion. Exhaustion is a psychophysiological state concluding a fatigue development process. It is a predictable consequence to any strenuous exercise. The state of exhaustion can also be considered as the time spent on the task. In a concomitant realization of a choice reaction time task and a fatiguing submaximal contraction,

* Correspondence to: Institut National du Sport, de l'Expertise et de la Performance. 11, avenue du Tremblay, 75 012 Paris, France.
E-mail address: cyril.schmit@insep.fr (C. Schmit).

http://dx.doi.org/10.1016/j.neuropsychologia.2015.01.006
0028-3932/

Lorist et al. (2002) showed that the more the time-on-tasks elapsed, the more error rate and force variability of hand muscles increased, suggesting an increasing detrimental effect of the dual-task on the dual-performance.

The detrimental effect of exercise on cognition occurring near exhaustion can be explained as neural competition for the limited cerebral resources between different centers of the brain (Dietrich, 2003, 2009; Dietrich and Audiffren, 2011). Specifically, the hypofrontality theory predicts that the prefrontal cortex (PFC), in response to the development of muscular fatigue, would be down-regulated to favor the allocation of resources to motor areas, which would in turn result in weaker cognitive functioning. The resource redistribution hypothesis has been supported by animal studies assessing cerebral activity during exercise. Tracing regional cerebral blood flow and local cerebral glucose utilization as indexes of cerebral activity showed a selective cortical and subcortical recruitment of brain areas during exercise (Delp et al., 2001; Gross et al., 1980; Holschneider et al., 2003; Vissing et al., 1996). Specifically, while motor and sensory cortices, basal ganglia, cerebellum, midbrain and brainstem nuclei were consistently activated at high intensities, the frontal cortex rather showed signs of deactivation. Positron emission tomography studies have also provided a similar pattern of findings in human (Tashiro et al., 2001, Kemppainen et al., 2005). If such neural balance may rationalize down-regulated cognitive performances during exercise (Dietrich and Audiffren, 2011), previous result remain based on an instantaneous description of the neural pattern during exercise, which does not inform about changes related to the proximity of exhaustion.

Near-infrared spectroscopy (NIRS) is a continuous tissue-monitoring technique which is able of tracking cerebral oxygenation (Cox) during exercise due to its relative robustness during movement (see Perrey, 2008). The NIRS method has been validated and correlates highly with both electro-encephalographic and functional magnetic resonance imaging responses (Timinkul et al., 2010; Toronov et al., 2001). NIRS studies during exercise have shown a very dynamic Cox pattern. A meta-analysis by Rooks et al. (2010) proposes an intensity based account with the second ventilatory threshold as the critical reversing point in the inverted-*U* relation between exercise intensity and PFC oxygenation determined through oxyhemoglobin concentration [HbO_2]. Nevertheless, this pattern is specific to untrained participants, as trained participants do not show any [HbO_2] decline at high intensities. In addition, most of the studies reviewed employed incremental protocols. In studies that push participants until exhaustion, results suggest that [HbO_2] reduction may be related to the individual time course to exhaustion. Timinkul et al. (2008) reported an individual timing of Cox desaturation, occurring before VT2 for some participants and after for others. During steady intense exhausting exercise, Shibuya et al. (2004a,b) reported Cox patterns identical to those observed in incremental exercise (e.g., Bhambhani et al., 2007; Rupp and Perrey, 2008). However, the paucity of the exercise-NIRS studies on intense as well as prolonged exercise to exhaustion in humans cannot ensure the validity of a fatigue-based PFC deactivation hypothesis.

The second aim of the present study was to investigate concurrent changes in cognitive performance and Cox in PFC while performing strenuous exercise until volitional exhaustion. More specifically, the protocol was designed to determine whether cognitive performance and Cox follow a similar dynamic. In an initial period of intense exercise, it was anticipated that cognitive performance would be facilitated and Cox elevated compared to the same initial period at rest. Then, in a critical period occurring just before exhaustion, we expected a decrease in cognitive performance and a drop of PFC [HbO_2] in comparison to the same period at rest. Cognitive performances were assessed using a modified version of the Eriksen flanker task (Eriksen and Eriksen, 1974), consisting in overcoming the irrelevant dimension of the stimulus to give the correct response, to probe the efficiency of selective response inhibition. The flanker task has been largely used to investigate the effects of exercise on cognitive control (Davranche et al., 2009a,b; McMorris et al., 2009; Pontifex and Hillman, 2007). Although mean RT and average error rate do provide valuable information relative to cognitive processes, more-detailed data analyzes uncover modulations that the sole consideration of central tendency indices cannot reveal. Indeed, combined to RT distribution analyzes, conflict tasks have proved to be powerful for assessing the processes implemented during decision-making tasks while exercising (Davranche and McMorris, 2009; Davranche et al., 2009a,b; Joyce et al., 2014).

On a substantial amount of trials, although the correct response was given, a subthreshold electromyographic (EMG) activity in the muscles involved in the incorrect response could be observed. Such subthreshold EMG activities, named "partial errors", reflect incorrect action impulses that were successfully corrected in order to prevent a response error (Hasbroucq et al., 1999). To evaluate the efficiency of the cognitive control during exercise, electromyographic (EMG) activity of response effector muscles were monitored to estimate the number of partial EMG errors. In order to measure Cox, NIRS recording was centered on the right inferior frontal cortex (rIFC) as this brain area is a main region involved in the brain network supporting the inhibition function (Aron et al., 2004, 2014), and has been described as the most responsive region while performing the Eriksen flanker task (Hazeltine et al., 2000). Additionally, the distribution-analytical technique and the delta plot analysis (Ridderinkhof, 2002; Ridderinkhof et al., 2004) were used to assess the efficiency of cognitive control and the propensity to make fast impulsive reactions through the analyzes of the percentage of correct responses (CAF) and the magnitude of the interference effect (delta curve) as a function of the latency of the response (van den Wildenberg et al., 2010). If exhausting exercise impairs the efficiency of cognitive control, the drop-off of the delta curve should be less pronounced in the terminal period than in the initial period. If the propensity to commit impulsive errors increases before exhaustion, more errors are expected for fast RT trials on distributional analyzes of response errors.

2. Method

2.1. Participants

Fifteen volunteers took part in this experiment. They were mostly classified as untrained following the $\dot{V}O_2$ max criteria of de Pauw et al. (2013) and had basic cycling experience (< 1 h a week). Informed written consent was obtained according to the declaration of Helsinki. Participants' anthropometrical and

Table 1
Anthropometrical and physiological characteristics of participants.

Variables	Mean ± SD All	Women	Men
Sample size	15	5	10
Age [years]	22.1 ± 0.6	23.2 ± 1.2	21.5 ± 0.6
Height [cm]	175.9 ± 2.6	166.3 ± 1.8	180.7 ± 2.6
Body mass [kg]	66.5 ± 3.1	52.9 ± 2.1	73.3 ± 2.3
$\dot{V}O_2$ max [ml kg^{-1} min^{-1}]	44.5 ± 1.9	45.2 ± 2.9	44.1 ± 2.6
Maximal HR [bpm]	178 ± 2.6	180 ± 1.9	177 ± 3.0
MAP [W]	261.3 ± 14.2	204.3 ± 10.3	290.2 ± 12.2

Results are presented as the mean group ± SD.
Notes. SD = standard deviation; MAP = maximal aerobic power; $\dot{V}O_2$max = maximal oxygen consumption; HR = heart rate.

physiological characteristics are presented in Table 1.

2.2. Apparatus and display

All sessions were performed on a cycle ergometer (Brain-bike NeuroActive, Motion Fitness, 1775 Winnetka Circle, Rolling Meadows, IL 60008) equipped with a handlebar and soft padding supports to comfortably support forearms. Two thumb response keys were fixed on the top of the right and left handle grips. A screen mounted on the ergometer at head height faced participants at a mean distance of 70 cm.

2.3. Cognitive task

The cognitive task consisted of a modified version of the Eriksen flanker task (Eriksen and Eriksen, 1974). Participants were required to complete 20 blocks of 40 trials during the control session and as many blocks as possible until exhaustion in the exercise session. There were two types of trials in each block: congruent trials (CO, 50%, all arrows uni-directional) and incongruent trials (IN, 50%, center arrow contra-directional). Each trial began with the presentation of a cross at the center as a fixation point. After 800 ms, the stimulus was presented and participants had to respond according to the direction pointed by the central arrow. The delivery of the response turned off the stimulus. When participants failed to respond within 2000 ms, the stimulus was terminated and the next trial began. The inter-stimulus interval was 800 ms. In this modified version, each group of arrows could randomly be displayed either at the top or at the bottom of the screen (Fig. 1). This modification ensured a higher processing of the flankers as participants could not anticipate the location of the central arrow.

2.4. Electromyographic measurement

Surface EMG activity of the *flexor pollicis brevis* (FPB) of each thumb and of the *vastus lateralis* (VL) of the right thigh were recorded using bipolar Ag/AgCl electrodes (diameter: 10 mm, inter-electrode distance (FPB, VL): 12 mm, 20 mm). The common reference electrode was situated on the head of the second metacarpal of the right hand. Electrodes locations were determined as proposed by Hasbroucq et al. (1999) and were marked in order to ensure congruent positioning in both sessions. Low inter-electrode impedance ($<3\,k\Omega$) was obtained via skin preparation. EMG signals were amplified (gain x1000), filtered (pass-band 10–500 Hz) and recorded at 2000 Hz (MP100 Biopac® Systems Inc., Holliston, MA, USA). During both sessions, the experimenter continuously monitored the signal of the thumbs and asked the subjects to relax their muscles if the signal became noisy through increased preactivation.

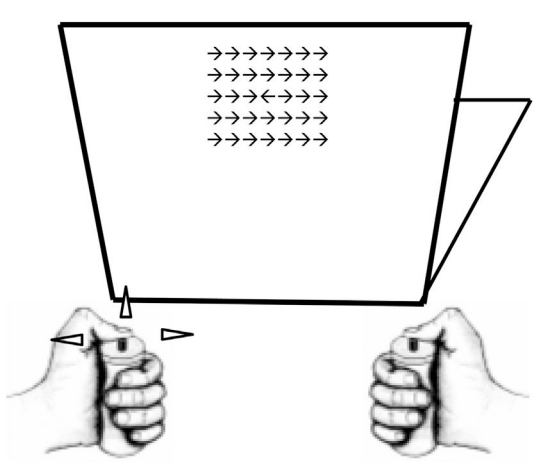


Fig. 1. Schematic drawing of an incongruent trial displayed at the top of the screen. In this example, participants had to respond by pressing the left response key according to the direction indicated by the central arrow.

2.5. Cerebral oxygenation measurement

Cox was monitored using a Near-infrared Spectroscopy (NIRS) system (Artinis Medical, PortaMon, The Netherlands). The NIRS evaluates the amount of infrared light that effectively traverses an investigated tissue from an emitter to a receptor. The received signal allows the description of a quantitative change from baseline in chromophore concentration. The PortaMon continuously emits two wavelengths of IR light, 773 and 853 nm, which are situated within the absorption spectrum of hemoglobin (Hb) and Myoglobin (Mb). Using two wavelengths allows for determination of the changes in [HbO_2] and deoxyhemoglobin ([HHb]) concentration. The inter-optode distance (IOD) was fixed through the apparatus at 30, 35 and 40 mm. Positioning of the NIRS probe was considered paramount to the study design (Strangman et al., 2003; Boas et al. 2001; Mansouri et al. 2010) and great care was taken that the probe was tangential to the curvature of the cranium and that no contamination through ambient light was present. Fixation was obtained through a custom-designed multi-density foam receptacle which was secured using a system of straps to prevent movement during exercise. Location of the rIFC was determined using AF8 references from the electro-encephalic 10–20 international system and measured in duplicate. The probe position was then extensively marked on the skin using a surgical marker and was also documented photographically for later reference to ensure congruent placement in the following session. Data was acquired at 10 Hz. Concentrations were calculated using the standard form of the modified Beer–Lambert Law (Beer, 1851; Delpy et al. 1988). For this calculation, the wavelength-specific extinction coefficients were extracted from Cope (1991) and the equally specific differential pathlength factors (DPF) were adopted from Duncan et al. (1995). HbO_2 concentration from the deepest source (IOD_{40}, ca. 2 cm) has been previously reported to be the most sensitive indicator of regional cerebral oxygenation changes and of neural activity (Hoshi, Kobayashi and Tamura, 2001) and was therefore selected as the primary outcome measure. Post-acquisition, the data was normalized to the 2-min rest period and the control data was truncated to retain the same duration as cognitive task time completion in the exercise condition. Data was subsequently reduced to 20 datapoints using a spline interpolation.

2.6. Experimental procedure

Three sessions (one training session and two experimental sessions) were conducted at standard (± 1 h) morning hours (from 8 to 12 pm) within close intervals (4 ± 3 days). Participants were instructed to abstain from any vigorous exercise for 24 h pre-session, to sleep at least seven hours in the night before each session and to avoid caffeinated drinks in the morning.

During the training session, subjects performed four blocks of 80 trials of the cognitive task to prevent a potential learning effect. Additional blocks were completed if the participant did not fulfill the following learning criteria: (a) RT intra-block variability below 5%, (b) RT variability with the previous block below 5%, (c) mean RT inferior to 600 ms, and (d) response accuracy superior to 85%. Participants then performed a maximal aerobic power (MAP) test on the cycle ergometer. The resistance was automatically regulated to ensure constant power output independent of pedal frequency. Power output was increased by 10 W every 30 s after a 4 min

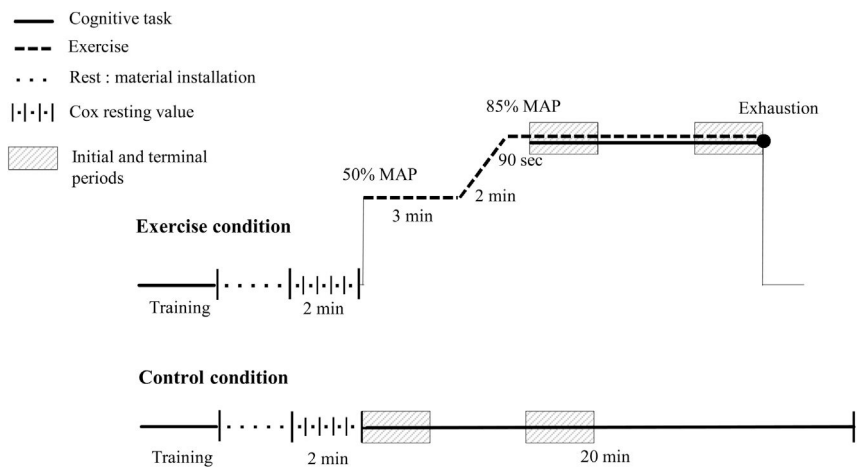


Fig. 2. Schematic representation of the experimental sessions which consisted of performing an Eriksen flanker task for 20 min without exercising (control condition) or while cycling at 85% of maximal aerobic power (MAP) (exercise condition). The initial period of exercise corresponds to the first hundred trials and the terminal period to the last hundred trials in the exercise condition. These two time windows were matched with the control condition. Cox resting values were measured prior to any cognitive and/or physical solicitation.

warm-up at light intensity (women: 70 W; men: 80 W). Oxygen consumption (ml kg^{-1} min^{-1}) and ventilatory output (L min^{-1}) were recorded using the Fitmate Pro gas analyzer (COSMED, Miami, USA) validated by Nieman et al. (2007). Cardiac frequency was recorded by a Polar system (Polar RS800CX, Polar Electror Oy, Kempele, Finland). Voluntary exhaustion was defined as the point where participants voluntarily stopped or when they could no longer maintain a pedaling frequency above 50 rotations per minute (RPM) more than 10 s despite strong verbal encouragement.

The experimental sessions consisted of a control condition and an exercise condition (Fig. 2). The order of the sessions was counterbalanced across participants. Each session began with two training blocks of 80 trials of the cognitive task (about 3 min). Following the installation of the NIRS and EMG sensors (about 30 min), the measurement of Cox resting values was conducted. The recording of baseline values only began if Cox level was visually stable during the last 2 min. Participants then performed the cognitive task for twenty minutes[1] without pedaling (control condition) or while pedaling at 85% of MAP until exhaustion (exercise condition). The intensity (regulated independently from pedal frequency) was chosen to be intense enough to exceed the second ventilatory threshold (VT2) while allowing an exercise duration long enough to obtain sufficient data to define two time periods: an initial period and a terminal period. In each condition, the initial period corresponded to the first hundred trials of the cognitive task performed by the participant. In the exercise condition, the terminal period corresponded to the last hundred trials performed just before exhaustion and was determined according to individual time to exhaustion. To obtain a matching terminal period in the control condition, the time frame corresponding to the cessation of pedaling during the exercise condition was extracted and the period calculated from there. The exercise condition began after a 3-min warm-up at 50% of MAP. The intensity was then increased every 20 s for 2 min until reaching 85% of MAP. To ensure that a steady-state of oxygen consumption was attained, a 90 s delay was imposed between the attainment of target intensity and the beginning of the cognitive task. Cox, heart rate (HR), pedaling frequency and electromyographic activity of the agonist muscles involved in the task were continuously monitored from rest to exhaustion.

2.7. Psychological measurement

Participants were asked to provide a verbal rating of perceived exertion (RPE) between each block of the cognitive task, using a visual Borg (6–20) scale (Borg, 1998). RPE was defined as the "perceived difficulty to exert at the same time the physical plus the cognitive tasks". The next block followed immediately after participants' RPE responses.

2.8. Data analysis

The EMG signals of the *flexor pollicis brevis* recorded during each trial were aligned to the onset of the imperative stimulus and the onsets of the changes in activity were visually determined. EMG signals with a background activity superior to 10% of the burst peak were rejected (Rejection rate: 20%). RT for pure correct trials–trials with no sign of EMG activation associated with the incorrect activation-was measured for each condition from onset of the stimulus to the onset of the EMG involved in the response. RTs of less than 100 ms and RTs higher than 1500 ms (3% of the total number of trials) were considered anticipated responses and omissions, and were excluded from further analysis. The incorrect activation trials were differentiated into two categories of trials: errors and partial EMG errors. Partial EMG errors were incorrect action impulses, mostly undetected consciously, that were successfully corrected (Hasbroucq et al., 1999; Rochet et al., 2014). The force exerted by the non-required effector was not sufficient to elicit an error and was followed by a correct activation which reached the response threshold. Trials containing a partial error are of particular interest, since they indicate that although an error was about to be made, the nervous system was able to overcome and provide the correct action. Error rate and partial EMG error rate were calculated according to the number of trials after artifact rejection. Additionally, the correction rate which represents the efficiency of the nervous system to overcome and provide the correct action after incorrect activations was calculated. It corresponds to the number of partial EMG errors divided by the total number of incorrect activations (partial EMG errors and errors).

For the exercise condition, the root mean square (RMS) from EMG signal of the VL was calculated using a custom signal detection routine in Matlab between the onset and the end of each burst recorded from the beginning of the cognitive task and until

[1] This corresponds to 1.5 times the best time-to-exhaustion performed during pre-tests and thus is ensured to completely cover the cognitive task duration during the exercise condition for all participants.

exhaustion. Results of each subject were reduced to 20 measurement points using a spline interpolation function in Matlab (R2012b, the MathWorks, Inc., Natick, MA) in order to depict the mean evolution of VL muscular activation during the task.

Through the analyzes of the percentage of correct responses (conditional accuracy functions, CAF) and the magnitude of the interference effect (delta curve) as a function of RT, the activation-suppression model provides a powerful framework to assess conflict resolution (for details see, van den Wildenberg et al., 2010). This model specifically allows for the assessment of both the initial phase linked to an individual's susceptibility to making fast impulsive errors (early automatic response activation) and, the later phase associated with the efficiency of the cognitive control (build-up of a top-down response suppression mechanism). Reaction time distribution was obtained using individual RTs "vincentized" into four equal-size speed bins (quartiles) for CO and IN trials separately. The lack of data for three participants did not permit a relevant vincentization, consequently they have not been taken into account in this analysis. Delta plots were constructed by calculating interference effect as a function of the response speed (average of difference between RT in IN and RT in CO trials for each quartile). Curve accuracy functions (CAF) were constructed by plotting accuracy as a function of the response speed. The data presented are the mean values of each set averaged across participants.

Separated ANOVAs were performed on each dependent variable (i.e., mean RT, partial EMG errors, errors and correction rates). The analyzes involved conditions (control vs. exercise) congruency (CO vs. IN) and periods (initial vs. terminal) as within-subject factors. In order to control for the effect of the order of the experimental session, this was initially included as a between-subject factor along with all its interaction terms with the other predictors in the analyzes. However, given that none of these variables reached significance ($ps > .10$), the order and its interaction terms were removed from the analyzes to optimize the parsimony of the models. An ANOVA including condition (control vs. exercise) and period (initial vs. terminal) as within-subjects factors was performed on hemoglobin concentration measures to determine whether cerebral oxygenation diverged. The analyzes conducted on the RMS of VL activation and RPE data recorded during the exercise session only included period (initial vs. terminal) as a within-subject factor. Exploratory Pearson correlations were performed between [HbO_2] concentration, cognitive performances (mean RT, error rate, partial error rate, correction rate) and root mean square (RMS) from EMG signal of the VL. Data presented on these measures correspond to mean values averaged across participants. The SPSS software (IBM® SPSS® Statistics 20) was employed for all analyzes. Planned comparisons were used in the GLM as post-hoc analyzes when significant p-values ($p < .05$) were found. Values are expressed as mean ± standard deviation (SD).

3. Results

3.1. Time to task failure, heart rate and rating of perceived exertion

In the exercise condition, the strenuous exercise until exhaustion lasted 360 ± 43 s, which enabled participants to complete 5.5 ± 0.4 blocks of the cognitive task (*i.e.*, average of 220 trials, ranged from 200 to 320 trials). Participants always stopped cycling at the end of a block of the cognitive task, meaning they always fully completed all the blocks they have started.

HR showed a main effect of condition ($F(1,14)=1507.95$, $p < .001$, $\eta_p^2=.99$), a main effect of period ($F(1,14)=43.83$, $p < .001$, $\eta_p^2=.76$) and an interaction between these two factors ($F(1,14)=33.27$, $p < .001$, $\eta_p^2=.70$). In the control condition, HR remained stable from the initial (67 ± 4 beat per minute, bpm) to the terminal period (66 ± 3 bpm, $F(1,14) < 1$, $p=.73$), but increased from 162 ± 2 bpm to 176 ± 2 bpm in the exercise condition ($F(1,14)=152.12$, $p < .001$, $\eta_p^2=.92$).

RPE results showed a main effect of period ($F(1,14)=387.18$, $p < .001$, $\eta_p^2=.96$) and condition ($F(1,14)=450.92$, $p < .001$, $\eta_p^2=.97$). An interaction between these two factors was observed ($F(1,14)=6.91$, $p=.02$, $\eta_p^2=.33$). The increase in RPE was greater in the exercise condition (from 14.2 ± 0.6 to 19.4 ± 0.3) compared to the control condition (6.5 ± 0.2 to 8.9 ± 0.7).

3.2. Reaction time

Results showed a main effect of congruency ($F(1,14)=29.72$, $p < .001$, $\eta_p^2=.68$) with longer RT for IN trials (454 ± 13 ms) than for CO trials (410 ± 8 ms). No other main effect or interaction was significant.

3.3. Error rate

Results showed a main effect of congruency ($F(1,14)=60.18$, $p < .001$, $\eta_p^2=.81$), a main effect of condition ($F(1,14)=9.54$, $p < .01$, $\eta_p^2=.40$) as well as an interaction between these two factors ($F(1,14)=11.74$, $p < .01$, $\eta_p^2=.45$, Fig. 3). The frequency of errors was lower during rest than when exercising for IN trials (Ctrl: 9.93 ± 1.14%; Exer: 18.15 ± 3.67%; $p < .01$) but not for CO trials (Ctrl: 0.86 ± 0.53%; Exer: 1.21 ± 0.52%; $p=.58$) (Fig. 3). Interestingly, the interaction between condition and period showed a trend ($F(1,14)=3.37$, $p=.08$, $\eta_p^2=.19$) and suggests that the evolution of accuracy differs between control and exercise conditions. In the terminal period, participants committed more errors during exercise (10.73 ± 3.84%) than at rest (5.32 ± 1.59%, $p < .01$, $\eta_p^2=.40$), whereas they had an equivalent error rate in the initial period (Exer: 8.31 ± 2.54%; Ctrl: 5.88 ± 1.41%; $p > .05$).

3.4. Partial EMG error

Results showed a main effect of congruency ($F(1,14)=143.76$, $p < .001$, $\eta_p^2=.91$), condition ($F(1,14)=11.32$, $p < .01$, $\eta_p^2=.45$) and period ($F(1,14)=7.21$, $p=.01$, $\eta_p^2=.34$). The number of partial EMG errors was increased for IN trials (36.92 ± 4.13%) compared to CO trials (10.65 ± 2.81%), exercise (28.09 ± 5.58%) compared to rest (19.49 ± 4.76%) and greater in the terminal period (25.83 ± 5.90%) than in the initial period (21.74 ± 4.26%). No interaction reached

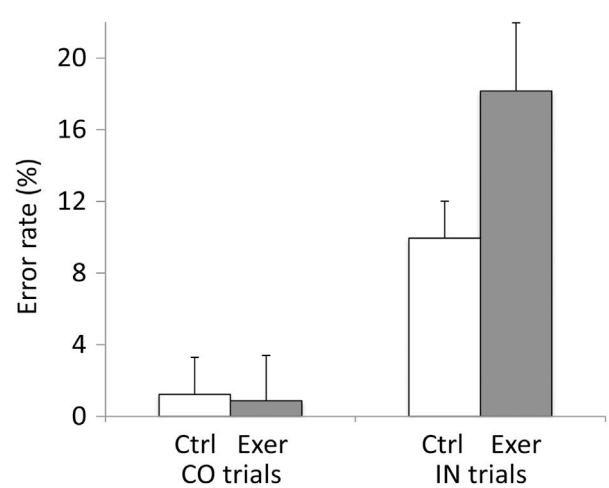


Fig. 3. Error rate (in percentage) during control (Ctrl, white bars) and exercise (Exer, grey bars) conditions for congruent (CO) and incongruent (IN) trials. Error bars represent standard deviation.

Table 2
Mean reaction times, accuracies and partial errors per condition and period in the Eriksen flanker task.

Variables		Control Initial	Terminal	Exercise Initial	Terminal
CO	RT (ms)	411 ± 34	414 ± 43	406 ± 40	410 ± 49
	Acc (%)	1.7 ± 2.3	0.7 ± 1.7	1 ± 1.9	0.7 ± 2.1
	PE (%)	7 ± 6.8	8.2 ± 6.5	12.8 ± 9.1	14.7 ± 10.3
IN	RT (ms)	454 ± 50	461 ± 46	447 ± 63	456 ± 76
	Acc (%)	10 ± 3.7	9.9 ± 4.9	15.7 ± 8.2	20.6 ± 14.2
	PE (%)	27.6 ± 14.8	35.2 ± 14.5	39.6 ± 14	45.3 ± 17.3

Results are presented as the mean group ± SD.
Notes. SD=standard deviation; CO=congruent trials; IN=incongruent trials; RT=reaction time; Acc=accuracy; PE=partial error.

significance. The interaction between condition and period did not reached significance (F(1;14)=.04; p=.84; η_p^2=.003). All the results for RT, error rate (accuracy) and partial EMG error are presented in Table 2.

3.5. Correction rate

Correction rate trended towards significant interaction between condition and period (F(1,14)=4.12, p=.06, η_p^2=.23, Fig. 4). In the initial period, the correction rate was equivalent at rest and during exercise (F(1,14)=0.82, p=.37, η_p^2=.05). However, in the terminal period, participants were less capable to correct incorrect action impulses during exercise (68.92 ± 3.42%) than in the control condition (78.05 ± 3.56%) (F(1,14)=7.03, p=.01, η_p^2=.33). This finding suggests a deficit in cognitive control just before exhaustion, incorrect action impulses were not corrected effectively and more errors were committed.

3.6. Distributional analysis

Reaction time distributions were submitted to an ANOVA involving condition (control vs. exercise), congruency (CO vs. IN), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subjects factors. Results confirmed the main effect of congruency previously observed on mean RT (F(1, 11)=73.07, p<.001, η_p^2=.87). More interestingly, the analysis showed an interaction between condition and quartile (F(3,33)=5.16, p<.01, η_p^2=.32)

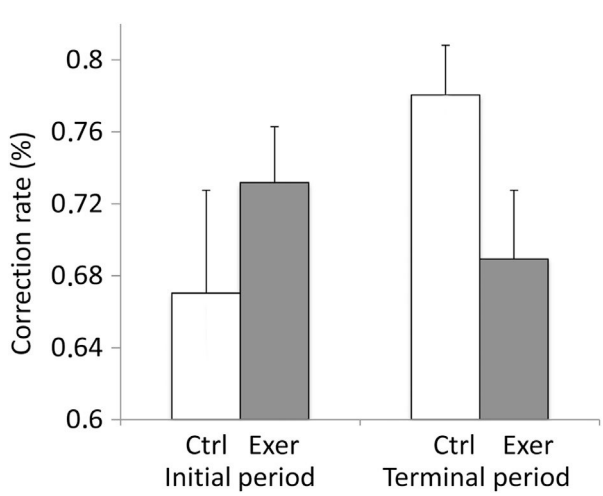


Fig. 4. Correction rate (in percentage) during rest (Ctrl, white bars) and exercise (Exer, grey bars) conditions for the initial and terminal periods. Error bars represent standard deviation.

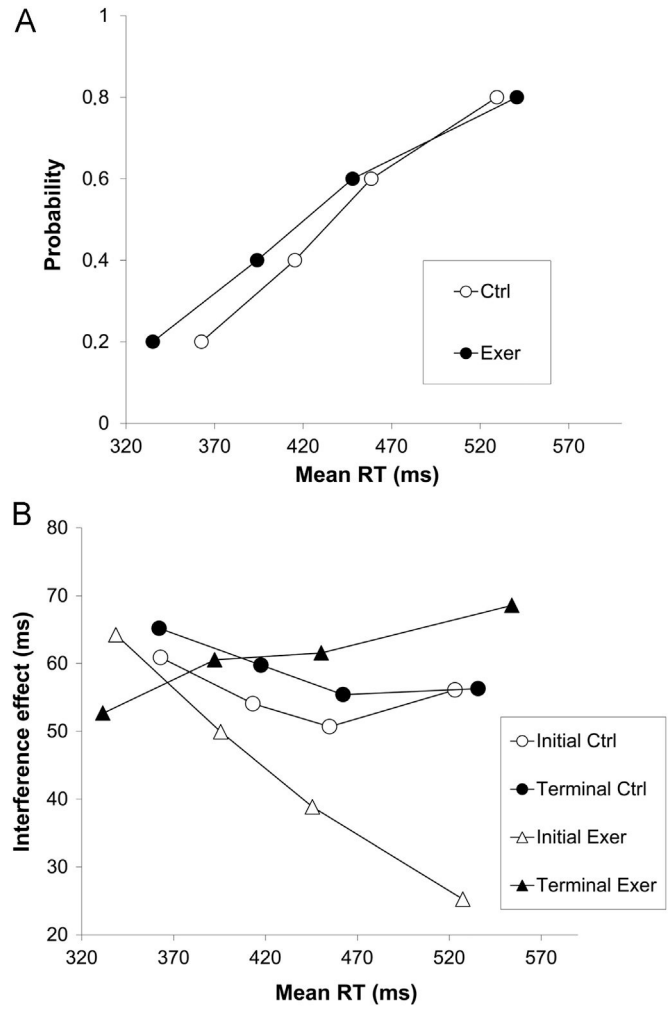


Fig. 5. (A) Cumulative density functions as a function of reaction time (RT) during rest (Ctrl, empty symbols) and exercise (Exer, full symbols) conditions. (B) Delta plots of RT illustrating the magnitude of the interference (in ms) as a function of RT during rest (Ctrl, circle) and exercise (Exer, triangle) conditions for the initial (empty symbols) and terminal (full symbols) periods.

which revealed that exercise differently affects RT performance as a function of the response speed (Fig. 5A). A beneficial effect of exercise was actually observed for the first quartile (Q1: −27 ms, p<.01), and the second quartile (Q2: −21 ms, p<.05) and disappeared for the last two quartiles (Q3: −11 ms, p=.19; Q4, +13 ms, p=.10).

According to van den Wildenberg et al. (2010), the magnitude of interference as RT lengthened is associated with the efficiency of the cognitive control (build-up of a top-down response suppression mechanism). Then, a second analysis focused on delta plot slopes was conducted to examine whether exercise altered the magnitude of the interference. The analysis involved condition (control vs. exercise), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subject factors. Results showed that the magnitude of interference did not fluctuate as RT lengthened, except during the initial period of exercise (Fig. 5B). In this period, the interference decreased from 64 ms (±9 ms) in the first quartile to 25 ms (±12 ms) in the last quartile (F(1,11)=14.19, p<.01) suggesting that non-exhausting intense exercise enhances cognitive control. It is noteworthy that no sign of any deficit in cognitive control was observed in the terminal period of exercise when exhaustion was about to occur (F(1,11)=0.07, p=.79).

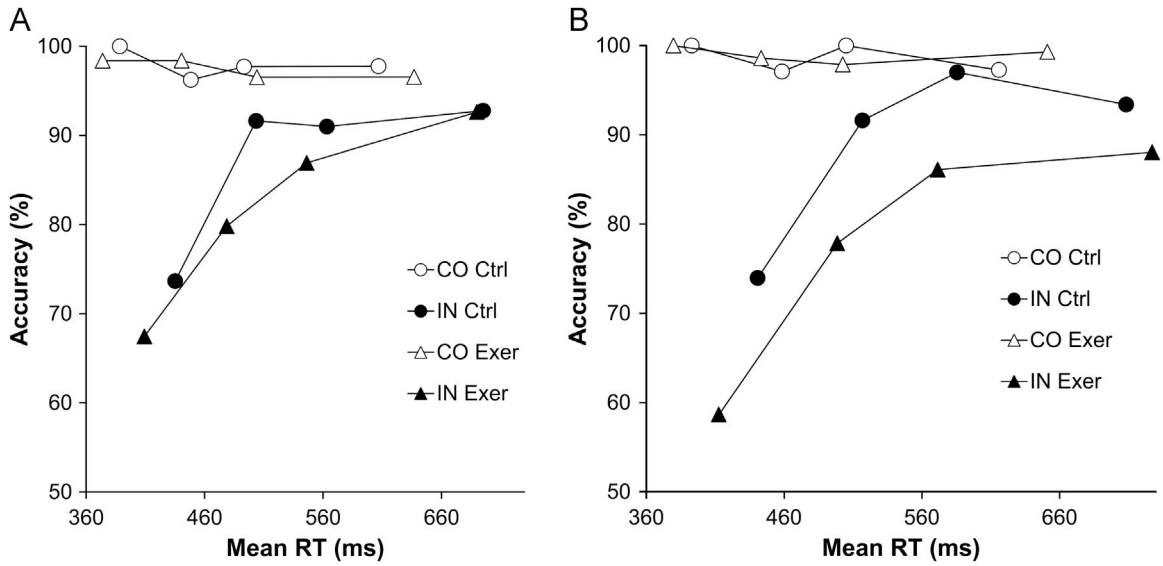


Fig. 6. Conditional accuracy function (CAF) representing the percentage of accuracy for congruent (CO, empty symbols) and incongruent (IN, full symbols) trials as a function of reaction time (RT) during control (Ctrl, circle) and exercise (Exer, triangle) conditions for the initial (A) and terminal (B) periods.

3.7. Conditional accuracy functions (CAF)

An ANOVA involving condition (control vs. exercise), congruency (CO vs. IN), period (initial vs. terminal) and quartile (Q1, Q2, Q3, Q4) as within-subject factors was conducted on error rates (Fig. 6). Results confirmed the main effects of condition ($F(3,14)=9.78$, $p<.01$, $\eta_p^2=.41$), congruency ($F(3,14)=40.35$, $p<.001$, $\eta_p^2=.74$) and the interaction between the two ($F(3,14)=11.14$, $p<.01$, $\eta_p^2=.44$) previously observed on mean error rate. In line with the activation–suppression model (Ridderinkhof, 2002), the interaction between congruency and quartile, which illustrated the strength of the automatic response triggered by the flankers, was significant ($F(3, 42)=25.02$, $p<.001$, $\eta_p^2=.64$). The interference was more pronounced for the first quartile than for the second quartile (Q1: 31 ms *vs.* Q2: 12 ms, $p<.001$) and for the second quartile compared to the third quartile (Q2: 12 ms vs. Q3: 8 ms, $p<.01$), whereas the interference was equivalent for the last two quartiles (Q3: 8 ms vs. Q4: 6 ms, $p=.23$). The interaction between condition, congruency and period ($F(1,14)=3.89$, $p=.07$, $\eta_p^2=.22$) and the interaction between condition, congruency and quartile ($F(3,14)=2.71$, $p=.07$, $\eta_p^2=.16$) tended to be significant.

A second series of analyzes, focusing on the first quartile of the CAF, was conducted to examine whether exercise alters the rapid response impulse (van den Wildenberg et al., 2010). The analysis carried out on the initial period of exercise did not reveal an interaction between condition and congruency ($F(1,14)=1.05$, $p=.32$, Fig. 6A). However, in the terminal period of exercise, there was a significant interaction ($F(1,14)=12.68$, $p<.01$, $\eta_p^2=.49$). Just before exhaustion, participants committed about 15% more errors than at rest in IN trials (Fig. 6B) while the accuracy rate in CO trials remained unchanged.

3.8. Cerebral oxygenation

An interaction between condition and period was observed on Cox fluctuations ($F(1,14)=5.98$, $p<.001$, $\eta_p^2=.30$) (Fig. 7). During the control condition, the time spent on the task did not impact Cox ($F(1,14)=0.71$, $p=.98$, $\eta_p^2=.04$). During exercise, $\Delta[HbO_2]$ linearly (linearity, $p<.05$) decreased from 2.71 ± 0.05 μmol cm in the initial period to 0.21 ± 0.06 μmol.cm in the terminal period

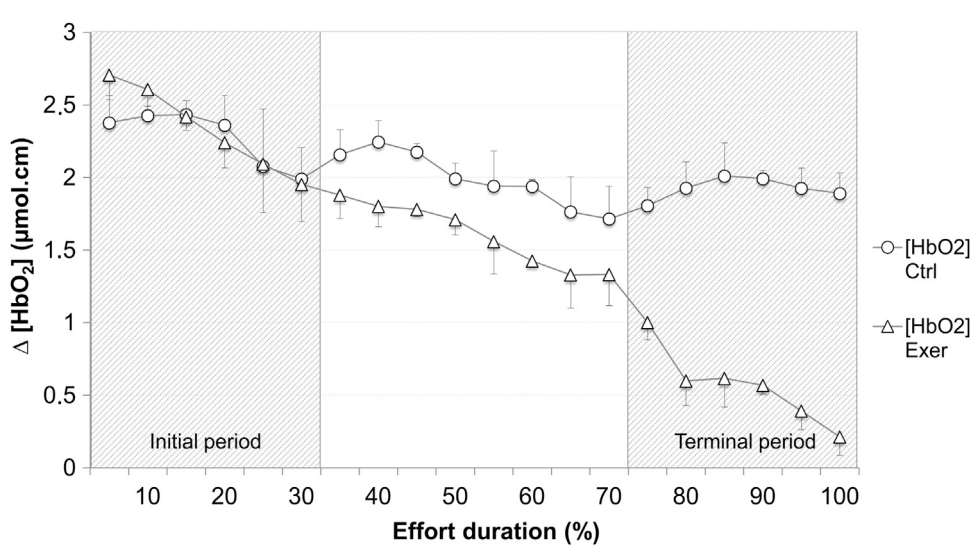


Fig. 7. Changes from resting values (baseline) in cerebral oxyhemoglobin $[HbO_2]$ during control (Ctrl, circles) and exercise (Exer, triangles) conditions. Error bars represent standard deviation.

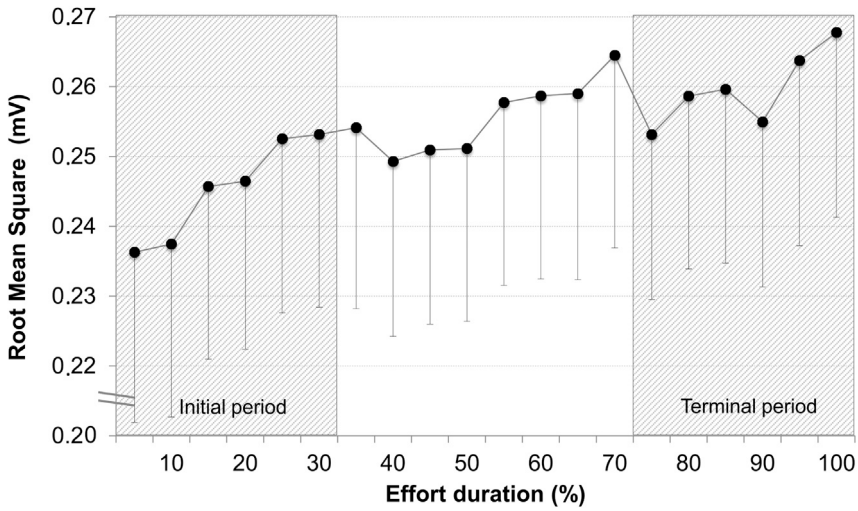


Fig. 8. Electromyographic root mean square (in mV) activity of the *vastus lateralis* muscle during the time-to-exhaustion cycling test. Error bars represent standard errors.

($F(1,14)=9.16$, $p<.001$, $\eta_p^2=.40$)[2]. In the terminal period, Cox was lower while exercising than in the control condition (1.88 ± 0.1 μmol cm, $F(1,14)=5.64$, $p=.03$, $\eta_p^2=.30$).

[HbO_2] levels recorded per condition and period negatively correlated with error rate ($r=-.40$; $p=.001$) and, more specifically, with error rate on IN trials ($r=-.39$; $p<.01$). [HbO_2] levels also negatively correlated with partial errors both on CO ($r=-.38$; $p<.01$) and IN trials ($r=-.26$; $p<.05$). Mean RT ($r=.04$; $p=.79$) and the correction rate ($r=.17$; $p=.19$) were not associated to changes in [HbO_2] levels.

3.9. Vastus lateralis activation

A significant main effect of time was observed on the VL activity ($F(1,14)=3.92$, $p<.001$, $\eta_p^2=.23$), which illustrates an increased activation of the muscle throughout exercise duration (Fig. 8). Furthermore, it is interesting to note that the RMS values are significantly and negatively correlated with exercise [HbO_2] ($r=-.23$; $p<.05$).

4. Discussion

This study aimed at investigating concomitant changes in cognitive control and Cox in the prefrontal area during strenuous exercise performed until exhaustion. In an initial period, Cox recorded from the rIFC remained unchanged by intense exercise. Also, the more pronounced drop-off of the delta curve suggested that the cognitive processes, underpinning selective response inhibition, are fully efficient in the first part of the exercise bout. Throughout intense exercise and until exhaustion Cox linearly decreased but without falling below baseline values. In the terminal period, no sign of deficit in selective response inhibition was observed. However, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed just before exhaustion. The main findings of this study are that (i) cognitive functioning evolves during exercise, (ii) intense exercise does not systematically impair cognitive performances, (iii) selective response inhibition efficiency and PFC Cox do not follow a similar dynamic, (iv) the propensity to commit impulsive errors increases and online correction of incorrect activation are disrupted near exhaustion, and (v) Cox patterns suggest a decline in hyperfrontality instead of a hypofrontality.

4.1. Intense exercise and cognitive performance

The experimental as well as theoretical literature on the cognitive effect of intense exercise converges towards an impairment of cognitive performances (Ando et al., 2005; Chmura et al., 1994; Chmura and Nazar, 2010; Cooper, 1973; Dietrich and Audiffren, 2011; McMorris et al., 2008; Yerkes and Dodson, 1908). By investigating changes in cognitive functioning through distributional analyzes, the present study highlights a facilitating effect of intense exercise on cognitive control. This effect was localized in the initial part of the exercise bout. Both RT performances and selective response inhibition were indicative of this improvement. More precisely, the exercise-related speeding effect focused on the first two quartiles of the RT distribution, *i.e.* the fastest RT. This does not appear surprising since faster RT have been consistently reported from several meta-analyzes and integrative reviews (Brisswalter et al., 2002; McMorris and Hale, 2012, Tomporowski, 2003). In contrast, the benefit of intense exercise on selective response inhibition constitutes an innovative result. Concretely, the steep negative slope of the delta plots indicates an exercise-related lower interference effect compared to rest. The activation-suppression model of Ridderinkhof (2002) proposes that such pronounced leveling-off in the delta curve is indicative of a greater ability to suppress the automatic response generated by task-irrelevant aspect of the stimulus. The fact that the correction rate remained constant during the initial part of the exercise suggests that online control mechanisms, involved in the correction of incorrect activation, are fully efficient. Together, these findings reveal that the facilitating effect usually reported during moderate exercise can also occur in the first moments of intense exercise.

A cognitive facilitation during intense exercise is not in discrepancy with the main theories on exercise and cognition. Specifically, the catecholamine theory predicts cognitive functioning impairment above the "catecholamine threshold" *i.e.* a pivotal point into the exercise-induced increase in adrenaline, noradrenaline and dopamine (Cooper, 1973; McMorris et al., 2008). During steady exercise, since the level of monoamines increases over time regardless the intensity (Chmura et al., 1998), a certain amount of time is necessary before overreaching this threshold. Accordingly, the first stages of our exercise bout may have spared

[2] During exercise, the same pattern was observed in total hemoglobin ([HbO_2]+[HHb]) which is considered another index of neural activity (e.g., Perrey, 2008). Total hemoglobin decreased from 5.49 ± 0.15 μmol cm in the initial period to 0.69 ± 0.36 μmol cm in the terminal period ($F(1,14)=28.35$, $p<.001$, $\eta_p^2=.67$).

central processes from neural noise, while simultaneously arousing central nervous system and benefiting RT. The transient hypofrontality theory (Dietrich, 2003; Dietrich and Audiffren, 2011) purports another perspective, in which exercise leads to rearrangement of neural resources within the brain. Given the limited cerebral resources, exercise would lead to their redistribution from the brain regions that are not directly involved in the management of exercise such as the PFC to motor areas. Our fatiguing exercise required an increasing magnitude of motor cortex output to increase the firing rate of motor neurons in order to compensate muscle fiber fatigue, as supported by the recorded increase in RMS value of the VL muscle. Instead of exercise intensity *per se*, maintaining the steady power output over time may thus have progressively acted in favor of a PFC down-regulation.

4.2. Exhausting exercise and cognitive performance

Based on the distribution-analytical technique and the delta plot analysis, the present results show that, when exhaustion was about to occur, selective inhibition processes remained unaltered and were thus not responsible for inferior behavioral performances. The delta curve indeed highlights that the selective inhibition is as efficient as in the control condition. Nevertheless, the number of incorrect response activations – including both overt and partials EMG errors – increased. The analyzes of the percentage of correct responses (CAF) showed that, when exhaustion was about to occur, the individual's susceptibility to produce incorrect responses increases. In other words, exhaustion state increases the strength of the automatic response capture activated by irrelevant information and more overt errors are committed.

Interestingly, the recording of the EMG of the FPB muscle involved in the cognitive task allowed us to identify partial error trials *i.e.* incorrect action impulses that were detected and successfully corrected. Accordingly, partial errors provide a direct measure of the effectiveness of the control mechanism involved in the suppression of the activation of an incorrect response. The present results showed that, in the terminal period, participants were less capable to correct incorrect action impulses during exercise than in the control condition and more overt errors were committed. This finding suggests that, near exhaustion, the online correction mechanism is disrupted and the nervous system seems not able to overcome the incorrect activation and provide the correct action. Since the participants aimed at pursuing exercise as long as they could, it is possible that, near exhaustion, motor task-related regulations (cardio-respiratory, velocity, coordination, power output) became such imperative that they were managed in priority at the expense of cognitive task-related regulations (see Section 4.4).

By dissociating our exhausting exercise into analysis periods, we also investigate a new perspective of exercise-cognition studies. This perspective, actually based on a fatigue-induced reorganization, may help to clarify current inconsistencies in the literature. Concretely, it rationalizes why a sustained cognitive solicitation during last stages of a 65-min exercise bout may reveal diminished performance (Dietrich and Sparling, 2004) while 40 min of an intermittent assessment does not (Lambourne et al., 2010), in spite of similar moderate intensities and cognitive tasks. According to this proposal, the impaired cognitive performance associated with intense exercise do not appear illogical. Indeed, fatigue development is obviously quicker at such higher intensities. Considering this accumulation of fatigue, other moderators should also be taken into account. Fitness level, for example, may explain the better (Chang et al., 2012) and steadier (Labelle et al., 2012) cognitive performance of trained participants. Beyond this, we would like to encourage the general idea of temporal differentiation within data sets to better understand integrated fatigue development.

4.3. Cerebral oxygenation and cognitive performance

In the present study, Cox recorded during exercise from the rIFC was at first at a similarly elevated level as in the control condition before linearly declining until exhaustion. This type of [HbO_2] decrease is common during exhausting exercise and in accordance with previous reports from PFC NIRS-monitoring studies (for details see Ekkekakis, 2009).

More particularly, we found that the Cox level was reduced during the terminal period of exercise compared to the control condition where the cognitive task was conducted at rest. In spite of this decline, we observed that the implementation of selective response inhibition remained fully efficient (comparable to the score of the control condition). This is intriguing since rIFC activity is an important component in inhibition processes (Aron et al., 2004) and a debilitative cognitive effect might thus be expected from its down-regulation. This report is not isolated though. A recent study observed a similar discrepancy: cognitive performance improved at moderate intensity (60% $\dot{V}O_2$) in the absence of any changes in Cox values (Ando et al., 2011). This might lead to the suggestion that an uncoupling of Cox level in PFC areas and corresponding cognitive processes may be happening. This type of uncoupling would not ineluctably hamper, but could maintain PFC functionality. As an explanation, the PFC may preserve its metabolic activity by increasing oxygen extraction from arterial vessels to compensate for reduced perfusion (Nybo and Secher, 2004). It is also possible that the Eriksen flanker task was not demanding enough to elicit observable behavioral effects from reduced Cox level.

Exercise-induced hyperventilation is considered to be the main mechanism for the lowering of cerebral blood flow and, in turn, Cox level (Ogoh and Ainslie, 2009). In our study, ventilatory muscle fatigue may have led to this progressive drift into ventilation and hypocapnia. In spite of this process, the [HbO_2] concentration never reached values lower than baseline (*i.e.* a state that could be characterized as hypofrontal). Since [HbO_2] level consistently remained positive, our Cox pattern rather supports the decline of a hyperfrontality state. This contrasts with the reticular-activating hypofrontality theory (Dietrich and Audiffren, 2011) but not with some of its principles. Specifically, when viewed in light of the redistribution of cerebral resources, some findings may be considered as a support to the theory. Indeed, one possible explanation is that PFC was progressively inhibited as a side-effect of fatigue development to favor activity in motor areas, as supported by the Cox-RMS correlation. In this case, both the correlations between [HbO_2] and error rates, between [HbO_2] and partial error rates, and CAF results near exhaustion support the hypothesis of a reallocation, since impulsive errors relate to activity of the pre-supplementary motor area (Forstmann et al., 2008).

4.4. Rationalize the relation between exercise and cognitive performance

Our results reinforce the idea of an interaction between exercise and cognition for the complete duration of an exercise bout. This interference has previously been proposed using strength (Lorist et al., 2002; Schmidt et al., 2009) and aerobic exercises (Marcora et al., 2009; McCarron et al., 2013). Accordingly, we assume that behavioral performance relative to cognitive tasks is punctual and systemic and depends on the constraints supported by the subject at a given time. This idea of a dynamical cognitive control is supported from several perspectives.

Marcora (2008, 2009) proposes a psychobiological model of

exercise, within which the anterior cingulate cortex (ACC) appears as the keystone of both exercise and cognitive parameters. ACC is known to be involved in cognitive functioning (Carter et al., 1998), the pain matrix (Peyron et al., 2000), perceived effort (Williamson et al., 2006) and effort-related decision-making. Regarding our protocol and its demands, a hyper-solicitation of the ACC over time may compromise its efficacy to deal with interfering stimuli.

The insular cortex and hypothalamus are other brain areas that are increasingly activated with fatigue development (Meyniel et al., 2013). In response to exercise duration and increasing body afferences, it is possible that these regions act to reduce basal ganglia activation. Such inhibition would prevent the subject from experiencing untolerable perceived effort or any excessive homeostasis disruption, but would be enforced at the expense of the overall performance. Indeed, basal ganglia (specifically the ventral striatum) activation determines both cognitive and motor efforts (Schmidt et al., 2012). Near exhaustion the subject may thus, voluntarily or not, opt for a facilitating strategy leading him to progressively act on the basis of impulsive activations rather than on the basis of high-order processes.

The neuro-hormonale rationale of the "catecholamines hypothesis" may also determine the way participants respond to a cognitive task during exercise (McMorris et al., 2009). Due to the role of monoamines in glycolysis, lipolysis and cardio-respiratory regulation (Borer, 2003), sustained exercise induces increases in adrenaline, noradrenaline and dopamine irrespective of its intensity (Chmura et al., 1998). Such accumulation may progressively lead to overreach the "catecholamine threshold" that would induce neural noise and contributes to the cessation of exercise-induced cognitive facilitation.

5. Conclusion

In conclusion, this study is innovative in that changes in cognitive performances during a steady exercise were characterized. The benefit of intense exercise on selective response inhibition constitutes an original result. Moreover, the use of the distribution-analytical technique highlighted that, when exhaustion was about to occur, selective inhibition processes remained unaltered. Despite this, individual's susceptibility to making fast impulsive errors increased and less efficient online correction of incorrect activation was observed, suggesting that the online correction mechanism is disrupted. Interestingly, the dynamical pattern of selective response inhibition efficiency did not follow the same pattern as [HbO_2], letting Cox-related explanations of cognitive functioning during exercise uncertain. These results reinforce the idea of a complex interaction between exercise and cognition and include fatigue stressors as a determinant component into cognitive performances.

Declaration of interest

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

Acknowledgements

The authors would like to thank Pelle Bernhold for his constructive help in the study reasoning, and Laura Gray for English revisions. The Brainbike was provided by Hitech Fitness, Valbonne, France, although the authors have no conflict of interest to report.

References

Ando, S., Kimura, T., Hamada, T., Kokubu, M., Moritani, T., Oda, T., 2005. Increase in reaction time for the peripheral visual field during exercise above the ventilatory threshold. Eur. J. Appl. Physiol. 94, 461–467.
Ando, S., Kokubu, M., Yamada, Y., Kimura, M., 2011. Does cerebral oxygenation affect cognitive function during exercise? Eur. J. Appl. Physiol. 111, 1973–1982.
Aron, A.R., Robbins, T.W., Poldrack, R.A., 2004. Inhibition and the right inferior frontal cortex. Trends Cogn. Sci. 8, 170–177.
Aron, A.R., Robbins, T.W., Poldrack, R.A., 2014. Inhibition and the right inferior frontal cortex: one decade on. Trends Cogn. Sci. 18, 177–185.
Beer, A., 1851. Versuch der Absorptions-Verhältnisse des Cordierites für rothes Licht zu bestimmen. Ann. Phys. Chem. 84, 37–52.
Bhambhani, Y., Malik, R., Mookerje, S., 2007. Cerebral oxygenation declines at exercise intensities above the respiratory compensation threshold. Respir. Physiol. Neurobiol. 156, 196–202.
Boas, D.A., Gaudette, T., Strangman, G., Cheng, X., Marota, J.J.A., Mandeville, J.B., 2001. The Accuracy of Near Infrared Spectroscopy and Imaging during Focal Changes in Cerebral Hemodynamics. NeuroImage 13 (1), 76–90.
Borer, K.T., 2003. Exercise Endocrinology. Human Kinetics, Champaign, IL.
Borg, G., 1998. Borg's Perceived Exertion and Pain Scales. Human Kinetics, Champaign, IL.
Brisswalter, J., Collardeau, M., Arcellin, R., 2002. Effects of acute physical exercise characteristics on cognitive performance. Sports Med. 32, 555–566.
Carter, C.S., Braver, T.S., Barch, D.M., Botvinick, M.M., Noll, D., Cohen, J.D., 1998. Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280 (5364), 747–789.
Chang, Y.K., Labban, J.D., Gapin, J.I., Etnier, J.L., 2012. The effect of acute exercise on cognitive performance: a meta-analytic review. Brain Res. 1453, 87–101.
Chmura, J., Nazar, K., Kacuba-Uscilko, H., 1994. Choice reaction time during graded exercise in relation to blood lactate and plasma catecholamine thresholds. Int. J. Sports Med. 15 (4), 172–176.
Chmura, J., Krysztofiak, H., Ziemba, A.W., Nazar, K., Kacuba-Uscilko, H., 1998. Psychomotor performance above and below the bloos lactate threshold. Eur. J. Appl. Physiol. 77, 77–80.
Chmura, J., Nazar, K., 2010. Parallel changes in the onset of blood lactate accumulation (OBLA) and threshold of psychomotor performance deterioration during incremental exercise after training in athletes. Int. J. Psychophysiol. 75, 287–290.
Cooper, C.J., 1973. Anatomical and physiological mechanisms of arousal with specific reference to the effects of exercise. Ergonomics 16, 601–609.
Cope, M., 1991. The Development of a Near-Infrared Spectroscopy System and Its Application for Noninvasive Monitoring of Cerebral Blood and Tissue Oxygenation in the Newborn Infant. Univ. College London, London (Ph.D. thesis).
Davranche, K., Hall, B., McMorris, T., 2009a. Effect of acute exercise on cognitive control required during an Eriksen flanker task. J. Sport Exerc. Psychol. 31, 628–639.
Davranche, K., Paleresompoulle, D., Pernaud, R., Labarelle, J., Hasbroucq, T., 2009b. Decision making in elite white-water athletes paddling on a kayak ergometer. J. Sport Exerc. Psychol. 31 (4), 554–565 (2009 Aug).
Davranche, K., McMorris, T., 2009. Specific effects of acute moderate exercise on cognitive control. Brain Cogn. 69 (3), 565–570.
Delp, M.D., Armstrong, R.B., Godfrey, D.A., Laughlin, M.H., Ross, C.D., Wilkerson, M. K., 2001. Exercise increases blood flow to locomotor, vestibular, cardiorespiratory and visual regions of the brain in miniature swine. J. Physiol. 533, 849–859.
Delpy, D.T., Cope, M., Zee, P., van der., Arridge, S., Wray, S., Wyatt, J., 1988. Estimation of optical pathlength through tissue from direct time of flight measurements. Phys. Med. Biol. 33, 1433–1442.
De Pauw, K., Roelands, B., Cheung, S.S., de Geus, B., Meeusen, R., 2013. Guidelines to classify subject groups in sport-science research. Int. J. Sport Physiol. Perform. 8 (2), 111–122.
Dietrich, A., 2003. Functional neuroanatomy of altered states of consciousness: the transient hypofrontality hypothesis. Conscious. Cogn. 12, 231–256.
Dietrich, A., 2009. The cognitive neuroscience of exercise: the transient hypofrontality theory and its implications for cognition and emotion. In: McMorris, T., Tomporowski, P.D., Audiffren, M. (Eds.), Exercise and Cognitive Function. Wiley, New-York, pp. 69–90.
Dietrich, A., Audiffren, M., 2011. The reticular-activating hypofrontality (RAH) model of acute exercise. Neurosci. Biobehav. Rev. 35, 1305–1325.
Dietrich, A., Sparling, P.B., 2004. Endurance exercise selectively impairs prefrontal-dependent cognition. Brain Cogn. 55, 516–524.
Duncan, A., Meek, J.H., Clemence, M., Elwell, C.E., Tyszczuk, L., Cope, M., Delpy, D., 1995. Optical pathlength measurements on adult head, calf and forearm and the head of the newborn infant using phase resolved optical spectroscopy. Phys. Med. Biol. 40 (2), 295.
Eriksen, B.A., Eriksen, C.W., 1974. Effects of noise letters upon the identification of a target letter in a non search task. Percept. Psychophys. 16, 143–149.
Ekkekakis, P., 2009. Illuminating the black box: investigating prefrontal cortical hemodynamics during exercise with near-infrared spectroscopy. J. Sport Exerc. Psychol. 2009, 505–553.
Forstmann, B.U., van den Wildenberg, W.P.M., Ridderinkhof, K.R., 2008. Neural mechanisms, temporal dynamics, and individual differences interference control. J. Cogn. Neurosci. 20, 1854–1865.
Gross, P.M., Marcus, M.L., Heistad, D.D., 1980. Regional distribution of cerebral blood

flow during exercise in dogs. J. Appl. Physiol. 48, 213–217.
Hasbroucq, T., Possamaï, C.-A., Bonnet, M., Vidal, F., 1999. Effect of the irrelevant location of the response signal on choice reaction time: an electromyographic study in humans. Psychophysiology 36 (4), 522–526.
Hazeltine, E., Poldrack, R., Gabrieli, J.D., 2000. Neural activation during response competition. J. Cogn. Neurosci. 12 (suppl. 2), 118–129.
Holschneider, D.P., Maarek, J.-M.I., Yang, J., Harimoto, J., Scremin, O.U., 2003. Functional brain mapping in freely moving rats during treadmill walking. J. Cereb. Blood Flow Metab. 23, 925–932.
Hoshi, Y., Kobayashi, N., Tamura, M., 2001. Interpretation of near-infrared spectroscopy signals: a study with a newly developed perfused rat brain model. J. Appl. Physiol. 90, 1657–1662.
Joyce, J., Smyth, P.J., Donnelly, A.E., Davranche, K., 2014. The Simon task and aging: does acute moderate exercise influence cognitive control? Med. Sci. Sports Exerc. 46 (3), 630–639.
Kemppainen, J., Aalto, S., Fujimoto, T., Kalliokoski, K.K., Langsjo, J., Oikonen, V., Rinne, J., Nuutila, P., Knuuti, J., 2005. High intensity exercise decreases global brain glucose uptake in humans. J. Physiol. 568, 323–332.
Labelle, V., Bosquet, L., Mekary, S., Bherer, L., 2012. Decline in executive control during acute bouts of exercise as a function of exercise intensity and fitness level. Brain Cogn. 81, 10–17.
Lambourne, K., Audiffren, M., Tomporowski, P.D., 2010. Effects of acute exercise on sensory and executive processing tasks. Med. Sci. Sport Exerc., 1396–1402.
Lambourne, K., Tomporowski, P.D., 2010. The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. Brain Res. 1341, 12–24.
Lorist, M.M., Kernell, D., Meijman, T.F., Zijdewind, I., 2002. Motor fatigue and cognitive task performance in humans. J. Physiol. 545 (1), 313–319.
Mansouri, C., L'Huillier, J.P., Kashou, N.H., Humeau, A., 2010. Depth sensitivity analysis of functional near-infrared spectroscopy measurement using three-dimensional Monte Carlo modelling-based magnetic resonance imaging. Lasers Med. Sci. 25 (3), 431–438.
Marcora, SM., 2008. Do we really need a central governor to explain brain regulation of exercise performance? Eur J App Physiol. 104, 929–931.
Marcora, S.M., Staiano, W., Manning, V., 2009. Mental fatigue impairs physical performance in humans. J. Appl. Physiol. 106, 857–864.
McCarron, J., Hodgson, T.L., Smith, M.F., 2013. Brain drain: evaluating thethe impact of increased cognitive load during self-paced running performance. Br. J. Sports Med. 47, 13–17.
McMorris, T., Collard, K., Corbett, J., Dicks, M., Swain, J.P., 2008. A test of the catecholamines hypothesis for an acute exercise–cognition interaction. Pharmacol. Biochem. Behav. 89, 106–115.
McMorris, T., Davranche, K., Jones, G., Hall, B., Corbett, J., Minter, C., 2009. Acute incremental exercise, performance of a central executive task, and sympathoadrenal system and hypothalamic-pituitary-adrenal axis activity. Int. J. Psychophysiol. 73, 334–340.
McMorris, T., Sproule, J., Turner, A., Hale, B.J., 2011. Acute, intermediate intensity exercise, and speed and accuracy in working memory tasks: a meta-analytical comparison of effects. Physiol. Behav. 102, 401–408.
McMorris, T., Hale, B.J., 2012. Differential effects of differing intensities of acute exercise on speed and accuracy of cognition: a meta-analytical investigation. Brain Cogn. 80, 338–351.
Meyniel, F., Sergent, C., Rigoux, L., Daunizeau, J., Pessiglione, M., 2013. Neurocomputational account of how the human brain decides when to have a break. Proc. Natl. Acad. Sci. USA 110, 2641–2646.
Nieman, D.C., Lasasso, H., Austin, M.D., Pearce, S., McInnis, S., Unick, J., 2007. Validation of Cosmed's Fitmate in measuring exercise metabolism. Research in Sports Medicine 15, 67–75.
Nybo, L., Secher, N.L., 2004. Cerebral perturbations provoked by prolonged exercise. Prog. Neurobiol. 72, 223–261.
Ogoh, S., Ainslie, P.L., 2009. Cerebral blood flow during exercise: mechanisms of regulation. J. Appl. Physiol. 107, 1310–1380.
Perrey, S., 2008. Non-invasive NIR spectroscopy of human brain function during exercise. Methods 45 (4), 289–299.
Peyron, R., Laurent, B., Garcïa-Larrea, L., 2000. Functional imaging of brain responses to pain. A review and meta-analysis. Clin. Neurophysiol. 30, 263–288.
Pontifex, M.B., Hillman, C.H., 2007. Neuroelectric and behavioral indices of interference control during acute exercise. Clin. Neurophysiol. 118, 570–580.
Ridderinkhof, K.F., 2002. Micro- and macro-adjustments of task set: activation and suppression in conflict task. Psychol. Res. 66, 312–323.
Ridderinkhof, K.R., van den Wildenberg, W.P.M., Wijnen, J., Burle, B., 2004. Response inhibition in conflict tasks is revealed in delta plots. In: Posner, M. (Ed.), Cognitive Neuroscience of Attention. Guilford Press, New York.
Rochet, N., Spieser, L., Casini, L., Hasbroucq, T., Burle, B., 2014. Detecting and correcting partial errors: evidence for efficient control without conscious access. Cogn. Affect. Behav. Neurosci. 14 (3), 970–982.
Rooks, C.R., Nathaniel, J.T., McCully, K.K., Dishman, R.K., 2010. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy: a systematic review. Prog. Neurobiol. 92, 134–150.
Rupp, T., Perrey, S., 2008. Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. Eur. J. Appl. Physiol. 102, 153–163.
Schmidt, L., Cléry-Melin, M.-L., Lafargue, G., Valabrègues, R., Fossati, P., Dubois, B., Pessiglione, M., 2009. Get aroused and be stronger: emotional facilitation of physical effort in the human brain. J. Neurosci. 29 (30), 9450–9457.
Schmidt, L., Lebreton, M., Cléry-Melin, M.-L., Daunizeau, J., Pessiglione, M., 2012. Neural mechanisms underlying motivation of mental versus physical effort. Plos Biol. 10 (2).
Shibuya, K., Tanaka, J., Kuboyama, N., Murai, S., Ogaki, T., 2004a. Cerebral cortex activity during supramaximal exhaustive exercise. Respir. Physiol. Neurobiol. 140, 165–172.
Shibuya, K., Tanaka, J., Kuboyama, N., Ogaki, T., 2004b. Cerebral cortex activity during intermittent supramaximal exercise. J. Sports Med. Phys. Fit. 44, 215–219.
Strangman, G., Franceschini, M.A., Boas, D.A., 2003. Factors affecting the accuracy of near-infrared spectroscopy concentration calculations for focal changes in oxygenation parameters. NeuroImage 18, 865–879.
Tashiro, M., Itoh, M., Fujimoto, T., Fujiwara, T., Ota, H., Kubota, K., Higuchi, M., Okamura, N., Ishi, K., Berecski, D., Sasaki, H., 2001. 18F FDG PET mapping of regional brain activity in runners. J. Sports Med. Phys. Fit. 41, 11–17.
Timinkul, A., Kato., M., Omori., T., Deocaris., C.C., Ito., A., Kizuka, T., et al., 2008. Enhancing effect of cerebral blood volume by mild exercise in healthy young men: a near- infrared spectroscopy study. Neurosci. Res. 61, 242–248.
Timinkul, A., Omori, T., Wanpen, S., Yamada, Y., Soya, H., 2010. High frequency brain waves increase depending upon exercise intensity and associated with cerebral blood volume: a simultaneous NIRS-EEG study. J. Med. Tech. Phys. Ther. 22 (1), 81–91.
Tomporowski, P.D., 2003. Effects of acute bout of exercise on cognition. Acta Psychol. 112, 297–324.
Toronov, V., Webb, A., Choi, J.H., Wolf, M., Michalos, A., Gratton, E., Hueber, D., 2001. Investigation of human brain hemodynamics by simultaneous near-infrared spectroscopy and functional magnetic resonance imaging. Med. Phys. 28.
van den Wildenberg, W.P.M., Wylie, S.A., Forstmann, B.U., Burle, B., Hasbroucq, T., Ridderinkhof, K.R., 2010. To head or heed? Beyond the surface of selective action inhibition: a review. Front. Hum. Neurosci. 4, 1–13.
Vissing, J., Anderson, M., Diemer, N.H., 1996. Exercise-induces changes in local cerebral glucose utilization in the rat. J. Cereb. Blood Flow Metab. 16, 729–736.
Williamson, J.W., Fadel, P.J., Mitchell, J.H., 2006. New insights into central cardiovascular control during exercise in humans: a central command update. Exp. Physiol. 91, 51–58.
Yerkes, R.M., Dodson, J.D., 1908. The relation of strength of stimulus to rapidity of habit-formation. J. Comp. Neurol. Psychol. 18, 459–482.

Schmit, C., & Brisswalter, J. Executive functioning during prolonged exercise: A fatigue based-neurocognitive perspective. *Under review* (in *International Review of Sport and Exercise Psychology*).

Résumé en français

Un consensus reste à établir pour expliquer la potentielle détérioration du fonctionnement exécutif à l'exercice, malgré les incidences émotionnelles, techniques ou d'endurance pour les athlètes. En particulier, les recherches récentes challengent l'idée d'un effet linéaire en 'dose-réponse' de l'intensité d'exercice sur le fonctionnement exécutif et la physiologie cérébrale. Dans ce contexte, une perspective neurocognitive du fonctionnement exécutif pendant l'exercice, basée sur le phénomène de fatigue est proposée. Cette perspective repose sur le fait que les processus 'top-down' (*i.e.,* les efforts physiques et cognitifs, ou efférences) et 'bottom-up' (*i.e.,* les sensations de l'organisme, ou afférences) inhérents à l'exercice prolongé agissent en parallèle des processus d'activation pour influencer les opérations cognitives. En accord avec ce raisonnement, le fonctionnement exécutif au cours de l'exercice prolongé est estimé être i) dynamique, ii) positivement, puis négativement impacté par l'exercice, iii) à interpréter au regard du moment d'arrêt de l'exercice plutôt que de son intensité, et iv) modulable au cours d'un exercice fatiguant.

Ce travail a permis de réaliser un état de la littérature dans le but de suggérer un rationnel neuronal du fonctionnement exécutif à l'exercice prolongé et fatiguant. Si cette perspective peut être envisagée à l'exercice aigu, son application à moyen-terme chez des sujets conduits à un état de fatigue sévère (*i.e.,* en état de surmenage fonctionnel) reste à définir.

Executive functioning during prolonged exercise: a fatigue-based neurocognitive perspective

Journal:	*International Review of Sport and Exercise Psychology*
Manuscript ID	RIRS-2016-0028
Manuscript Type:	Literature Review
Keywords:	exhaustion, self-regulation, self-control, cognitive performance, lucidity

Abstract

A consensus has yet to be reached to explain a possible executive functioning impairment during exercise, despite emotional, technical or endurance implications for athletes' performance on the field. In particular, recent researches challenge the assumption of a linear dose-response effect of exercise intensity on executive functioning and cerebral physiology. In this context, a fatigue-based neurocognitive perspective of executive functioning during exercise is proposed. The perspective lies on the fact that top-down processes (i.e., cognitive and physical efforts) and bottom-up processes (i.e., body sensations) inherent to prolonged exercise act in parallel of arousing processes to influence cognitive operations. According to this reasoning, executive functioning during prolonged exercise is believed to be i) dynamical, ii) positively, then negatively impacted by exercise, ii) to interpret in regards of exercise termination rather than exercise intensity, and iv) modulable during fatiguing exercise.

Key words: exhaustion; self-regulation; self-control; cognitive performance; lucidity

1. Introduction

What to do right now? Athletes repeatedly exposed to uncertainty under time-pressure situations such as in team or racket sports, facing self-control requirements during running or during disconcerting referee decisions, have to solve this question. Solving the issue is even more challenging alongside energetic requirements. For instance, experimental reports have demonstrated how high physiological constraints exacerbate the challenge of self-control when performing in hot conditions (Gaoua et al., 2011, 2012) or dehydrated (Cian et al., 2001). For coaches and athletes, the ability to remain lucid *during* exercise has always been of interest. In contrast, the issue has only recently been addressed within the exercise-cognition literature (McMorris et al., 2009); yet cognitive processes underlying the capacity to self-control are crucial for behavioral outcome (Miyake et al., 2000).

Executive functions are the facets of cognitive processes that specifically subserve this capacity to deliberately align behavior according to one goal (Hofmann et al., 2012). Working memory updating, behavioral inhibition and mental set shifting are certain of those functions which, due to their ubiquity, are conventionally tested in laboratory settings (Miyake et al., 2000). Executive tests (e.g., Stroop, n-back), in contrast to simple ones (e.g., simple reaction time), are specific and systematic in relying on the prefrontal cortex (PFC) circuitry (Leh et al., 2010; Poldrack & Packard, 2003). This makes PFC efficiency mandatory to implementing field-related top-down mechanisms as controlling feint- or tactic-related relevant information (Jacobson & Matthaeus, 2014; Vestberg et al., 2012). The issue of a transfer from laboratory results to everyday incidences has been debated (Morrison & Chein, 2011); however, accumulating evidence does suggest ecological implications of decontextualized cognitive activity (e.g., for executive monitoring and training) (Houben & Jansen, 2011; Houben et al., 2011; Romeas et al., 2016; Veling et al., 2011).

Since Yerkes and Dodson (1908), a great amount of research has examined the concept of an inverted U-shape response of cognitive functioning efficiency during an acute bout of incremental exercise (e.g., recently, McMorris et al., 2011; McMorris and Hale, 2012). Notably, the empirical acceptance that high intensities induce lower executive performance than moderate ones has been controversially rationalized. The arousal-cognitive performance (Yerkes and Dodson, 1908) and the catecholamine (McMorris et al., 2008) theories predict higher cognitive processes in humans to suffer from high levels of arousal and neural 'noise', respectively; in contrast, the reticular-activating hypofrontality theory (Dietrich, 2003; Dietrich and Audiffren, 2011) defends a down-regulation of the PFC. Irrespective of the rationale advanced, further, recent original researches have challenged this assumption of a linear dose-response effect of exercise on executive functioning and cerebral physiology (Chang et al., 2012). For example, moderate and high exercise intensities were found to impair and improve executive efficiency, respectively (Pontifex and Hillman, 2007; Schmit et al., 2015). In addition, executive functioning has been uncoupled from the kinetics of PFC oxygenation during exercise (Ando et al., 2011; Ogoh et al., 2014). Within this framework, a qualitative approach encompassing the discreet exercise-cognition moderators has been suggested to represent a more appropriate method accounting for the nonlinear exercise-driven executive alterations (Brisswalter et al., 2002; Chang et al., 2012; Pesce, 2009, 2012).

A cross-disciplinary analysis of the exercise-cognition interactions has previously been performed by Dishman et al. (2006), highlighting executive benefits inherent to *chronic* exercise via changes in human brain structure and functioning (e.g., via neurotrophic factors). However, authors acknowledged that brain metabolic responses to *acute* physical activity were not as yet clear beyond motor, sensory and subcortical regions (Dishman et al., 2006), therefore limiting the understanding of PFC-based executive functioning during exercise. More recently, McMorris et al. (2016) proposed a neurochemical perspective providing

insights to potentially interpret above inconsistency. Specifically, authors assumed both heavy and moderate intensity exercises could facilitate, as well as impair, cognition, with exercised-induced concentrations in catecholamines, serotonin, hypothalamo-pituitary adrenal axis hormones and brain-derived neurotrophic factors as the possible cornerstone distinguishing these effects (McMorris et al., 2016). The present paper aims at drawing in an alternative perspective to explain the executive decline that could be observed during an acute exercise bout; though the idea that any exercise intensity (i.e., moderate or high) has the potential to incur this decline is consistent. Accordingly, we will consider along the text an exercise as 'prolonged' since its duration is sufficient to lead to a detrimental effect in an individual's ability to self-control.

Within this context, an exhaustive critical review of the literature is not the aim of this paper – and we refer the reader to meta-analyses referenced in parts 1 and 2 for this purpose. Rather, we attempt to propose a neurocognitive reasoning of the exercise-related executive impairment that could potentially help rationalizing sport behavior and performance. After suggesting that executive functioning during exercise must be interpreted (and studied) as dynamical, we discuss experimental findings of neural and cognitive processes that have the potential to explain the pivotal change from executive benefit to impairment during exercise. We then propose a model clarifying these exercise-induced evolutive executive effects.

2. Executive response during exercise: (re)interpreting results

Studies investigating the exercise-cognition relationship have examined cognitive processes using various types of task (e.g., information processing, reaction time, attention, crystallized intelligence, executive functioning) and, overall, highlighted a facilitating effect of exercise on cognitive performances (for details, see Lambourne & Tomporowski, 2010; McMorris & Graydon, 2000; Tomporowski, 2003). Notably, executive functions

systematically require the implementation of the PFC and, as such, differ from more simple cognitive processes. Meta-analyses specifically examining executive functioning performance during exercise have reported contrasting results. For example, Chang et al. (2012) observed a positive effect of exercise on executive functioning that was the largest among the different cognitive performances investigated (e.g., information processing, reaction time, memory). In contrast, Lambourne and Tomporowski (2010) and McMorris and Hale (2012) reported minimal effects of exercise on executive functioning, while McMorris et al. (2011) demonstrated a detrimental effect of moderate intensity exercise on working memory tasks. As explanations, are currently contrasted factors such as the type of studies included in meta-analyses, participants' fitness level, exercise mode and intensity, or task difficulty. However, regardless these influences, this is a common point that the experimental exercise-related cognitive effect is not apprehended as dynamical.

Methodologically, original studies that have characterized high-order processes *in situ* of exercise have mainly addressed the time-window of cognitive data recorded as a whole i.e., they provided time-averaged cognitive performances (e.g., Ando et al., 2011; Chmura et al., 1994; Chmura and Nazar, 2010; Dietrich, 2003; McMorris et al., 2009; Ogoh et al., 2014; Pontifex and Hillman, 2007). For instance, Dietrich and Sparling (2004) reported participants' accuracy from an entire 25min-testing period on the Wisconsin Card Sorting Task. Davranche and McMorris (2009) reported distribution analyses of reaction times recorded over a ~18min-testing period on the Simon task. Del Giorno et al. (2010) provided one measurement of executive functioning (using the Contingent Continuous Performance Task and the Wisconsin Card Sorting Task) over a ~25min exercise bout. Such pooling is restrictive in that it hinders the access to 'snapshots' of executive functioning efficiency *along* the exercise bout i.e., a dynamical perspective.

The temporal dynamics of executive functioning during exercise has received little attention – experimental studies mainly addressing 'temporality' as pre- *vs.* during- *vs.* post-exercise assessments (Chang et al., 2012). Few studies, nonetheless, allow grasping such dynamics. Lorist et al. (2002) reported an increasingly detrimental effect on both cognitive and motor performances during the concomitant realization of a choice reaction time task and a fatiguing submaximal contraction: the more the time-on-tasks elapsed, the more error rate *and* force variability increased. The completion of each task alone did not reveal such impairment, leading authors to conclude that concurrent tasks were sharing similar nervous system resources. Using electroencephalographic signals as indices of executive functioning, Grego et al. (2004) highlighted an evolutive allocation of attentional resources over a 3h-moderate intensity exercise and the completion of an auditory oddball task. In particular, the increased magnitude of the P300 wave (an index of working memory updating) appeared specific to the second hour of exercise and declined during the last hour; this decline paralleled increases in processing latency and scores of perceived exertion. Results were discussed in terms of concomitant *but* predominant effects of arousal or central fatigue as exercise lasted. Through a similar 3h-protocol, the same team of researchers replicated this observation (both in euhydrated and dehydrated subjects) demonstrating an initial exercise-induced facilitating effect on executive performances, followed by a cognitive inhibition that extended to simpler processes (i.e., perceptual performances) (Grego et al., 2005). More recently, a comparable observation has been reported when comparing the initial *vs.* terminal period of an inhibition task performed throughout a heavy exhausting fixed-paced aerobic exercise (Schmit et al., 2015). Specifically, when subjects approached exhaustion, the initial improvement observed in executive functioning eclipsed and error rate increased. This increase was associated to an exacerbated impulsivity, highlighted through electrography of the thumb muscles involved in the task (Schmit et al., 2015). The work by Olson et al. (2015)

also adds to these findings. Specifically, during the last stage of a 31-min cycling task, the authors showed attenuation in the initial exercise-induced increase of the P300 amplitude, despite no changes in behavioral performances. This discrepancy between electrical and behavioral measures is however not incoherent regarding the exercise bout duration, since electrical changes of the brain are detectable prior to behavioral manifestations (e.g., Hocking et al., 2001). Together, these observations highlight the necessity to consider executive functioning during exercise as dynamical over a given time period.

Few studies have sequenced cognitive measures during exercise, as presented above. Importantly, a dynamical perspective may provide a rationale as to why a sustained executive solicitation during last stages of a 65-minute exercise bout reveals diminished cognitive performance while 40 minutes of an intermittent assessment do not, in spite of similar moderate intensities and tasks (Dietrich and Sparling, 2004; Lambourne et al., 2010). It could also explain the exercise-related facilitating vs. detrimental effect on executive functioning of an 8-min vs. 30-min exercise bout despite similar intensities (Del Giorno et al., 2010; Lucas et al., 2012). Furthermore, it could account for the better (Bullock & Giesbrecht, 2014; Chang et al., 2012) and steadier (i.e., lower variability) (Labelle et al., 2012; Zwierko et al., 2014) executive performances that are observed in trained subjects during exercise, when compared to their untrained counterparts upon similar protocols. According to a dynamical response of executive functioning during exercise, the empirical association between intense exercise and impaired executive performance (Ando et al., 2005; Chmura et al., 1994; Chmura and Nazar, 2010; McMorris et al., 2011; McMorris and Hale, 2012) might also be reviewed. Specifically, considering that executive functioning impairment would be precipitated during heavy exercise compared to moderate intensity exercises, has the advantage of not precluding executive benefits to be temporarily reported during heavy exercise (e.g., Schmit et al., 2015). Further, this does not preclude heavy exercise to potentially impair low demanding processes

(i.e., congruent stimuli) when exercise is driven to exhaustion (McMorris et al., 2009), or moderate intensity, long duration exercise to induce executive functioning impairment (e.g., Grego et al., 2004, 2005).

According to this reasoning, the representation of an inverted U-shaped relationship between exercise and executive functioning could provide coherence within experimental reports, in so far as it is brought close to the phenomenon of exhaustion, rather than to exercise intensity. Indeed, both an enhancement and an impairment could be observed in executive functioning during exercise (even consecutively, in the case of prolonged exercise) regardless its intensity. Such a dynamical interpretation could be reasoned on the basis of neural (de)activations.

3. Cognitive and neural implications of executive decline during exercise

The exercise-induced arousing effect has been extensively, and unequivocally, reported in the past to account for the positive incidence of acute physical activity on cognitive performances (e.g., Everitt & Robbins, 1997; Meeusen et al., 2006; McMorris et al., 2016). Briefly, the release during exercise of monoamines (i.e., mostly norepinephrine, dopamine) from subcortical areas towards prefrontal regions mediates functions involving filtering inputs and energizing outputs (Ramos & Arnsten, 2007; Robbins & Everitt, 2007). Therefore, information processing necessitating enhanced signal-to-noise ratio (i.e., to discriminate a given stimulus), decisional processes or response mobilization benefit from monoamines release (Ramos & Arnsten, 2007; Robbins & Everitt, 2007). In contrast to the uncontroversial arousing effect of exercise, mechanisms suggested to originate the executive decline during exercise appear less consensual. The following sections address potential cognitive and neural facets of this phenomenon.

The idea of global and fatigable cognitive-physical resources has been extensively developed in the past (e.g., cognitivo-energetic models [Kahneman, 1973; Humphreys and Revelle, 1984; Hockey, 1997, Sanders, 1983]). As a result of technical improvements in neuroimagering, however, critique surfaced front these models due to the lack of distinction between explicit and implicit processes of cognitive functioning (e.g., Dietrich & Audiffren, 2011). Indeed, this distinction is the basis of dual-processing models of neuroanatomical systems that account for reflective vs. impulsive processes of information processing (Deutsch and Strack, 2006; Evans, 2008). Reflective processes require the implementation of PFC areas for goal-directed operations (Ashby and Casale, 2002; Dehaene and Naccache, 2001), while impulsive processes mostly involve basal ganglia, cerebellum and supplementary motor areas within associative mechanisms triggered by simple cognitive tasks (Forstmann et al., 2008). During exercise, Dietrich and Audiffren (2011) propose that the implicit system is enhanced as a result of increasing monoamines activity, and the explicit system inhibited consecutive to cerebral resources redistribution towards more demanding, motor areas. This inhibition is thought to limit the activation of the PFC and thereby induce a decline in executive performance during exercise (Dietrich & Audiffren, 2011). To date, however, the theory of a hypofrontality still needs to be experimentally validated – for example given the increased frontal resources noticed at almost all exercise intensities (e.g., Rooks et al., 2010). Though, the propensity of increased impulsive behaviors does seem effective during prolonged exercise (see part 2) and could actually relate to the prevalence of motor over frontal activations (Forstmann et al., 2008; Kim and Lee, 2010; van Duinen et al., 2007). In other words, beyond mechanistic incoherence, the *functional* perspective of diametrically influenced cognitive systems during a prolonged exercise bout (i.e., increased impulsivity associated to an executive decline) is theoretically supported. Laboratory studies on attentional focus during exercise provide insights in this direction.

Dynamic approaches claim the emergence within one system of spontaneous patterns on the basis of its intrinsic dynamics which, in the exercise-cognition field, has been studied relative to the nonlinear dimension of attentional focus (i.e., what the subject is focusing on) (Kelso, 1995; Nowak and Vallacher, 1998; Van Orden et al., 2003). Of note, attentional focus is not a measure of executive functioning *per se*; however, sustained attention (i.e., the ability to remain focus on a stimulus over time) does require the ability of the working memory to implement and maintain a specific task-set focus in the PFC (Hofmann et al., 2012). Recently, exhausting exercise was found to induce a progressive and involuntary narrowing of this ability. More precisely, as exhaustion became close the increasing prevalence of exercise-related thoughts (e.g., focus on the stride, on the breathe) hindered the subject's ability to *intentionally* focus towards exercise-unrelated thoughts (e.g., thinking about homework) (Ballagué et al., 2012; Tenenbaum and Connolly, 2008). In reference to dual-processing models, authors and others have interpreted this growing inability as a "switch from an implicit (…) mode of task execution to a consciously aware attempt to regulate behavior" (Edwards and Polman, 2013). In this perspective, exercise awareness and physical monitoring are believed to spontaneously develop during prolonged exercise at the expense of working memory capacity – initially fully efficient – to deal with task-unrelated thoughts (Aitchinson et al., 2013; Swart et al., 2012). Further, given executive functioning impairment during prolonged exercise is incoherent with the idea of a hypofrontality, mechanisms behind this narrowing process rather suggest physical and self-control demands competing for similar, but limited, PFC-based processing (Fox et al., 2005; see below). Of note, this competition could explain McMorris et al.'s anecdotal observation (2009) of detrimental incidence of exhausting exercise even on congruent trials. Processes behind these trials are commonly reported as improved during exercise since they mainly require autonomous and perceptive/attentional computations and only minimally involve the executive component

(Eriksen and Eriksen, 1974). However, the "possibility that the heavy exercise was such that it resulted in negative effects for both conditions (i.e., congruent and incongruent trials) cannot be discounted" (McMorris et al., 2009).

Together, previous reports suggest executive functioning during prolonged exercise (either moderate or intense) to become debilitative as exhaustion becomes close. To date, metabolic explanations have constituted the most accomplished rationale of this inhibitory process (Etnier, 2015; Piepmeier and Etnier, 2015; Singh and Staines, 2015) (we refer the reader to McMorris and Hale, 2012 and McMorris et al., 2016 for mechanistic reviews). Besides these propositions, however, recent researches provide neurofunctional insights relative to the possible role of increasing efferent and/or afferent mechanisms (i.e., what we mentioned as 'constraints') during exercise in executive functioning decline.

Extensive projections from the PFC (e.g., dorsolateral PFC, DLPFC, anterior cingulate cortex, ACC) to motor areas constitute the bases for a substantial depletion of frontal resources as soon as body efferences become increasingly necessary (Dettmers et al., 1995, 1996; Paus, 2001). Specifically, frontal implications have been evidenced during fatiguing exercise or sustained accurate motor control, both likely illustrating cognitive control exertion over motoric pathways (Fuster, 1997; Jenkins et al., 1994) (for a review, see Tanaka and Watanabe, 2012). In parallel, PFC activity has repeatedly been shown mandatory to attenuate amygdala reactivity to aversive stimuli, as those arising from exhausting exercise (e.g., thirst- or pain-related) (Buhle et al., 2013; Ochsner and Gross, 2005, 2008). Specifically, it is known that the stronger this PFC-amygdala connectivity, the greater the modulation of the amygdala activity (Cohen et al., 2015). This frontal-dependent inhibitory process further not restricts to amygdala reactivity but extends to brain areas selectively modulated by afferent mechanisms during exercise (e.g., insular cortex and ACC) (Hilty et al., 2011; Jouanin et al., 2009). As

such, due to its implication in top-down and bottom up processes related to exercising, the PFC activity is argued to play a key role in the timing of exercise termination (Meeusen et al., 2016). Notwithstanding, this role could incur detrimental incidences on PFC-related functions (e.g., executive ones), especially given one individual's limited information-processing capacity. Further, this role could partly be mediated upstream of the PFC, at a subcortical level.

Within basal ganglia, the ventral striatum has been identified as a generic node whose individual motivation-dependent activity was predictive of both motoric *and* executive neural activations and, ultimately, performances (Schmidt et al., 2012). Put another way, the higher the motivation, the greater the striatal activity, and the stronger the achieved physical performance (via the putamen then the primary motor cortex) *and* cognitive performance (via the caudate nucleus then the DLPFC). Thus, besides arousing effects of exercise, this node may represent a 'common denominator' also energizing behavioral performance. Interestingly, this dual circuitry is also regulated by the insular cortex activity (Schmidt et al., 2009; Sridharan et al., 2008) – a proprioceptive area associated with self-awareness. This connectivity is of particular interest from an executive viewpoint. Indeed, the insula is known to encode the 'cost evidence signal' inherent to self-paced physical tasks (Meyniel et al., 2013, 2014). More precisely, as exertion lasts, this signal linearly accumulates up to a maximal neural activation concomitant to exercise termination. Within this framework, executive functioning during exercise appears ratio-dependent. In particular, in regards of task complexity and initial motivation, executive performance impairment during exercise could be function of both the concomitant motor and cognitive demands *and* of the simultaneous level of exertion-encoded cost.

In some cases, it is possible that the concomitant motor and cognitive demands also be consubstantial at a cortical level i.e., rely on similar cortical circuits. For instance, due to its

multi-functionality i.e., in the pain matrix (Peyron et al., 2000), perceived exertion modulation (Williamson et al., 2006), effort-related decision-making in rodents (Walton et al., 2003, 2006) and human executive functioning (Carter et al., 1998), the ACC is suggested to become selective in critical situations by determining whether and how much cognitive control allocate to a task (Shenhav et al., 2013). Accordingly, in cases ACC is necessitated to adjust behavior *in situ* of exercise (e.g., for conflict monitoring, Botvinick et al., 2004), this multi-functionality would reduce ACC sensitivity to accumulating interferences and the subsequent implementation of the PFC (Kerns et al., 2004). Further, such neural overlap could not be specific to inhibiting impulse but may also occur in response to mental fatigue. In particular, the ACC has been suggested to mediate the feeling of tiredness consecutive to a prolonged period of demanding cognitive activity and leading to impaired endurance performance (Marcora et al., 2009). Moreover, the evidence of a neural overlap between motor and cognitive demands has been reported when up-regulating the difficulty of cognitive tasks performed while exercising was reported to increasingly impair strength and endurance performances (Bray et al., 2012; McCarron et al., 2013). In these two studies, changes in ACC and PFC activities were assumed to drive the alterations in physical performance.

These reports are nascent. Future neuroimaging-based research should highlight the role of prolonged exercise in activating/inhibiting neural circuitry of cognitive performances. However, although in its premise, a neurocognitive approach may subsume the reasoning of a dynamical response of executive functioning during prolonged exercise. According to this approach, top-down (i.e., cognitive and physical efforts, or efferences) and bottom-up processes (i.e., body monitoring, or afferences) of exercise would act in parallel of arousing mechanisms.

4. A neurocognitive rationale for executive functioning decline during fatiguing exercise

Drawing on the two previous sections, it is suggested that executive functioning during prolonged exercise is i) dynamical, ii) positively, then negatively impacted by exercise, and ii) to interpret in regards of exercise termination.

As mentioned, contemporary reviews insist on the highly inter-personal executive response to exercise, for example relative to fitness level, exercise mode automation or exercise intensity (Lambourne & Tomporowski, 2010; McMorris et al., 2011; McMorris and Hale, 2012). To formalize such complexity and integrate psychophysiological interactions during exercise, the intra-personal concept of *fatigue* is classically advanced (Gandevia, 2001). We below propose a fatigue-based neurocognitive perspective of executive functioning during exercise (Fig. 1), which could be of relevance given that mechanisms of physical fatigue might transpose to 'self-regulatory fatigue' i.e., ego depletion (Evans et al., 2015). Regardless its purpose (mental, muscular, spinal, supraspinal), fatigue has indeed consistently been depicted as a loss in efficiency i.e., a progressive deterioration in the demand/effort ratio (e.g., Boksem & Tops, 2008; Joyner & Coyle, 2008). Therefore, we will refer as to 'fatiguing exercise' an exercise-induced loss of efficiency in motor and/or cognitive processes that alter the ability to self-regulate (i.e., to implement executive functioning) and, ultimately, behavioral performance.

Firstly, as for autonomous processes, exercise (irrespective of its intensity) is thought to benefit executive functioning, through increased frontal concentrations in dopamine and noradrenalin. This has been shown both in animal and human studies. In parallel of arousing processes, actually, also run processes inherent to physical demand and potentially requiring PFC activity (e.g., exercise-induced muscle soreness, breathing). Importantly, since exercise is in its premises, the subject fit and/or the exercise mode automated, such requirement could remain minimal as motor functions are fully efficient (i.e., small additional involvement of the

PFC activity in supplement to the cognitive demand). As a consequence, performances relating to working memory updating, impulse inhibition or mental set shifting would mainly be arousal-dependent, and thus improve.

As exercise prolongs, however, changes in afferent/efferent mechanisms at play (e.g., pain, stride monitoring) increase the self-regulatory demand to maintain exercise intensity. Since the ACC and the PFC have been shown to play a direct or indirect role in managing these mechanisms, these changes pose an additional challenge to frontal areas. Importantly, due to the limited capacity of information processing, this challenge could evolve to such an extent that it comes to compensate the initial positive effects of exercise and progressively brings executive performance back to baseline levels. Mechanistically, a neural competition for frontal resources between task- and exercise-related demands could occur and negate arousal benefits. Of note, it has been proposed that, in such constraining situations, an individual could prioritize cognitive *or* physical performance by selectively attenuating self-control decrement in one of the tasks (Abernethy, 1988; Huang and Mercer, 2001). While appealing, this proposal has however received little empirical support.

As long as exercise continues beyond this 'back-to-square-one' point, an increasingly detrimental effect is expected in executive performance. Indeed, despite measurable, the arousing effect from exercise is thought to become insufficient to counteract the ineluctable drift of the *relative* physical demand (e.g., loss in muscular or supraspinal efficiency). In a neurocognitive perspective, exercise-related fatigue would then manifest in executive functioning becoming less effective than at rest! Delaying the detrimental effect of exercise here suggests a down-regulation in PFC-related exertion requirements (e.g., alleviating pain) or an up-regulation of arousing processes (e.g., increasing motivation).

< insert Figure about here >

5. Practical implications

Three potential implications are discussed. They relate to the interpretation of the Borg's scale (1998) in the context of the proposed model, and to potential ecological incidences and modulations of the progressive inability to self-control efficiently during exercise.

Scores of rating of perceived exertion (RPE) are widespread in sport sciences, and considered as a practical tool to estimate exercise-associated mental load underpinning exhaustion i.e., the conscious sensation of how hard, heavy and strenuous effort is (Borg, 1998). However, since this sensation is suggested to reflect top-down processes (i.e., increased prefrontal, premotor and motor functions) rather than the integration of bottom-up afferences (de Moree et al., 2012, 2014; Marcora, 2009; Takarada et al., 2014), RPE might not constitute a reliable marker to predict exercise-related executive impairment. Indeed, accordingly, RPE scores would evolve in proportion to efferent mechanisms but irrespective of afferences, which also potentially require top-down control from the PFC (e.g., for inhibitory control over amygdala activity). As a result, changes in executive performance could manifest in the absence of changes in RPE (e.g., in the case thirst is not quenched, or stitch while running). This uncoupling has not been tested; however, Dietrich and Sparling (2004) and Lambourne et al. (2010), when using similar cognitive tasks but exercise of different durations, did report different cognitive performances with similar RPE (~13 on a 6-to-20 scale).

Self-control failure has been associated to behavioral and social problems, such as drug abuse, obesity, violent acts, eating disorders (Muraven and Baumeister, 2000; Wills and Stoolmiller, 2002). In sport, many components of motor function rely on the efficiency of automatic processes; however, decline in self-control efficiency could alter various aspects of performance e.g., tactical, technical, emotional, intermittent and endurance dimensions (Badin

et al., 2016; Grillon et al., 2015; Marcora et al., 2009; Smith et al., 2014). For instance, self-control failure was found to incur diminished performance on basketball free-throw shooting and dart-tossing tasks – although this failure was induced using mental fatigue and not physical fatigue (Englert & Bertrams, 2012; McEwan et al., 2013). During prolonged exercise, presumably, the effects of self-control failure could extend to tactics omission (e.g., digressing from playmates' moves), difficulty in updating situation (e.g., systematically falling in opponent's feint in dual sports; nutritional, material or pathways forgettings in single sports), increased injury risks or impulsive actions (e.g., game rules transgression, act of violence). Given the potential of executive functioning efficiency to influence behavioral outcomes, coaches should remain reactive to players' primary acts of self-control failure while, on the long-term, facilitating individual and team habits of self-control failure management (Vealey, 2007). Similarly, athletes could gain from i) training self-control capacity, ii) using economic routines minimizing the irrelevant use of executive functioning during competition (i.e., dealing only with performance-related thoughts), and iii) learning and interpreting signs of self-control deficit (e.g., losing balance, mind-wandering in important situations). As soon as exercise becomes fatiguing, however, appropriate external strategies seem to encourage to benefiting executive functioning and delaying the decline in cognitive performance.

Progressive nociceptive afferences from digestive systems, hypoxia and dyspnea, hypoglycemia, hypohydration or hyperthermia are possible correlates of exercise, and affect independently both physical (see Millet et al., 2001) and executive functioning performance (Accarino et al., 1997; Bartholomew et al., 1999; Brands et al., 2005; Eccleston and Crombez, 1999; Lieberman, 2007; Lorist et al., 2005; Racinais et al., 2008). Specifically, these physiological status develop as exercise prolongs and, thus, parallel sensations like exercise-induced muscle or joint soreness. Importantly, each of these factors has been shown to alter

insular and thalamic activities before reaching consciousness (Bonaz, 2003; Egan et al., 2003; Henderson et al., 2007; Qian et al., 2014; The et al., 2010; Thomas et al., 2010; von Leupoldt et al., 2008), which makes them of relevance from a neurocognitive perspective of executive functioning during exercise. Indeed, in this perspective, any tools reducing these physiological constraints, or increasing the level of arousal, could have the potential to preserve executive performance when exercise becomes fatiguing. For instance, following consumption of an optimized citrus-flavored water ice, or following mouth rinsing with caffeine, respectively, Labbe et al. (2011) and De Pauw et al. (2015) reported improved activation of cortical circuits regulating working memory along with better executive performance. Bandelow et al. (2010) found a 50% correlation between core temperature and plasma glucose level, then suggesting that carbohydrates from sport drinks could partially attenuate the effects of hyperthermia and physical fatigue on cognitive performances. Gaoua et al. (2011) also showed that cold packs applied upon the body counteracted hyperthermia-related decline in executive performance; a benefit that could relate to thermoregulation-based improved activation of the PFC (Muzik & Diwadkar, 2016). Although these studies were not designed to investigate strategies counteracting exercise-related decline in executive functioning, they provide useful practical and mechanistic insights relative to ergogenic manipulations known to regulate central nervous system functioning (Rattray et al., 2015).

Together, these implications underlie the possibility for coaches and athletes to recognize and possibly counteract the effect of fatiguing exercise on cognitive performance. However, they also highlight the difficulty to anticipate self-control failure. Since such phenomenon could have serious consequences (e.g., for health purposes), future research should aim at identifying objective, easy-to-use, non-invasive and not easily manipulated tools enabling to predict/detect when exercise becomes fatiguing for athletes.

6. Conclusion

The aim of the present article was to fit experimental reports from the exercise-cognition literature together, and together with neurocognitive findings in an interactive framework. A dynamical, fatigue-based pattern of executive functioning during exercise could explain the positive and negative effects of both moderate intensity and heavy exercise on processes as working memory, impulse inhibition or mental set shifting. In particular, several cortical and subcortical areas involved both in exercise maintenance and high-order operations could progressively influence the ability to deal with complex operations during exercise. This reasoning could enable optimizing athletes' behavioral efficacy when self-regulatory failure becomes apparent during prolonged exercise.

The proposed model provides support for the potential effectiveness of long-term strategies' manipulating both cognitive and physical dimensions of athletes' training programs. Cross-over improvements between *chronic* exercise and cognition have already been evidenced; in contrast, recent innovative findings suggest cross-training effects between self-regulatory improvements and physical, aerobic performances (e.g., Bray et al., 2015). Neuroimaging techniques should enable determining underlying neural overlaps of such parallel effects, and lead to develop practical means discriminating when and how exercise becomes fatiguing for athletes.

References

Abernethy, B., 1988. Dual-task methodology and motor skills research: some applications and methodological constraints. J Hum Mov Stud 14, 101e32.

Accarino, A.M., Azpiroz, F., Malagelada, J.R., 1997. Attention and distraction: Effects on gut perception. Gastroenterology 113(2), 415–422.

Aitchison, C., Turner, L.A., Ansley, L., Thompson, K.G., Micklewright, D., St Clair Gibson, A., 2013. Inner dialogue and its relationship to perceived exertion during different running intensities. Perceptual and Motor Skills 117, 11–30.

Amann, M., Eldridge, M.W., Lovering, T.A., Stickland, M.K., Pegelow, D.F., Dempsey, J.A., 2006. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. J Physiol 575(3), 937–952.

Ando, S., Kimura, T., Hamada, T., Kokubu, M., Moritani, T., Oda, T., 2005. Increase in reaction time for the peripheral visual field during exercise above the ventilatory threshold. Eur J Appl Physiol 94, 461–467.

Ando, S., Kokubu, M., Yamada, Y., Kimura, M., 2011. Does cerebral oxygenation affect cognitive function during exercise? Eur J Appl Physiol 111, 1973–1982.

Ashby, G.F., Casale, M.B., 2002. The cognitive neuroscience of implicit category learning. In: Jiménez, L. Attention and Implicit Learning. John Benjamins Publishing Company, Amsterdam & Philadelphia. pp. 109–141.

Audiffren, M., André, A., 2015. The strength model of self-control revisited: Linking acute and chronic effects of exercise on executive functions. J Sport Health Sci 4, 30–46.

Badin, O. O., Smith, M. R., Conte, D., & Coutts, A. J., 2016. Mental Fatigue Impairs Technical Performance in Small-Sided Soccer Games. *International journal of sports physiology and performance*.

Balagué, N,. Hristovski, R., Aragonés, D., Tenenbaum, G., 2012. Nonlinear model of attention focus during accumulated effort. Psychol Sport Exer 13, 591–597.

Bandelow, S., Maughan, R., Shirreffs, S., Ozgünen, K., Kurdak, S., Ersöz, G., ... & Dvorak, J., 2010. The effects of exercise, heat, cooling and rehydration strategies on cognitive function in football players. *Scandinavian journal of medicine & science in sports*, *20*(s3), 148-160.

Bartholomew, C.J., Jensen, W., Petros, T.V., Ferraro, F.R., Fire, K.M., Biberdorf, D., et al., 1999. The effect of moderate levels of simulated altitude on sustained cognitive performance. International Journal of Aviation Psychology 9(4), 351−359.

Baumeister, R.F., Heatherton, T.F., 1996. Self-regulation failure: An overview. Psychol Inq 7, 1–15.

Baumeister, R.F., Bratslavsky, E., Muraven, M., Tice, D.M., 1998. Ego depletion: Is the active self a limited resource? J Pers Soc Psychol 74, 1252–1265.

Berse, T., Rolfes, K., Barenberg, J., Dutke, S., Kuhlenbäumer, G., Völker, K., ... & Knecht, S. (2015). Acute physical exercise improves shifting in adolescents at school: evidence for a dopaminergic contribution. *Frontiers in behavioral neuroscience*, *9*.

Boksem, M. A., & Tops, M., 2008. Mental fatigue: costs and benefits. Brain research reviews, 59(1), 125-139.

Bonaz, B., 2003. Visceral sensitivity perturbation integration in the brain–gut axis in functional digestive disorders. J Physiol Pharmacol 54(4), 27–42.

Borg G. *Borg's perceived exertion and pain scales*. Champaign: Human Kinetics; 1998.

Botvinick, M. M., Cohen, J. D., & Carter, C. S., 2004. Conflict monitoring and anterior cingulate cortex: an update. *Trends in cognitive sciences*, *8*(12), 539-546.

Brands, A.M., Biessels, G.J., de Haan, E.H., Kappelle, L.J., Kessels, R.P., 2005. The effects of type 1 diabetes on cognitive performance: a meta-analysis. Diabetes Care 28, 726–735.

Bray, S.R., Graham, J.D., Martin Ginis, K.A., Hicks, A.L., 2012. Cognitive task performance causes impaired maximum force production in human hand flexor muscles. Biol Psychol 89(1), 195-200.

Bray, S.R., Graham, J.D., Saville, P.D., 2015. Self-control training leads to enhanced cardiovascular exercise performance. J Sports Sci 33(5), 534-543.

Brisswalter, J., Collardeau, M., Arcellin, R., 2002. Effects of acute physical exercise characteristics on cognitive performance. Sports Med 32, 555–566.

Buhle, J.T., Silvers, J.A., Wager, T.D., Lopez, R., Onyemekwu, C., Kober, H., Weber, J., Ochsner, K.N., 2013. Cognitive reappraisal of emotion: a meta-analysis of human neuroimaging studies. Cerebral Cortex 24(11), 2981–2990.

Bullock, T., Giesbrecht, B., 2014. Acute exercise and aerobic fitness influence selective attention during visual search. Front Psychol. http://dx.doi.org/10.3389/fpsyg.2014.01290

Carter, C.S., Braver, T.S., Barch, D.M., Botvinick, M.M., Noll, D., Cohen, J.D., 1998. Anterior cingulate cortex, error detection, and the online monitoring of performance. Science 280, 747–749.

Chang, Y.K., Labban, J.D., Gapin, J.I., Etnier, J.L., 2012. The effect of acute exercise on cognitive performance : A meta-analytic review. Brain Res 1453, 87–101.

Chmura, J., Nazar, K., Kaciuba-Uściłko, H., 1994. Choice reaction time during graded exercise in relation to blood lactate and plasma catecholamine thresholds. Int J Sports Med 15(4), 172–176.

Chmura, J., Krysztofiak, H., Ziemba, A.W., Nazar, K., Kacuba-Uscilko, H., 1998. Psychomotor performance above and below the blood lactate threshold. Eur J Appl Physiol 77, 77–80.

Chmura, J., Nazar, K., 2010. Parallel changes in the onset of blood lactate accumulation (OBLA) and threshold of psychomotor performance deterioration during incremental exercise after training in athletes. Int J Psychophysiol 75, 287–90.

Cian C, Barraud PA, Melin B, Raphel C. Effects of fluid ingestion on cognitive function after heat stress or exercise-induced dehydration. Int J Psychophysiol. 2001, 42(3): 243-51.

Cohen, N., Marguliers, D.S., Ashkenazi, C., Schaefer, A., Taubert, M., Henick, A., Villringer, A., Okon-Singer, H., 2015. Using executive control training to suppress amygdala reactivity to aversive information. Neuroimage, doi:10.1016/j.neuroimage.2015.10.069.

Davranche, K., McMorris, T., 2009. Specific effects of acute moderate exercise on cognitive control. Brain Cogn 69(3), 565–570.

Dehaene, S., Naccache, L., 2001. Towards a cognitive science of consciousness: basic evidence and a workspace framework. Cognition 79, 1–37.

Del Giorno, J.M., Hall, E.E., O'Leary, K.C., Bixby, W.R., Miller, P.C., 2010. Cognitive function during acute exercise: a test of the transient hypofrontality theory. J Sport Exer Psychol 32, 312–323.

Morree, H. M., Klein, C., & Marcora, S. M., 2012. Perception of effort reflects central motor command during movement execution. Psychophysiology, *49*(9), 1242-1253.

Dettmers, C., Fink, G.R., Lemon, R.N., Stephan, K.M., Passingham, R.E., Silbersweig, D., Holmes, A., Ridding, M.C., Brooks, D.J., Frackowiak, R.S., 1995. Relation between cerebral activity and force in the motor areas of the human brain. J Neurophysiol 74, 802–815.

Dettmers, C., Ridding, M.C., Stephan, K.M., Lemon, R.N., Rothwell, J.C., Frackowiak, R.S., 1996. Comparison of regional cerebral blood flow with transcranial magnetic stimulation at different forces. J Appl Physiol 81, 596–603.

Deutsch, R., Strack, R., 2006. Duality Models in Social Psychology: From Dual Processes to Interacting Systems. Psychol Inquiry 17(3), 166–172.

Dietrich, A., 2003. Functional neuroanatomy of altered states of consciousness: the transient hypofrontality hypothesis. Consciousness and cognition 12, 231–256.

Dietrich, A., Sparling, P.B., 2004. Endurance exercise selectively impairs prefrontal-dependent cognition. Brain Cogn 55, 516–524.

Dietrich, A., Audiffren, M., 2011. The reticular-activating hypofrontality (RAH) model of acute exercise. Neurosci Biobehav Rev 35, 1305–1325.

Eccleston, C., Crombez, G., 1999. Pain demands attention: a cognitive-affective model of the interruptive function of pain. Psychol Bull 125, 356–366.

Edwards, A.M., Polman, R.C., 2013. Pacing and awareness: brain regulation of physical activity. Sports Med 43, 1057–1064.

Egan, G., Silk, T., Zamarripa, F., et al., 2003. Neural correlates of the emergence of consciousness of thirst. Proc. Natl Acad. Sci. USA 100, 15241–15246.

Englert, C., & Bertrams, A., 2012. Anxiety, ego depletion, and sports performance. *Journal of Sport and Exercise Psychology*, *34*(5), 580.

Eriksen, B.A., Eriksen, C.W., 1974. Effects of noise letters upon the identification of a target letter in a non search task. *Perception & Psychophysics*, 16, 143–149.

Etnier, J.L., 2015. Brain-derived neurotrophic factor (BDNF) as a potential mechanism of the effects of acute exercise on cognitive performance. J Sport Health Sci 4(1), 14–23.

Evans J.St.B.T., 2008. Dual-Processing accounts of reasoning, judgment, and social cognition. Annu Rev Psychol 59, 255-278

Evans, D.R., Boggero, I.A., Segerstrom, S.C., 2015. The nature of self-regulatory fatigue and « ego depletion »: lessons from physical fatigue. Personality and Social Psychology Review, 1088868315597841.

Everitt, B.J., Robbins, T.W., 1997. Central cholinergic systems and cognition. Annual review of Psychology 48, 649-684.

Forstmann, B.U., van den Wildenberg, W.P.M., Ridderinkhof, K.R., 2008. Neural mechanisms, temporal dynamics, and individual differences interference control. Journal of Cognitive Neuroscience 20, 1854–1865.

Fox, M. D., Snyder, A. Z., Vincent, J. L., Corbetta, M., Van Essen, D. C., & Raichle, M. E., 2005. The human brain is intrinsically organized into dynamic, anticorrelated functional networks. *Proceedings of the National Academy of Sciences of the United States of America*, *102*(27), 9673-9678.

Fuster, J.M., 1997. Network memory. Trends in Neurosciences 20, 451–459.

Gailliot, M.T., Plant, E.A., Butz, D.A., Baumeister, R.F., 2007. Increasing self-regulatory strength can reduce the depleting effect of suppressing stereotypes. Pers Soc Psychol Bull 33, 281–294.

Gandevia, S.C., 2001. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev 81, 1725–1789.

Gaoua, N., Racinais, S., Grantham, J., El Massioui, F., 2011. Alterations in cognitive performance during passive hyperthermia are task-dependent. Int J Hypertherm 27(1), 1–9.

Gaoua, N., Grantham, J., Racinais, S., El Massioui, F., 2012. Sensory displeasure reduces complex cognitive performance in the heat. Journal of environmental psychology 32(2), 158–163.

Grego, F., Vallier, J-M., Collardeau, M., Bermon, S., Ferrari, P., Candito, M., Bayer, P., Magnié, M-N., Brisswalter, J., 2004. Effects of long duration exercise on cognitive function, blood glucose, and counter regulatory hormones in male cyclists. Neuroscience Letters 364, 76–80.

Grego, F., Vallier, J. M., Collardeau, M., Rousseu, C., Cremieux, J., & Brisswalter, J., 2005. Influence of exercise duration and hydration status on cognitive function during prolonged cycling exercise. *International journal of sports medicine*, *26*(1), 27-33

Grillon, C., Quispe-Escudero, D., Mathur, A., & Ernst, M., 2015. Mental fatigue impairs emotion regulation. *Emotion*, *15*(3), 383.

Henderson, L.A., Gandevia, S.C., Macefield, V.G., 2007. Somatotopic organization of the processing of muscle and cutaneous pain in the left and right insula cortex: a single-trial fMRI study. Pain 128, 20–30.

Hilty, L., Jäncke, L., Luechinger, R., Boutellier, U., & Lutz, K., 2011. Limitation of physical performance in a muscle fatiguing handgrip exercise is mediated by thalamo insular activity. *Human brain mapping*, *32*(12), 2151-2160.

Hockey, G.R.J., 1997. Compensatory control in the regulation of human performance under stress and high workload: a cognitive-energetical framework. Biol Psychol 45, 73–93.

Hocking C, Silberstein RB, Lau WM, Stough C, Roberts W., 2001. Evaluation of cognitive performance in the heat by functional brain imaging and psychometric testing. Comp Biochem Physiol: 128: 719-734.

Houben, K., & Jansen, A., 2011. Training inhibitory control. A recipe for resisting sweet temptations. *Appetite*, *56*(2), 345-349.

Houben, K., Wiers, R. W., & Jansen, A., 2011. Getting a grip on drinking behavior training working memory to reduce alcohol abuse. *Psychological Science*.

Huang, H.J., Mercer, V.S., 2001. Dual-task methodology: applications in studies of cognitive and motor performance in adults and children. Pediatr Phys Ther 13:133e40.

Humphreys, M.S., Revelle, W., 1984. Personality, motivation, and performance : A theory of the relationship between individual differences and information processing. Psychological Review 91, 153–184.

Jacobson, J., & Matthaeus, L., 2014. Athletics and executive functioning: How athletic participation and sport type correlate with cognitive performance. *Psychology of Sport and Exercise*, *15*(5), 521-527.

Jenkins, I.H., Brooks, D.J., Nixon, P.D., Frackowiak, R.S.J., Passingham, R.E.., 1994. Motor sequence learning: a study with positron emission tomography. Journal of Neuroscience 14, 3775–3790.

Jouanin, J. C., Pérès, M., Ducorps, A., & Renault, B., 2009. A dynamic network involving M1−S1, SII−insular, medial insular, and cingulate cortices controls muscular activity during an isometric contraction reaction time task. *Human brain mapping*, *30*(2), 675-688.

Joyner, M. J., & Coyle, E. F. (2008). Endurance exercise performance: the physiology of champions. *J Physiol*, *586*, 35-44.

Kahneman, D., 1973. Attention and Effort. Prentice–Hall, EnglewoodCliffs, NJ.

Kelso, J.A.S., 1995. Dynamic patterns: The self-organization of brain and behavior. Cambridge, MA: MIT Press.

Kempainnen, J., Aalto, S., Fujimoto, T., Kalliokoski, K.K., Långsjö, J., Oikonen, V., Rinne, J., Nuutila, P., Knuuti, J., 2005. High intensity exercise decreases global brain glucose uptake in humans. J Physiol 568(1), 323–332.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S., 2004. Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*(5660), 1023-1026.

Kim, S., Lee, D., 2010. Prefrontal cortex and impulsive decision making. Biol Psychiatry. doi:10.1016/j.biopsych.2010.07.005.

Labelle, V., Bosquet, L., Mekary, S., Bherer, L., 2012. Decline in executive control during acute bouts of exercise as a function of exercise intensity and fitness level. Brain Cogn 81, 10–17.

Lambourne, K., Audiffren, M., Tomporowski, P.D., 2010. Effects of acute exercise on sensory and executive processing tasks. Med Sci Sport Exer 47, 1396–1402.

Lambourne, K., Tomporowski, P., 2010. The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. Brain Research 1341, 12-24.

Leh, S. E., Petrides, M., & Strafella, A. P., 2010. The neural circuitry of executive functions in healthy subjects and Parkinson's disease. *Neuropsychopharmacology*, *35*(1), 70-85.

Lieberman, S., 2007. Dehydration and cognitive performance. J Am College Nutrition 26(5), 5495–5545.

Lorist, M.M., Kernell, D., Meijman, T.F., Zijdewind, I., 2002. Motor fatigue and cognitive task performance in humans. J Physiol 545(1), 313–319.

Lorist, M.M., Boksem, M.A.S., Ridderinkhof, K.R., 2005. Impaired cognitive control and reduced cingulate activity during mental fatigue. Cognitive Brain Research 24, 199 –205.

Lucas, S.J., Ainslie, P.N., Murrell, C.J., Thomas, K.N., Franz, E.A., Cotter, J.D., 2012. Effect of age on exercise-induced alterations in cognitive executive function: relationship to cerebral perfusion. Exp Gerontol 47, 541–551.

Marcora, S.M., 2008. Do we really need a central governor to explain bran regulation of exercise performance? Eur J Appl Physiol 104, 929–931.

Marcora, S.M., 2009. Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart, and lungs. J Appl Physiol 106, 2060–2062.

Marcora, S.M., Staiano, W., 2010. The limit to exercise tolerance in humans: mind over muscle? Eur J Appl Physiol 109, 763–770.

McCarron, J., Hodgson, T.L., Smith, M.F., 2013. Brain drain: evaluating the impact of increased cognitive load during self-paced running performance. Br J Sports Med. doi:10.1136/bjsports-2013-093073.43.

McEwan, D., Martin Ginis, K. A., & Bray, S. R., 2013. The effects of depleted self-control strength on skill-based task performance. *Journal of sport and exercise psychology*, *35*, 239-249.

McMorris, T., Collard, K., Corbett, J., Dicks, M., Swain, J.P., 2008. A test of the catecholamines hypothesis for an acute exercise–cognition interaction. Pharmacol Biochem Behav 89, 106–115.

McMorris, T., Davranche, K., Jones, G., Hall, B., Corbett, J., Minter, C., 2009. Acute incremental exercise, performance of a central executive task, and sympathoadrenal system and hypothalamic-pituitary-adrenal axis activity. International Journal of Psychophysiology 73, 334–340.

McMorris, T., & Graydon, J., 2000. The effect of incremental exercise on cognitive performance. *International Journal of Sport Psychology*, *31*(1), 66-81.

McMorris, T., Tomporowski, P.D., Audiffren, M., 2009. (Eds) Exercise and cognitive function. Wiley, New-York.

McMorris, T., Sproule, J., Turner, A., Hale, B.J., 2011. Acute, intermediate intensity exercise, and speed and accuracy in working memory tasks: a meta-analytical comparison of effects. Physiol & Behav 102, 421–28.

McMorris, T., Hale, B.J., 2012. Differential effects of differing intensities of acute exercise on speed and accuracy of cognition: a meta-analytical investigation. Brain Cogn 80, 338–351.

McMorris, T., Turner, A., Hale, B. J., & Sproule, J., 2016. Beyond the catecholamines hypothesis for an acute exercise-cognition interaction: a neurochemical perspective. *Exercise-Cognition Interaction: Neuroscience Perspectives, edited by McMorris T. New York: Academic*, 65-104.

Medford, N., Critchley, H.D., 2010. Conjoint activity of anterior insular and anterior cingulate cortex: awareness and response. Brain Struct Funct 214, 535–549.

Meeusen, R., Pires, F. O., Lutz, K., Cheung, S. S., Perrey, S., Rauch, H. L., ... & Beedie, C., 2016. Commentaries on Viewpoint: A role for the prefrontal cortex in exercise tolerance and termination. *Journal of Applied Physiology*, *120*(4), 467-469.

Meeusen, R., Watson, P., Hasegawa, H., Roelands, B., Piacentini, M.F., 2006. Central fatigue: Teh serotonin hypothesis and beyond. Sports Med 36(10), 881–909.

Meyniel, F., Safra, L., Pessiglione, M., 2014. How the Brain Decides When to Work and When to Rest: Dissociation of Implicit-Reactive from Explicit-Predictive Computational Processes. PLoS Comput Biol. doi: 10:e1003584. 10.1371/journal.pcbi.1003584

Meyniel, F., Sergent, C., Rigoux, L., Daunizeau, J., Pessiglione, M., 2013. Neurocomputational account of how the human brain decides when to have a break. Proc Natl Acad Sci USA 110, 2641–2646.

Millet, G.Y., 2011. Can neuromuscular fatigue explain running strategies and performance in ultra-marathons?: the flush model. Sports Med 41, 489–506.

Miller, E.K., Wallis, J.D., 2009. Executive Function and Higher-Order Cognition: Definition and Neural Substrates, In: Squire LR. Encyclopedia of neurosciences. Volume 4. Oxfort Academic Press, pp. 99–104.

Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., 2000. The unity and diversity of executive functions and their contributions to complex frontal lobe tasks: A latent variable analysis. Cog Psychol 41, 49–100.

Morrison, A. B., & Chein, J. M., 2011. Does working memory training work? The promise and challenges of enhancing cognition by training working memory. *Psychonomic bulletin & review*, *18*(1), 46-60.

Muraven, M., Baumeister, R.F., 2000. Self regulation and depletion of limited resources: does self control resemble a muscle? Psychol Bull 126, 247–259.

Muzik, O., & Diwadkar, V. A., 2016. In vivo correlates of thermoregulatory defense in humans: Temporal course of sub-cortical and cortical responses assessed with fMRI. *Human brain mapping*.

Noakes, T.D., St Clair Gibson, A., Lambert, E.V., 2005. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. Br J Sports Med 39, 120–4.

Nowak, A., Vallacher, R.R., 1998. Dynamical Social Psychology. New York: Guilford.

Ochsner, K.N., Gross, J.J., 2005. The cognitive control of emotion. Trends Cogn Sci 9, 242–249.

Ochsner, K.N., Gross, J.J., 2008. Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. Current Directions in Psychological Science 17, 153–158.

Ogoh, S. Tsukamoto, H., Hirasawa, A., Hasegawa, H., Hirose, N., Hashimoto, T., 2014. The effect of changes in cerebral blood flow on cognitive function during exercise. Physiol Rep. doi:10.14814/phy2.12163.

Olson, R.L., Chang, Y.K., Brush, C.J., Kwok, A.N., Gordon, V.X., Alderman, B.L., 2015. Neurophysiological and behavioral correlates of cognitive control during and moderate exercise intensity. Neuroimage. http://dx.doi.org/10.1016/j.neuroimage.2015.10.011

Pageaux, B., 2014. The Psychobiological Model of Endurance Performance: An Effort-Based Decision-Making Theory to Explain Self-Paced Endurance Performance. Sports Med 44, 1319–1320.

Paus, T., 2001. Primate anterior cingulate cortex: where motor control, drive and cognition interface. Nature reviews 2, 414–424.

Pesce, C., 2009. An Integrated Approach to the Effect of Acute and Chronic Exercise on Cognition: The Linked Role of Individual and Task Constraints. In: McMorris, T., Tomporowski, P., Audiffren, M. Exercise and cognitive function. Wiley, New-York, pp. 213–226.

Pesce, C., 2012. Shifting the focus from quantitative to qualitative exercise characteristics in exercise and cognition research. J Sport Exer Psychol 34, 766–786.

Peyron, R., Laurent, B., Garcïa-Larrea, L., 2000. Functional imaging of brain responses to pain. A review and meta-analysis. Clin Neurophysiol 30, 263–288.

Piepmeier, A.T., Etnier, J.L., 2015. Brain-derived neurotrophic factor (BDNF) as a potential mechanism of the effects of acute exercise on cognitive performance. J Sport Health Sci 4(1), 14–23.

Poldrack, R. A., & Packard, M. G., 2003. Competition among multiple memory systems: converging evidence from animal and human brain studies. *Neuropsychologia*, *41*(3), 245-251.

Pontifex, M.B., Hillman, C.H., 2007. Neuroelectric and behavioral indices of interference control during acute exercise. Clin Neurophysiol. 118 570–580.

Qian, S., Li, M., Li, G., Liu, K., Li, B., Jiang, Q., Li, L., Yang, Z., Sun, G., 2014. Environmental heat stress enhances mental fatigue during sustained attention task performing: Evidence from an ASL perfusion study. Behav Brain Res. http://dx.doi.org/10.1016/j.bbr.2014.11.036

Racinais, S., Gaoua, N., Grantham, J., 2008. Hyperthermia impairs short-term memory and peripheral motor drive transmission. J Physiol 586, 4751–4762.

Ramos, B. P., & Arnsten, A. F., 2007. Adrenergic pharmacology and cognition: focus on the prefrontal cortex. *Pharmacology & therapeutics*, *113*(3), 523-536.

Rattray, B., Argus, C., Martin, K., Northey, J., & Driller, M., 2015. Is it time to turn our attention toward central mechanisms for post-exertional recovery strategies and performance?. *Frontiers in physiology*, *6*.

Robbins, T. W., & Everitt, B. J., 2007. A role for mesencephalic dopamine in activation: commentary on Berridge (2006). *Psychopharmacology*, *191*(3), 433-437.

Romeas, T., Guldner, A., & Faubert, J., 2016. 3D-Multiple Object Tracking training task improves passing decision-making accuracy in soccer players. *Psychology of Sport and Exercise*, *22*, 1-9.

Rooks, C.R., Nathaniel, J.T., McCully, K.K., Dishman, R.K., 2010. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy : a systematic review. Progress in Neurobiology 92, 134–150.

Sanders, A.F., 1983. Towards a model of stress and human performance. Acta Psychologica 53, 61–97.

Schmidt, L., Cléry-Melin, M-L., Lafargue, G., Valabrègues, R., Fossati, P., Dubois, B., Pessiglione M., 2009. Get aroused and be stronger: emotional facilitation of physical effort in the human brain. J Neurosci 29(30), 9450–9457.

Schmidt, L., Lebreton, M., Cléry-Melin, M-L., Daunizeau, J., Pessiglione, M., 2012. Neural mechanisms underlying motivation of mental versus physical effort. Plos Biol 10(2), e1001266.

Schmit, C., Davranche, K., Easthope, C.S., Colson, S.S., Brisswalter, J., Radel, R., 2015. Pushing to the limits: the dynamics of cognitive control during exhausting exercise. Neuropsychologia 68, 71–81.

Shenhav, A., Botvinick, M.M., Cohen, J.D., 2013. The expected value of control: an integrative theory of anterior cingulated cortex function. Neuron 79(2), 21 –40.

Singh, A.M., Staines, R., 2015. The Effects of Acute Aerobic Exercise on the Primary Motor Cortex. J Motor Behav. doi: 10.1080/00222895.2014.983450

Smith, M. R., Marcora, S. M., & Coutts, A. J., 2014. Mental fatigue impairs intermittent running performance. *Med Sci Sports Exerc*.

Stewart, C.C., Wright, R.A., Hui, S-K.A., Simmons, A., 2009. Outcome expectancy as a moderator of mental fatigue influence on cardiovascular response. Psychophysiol 46, 1141–1149.

Sridharan, D., Levitin, D.J., Menon, V., 2008. A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. Proc Natl Acad Sci USA 105, 12569–12574.

Swart, J., Lindsay, T.R., Lambert, M.I., Brown, J.C., Noakes, T.D., 2012. Perceptual cues in the regulation of exercise performance-physical sensations of exercise and awareness of effort interact as separate cues. Br J Sports Med 46, 42–8.

Takarada, Y., Mima, T., Abe, M., Nakatsuka, M., Taira, M., 2014. Inhibition of the primary motor cortex can alter one's "sense of effort": Effects of low-frequency rTMS. Neurosci Research 89, 54–60.

Tanaka, M., & Watanabe, Y., 2012. Supraspinal regulation of physical fatigue. *Neuroscience & Biobehavioral Reviews*, *36*(1), 727-734.

Teh, M.M., Dunn, J.T., Choudhary, P., et al., 2010. Evolution and resolution of human brain perfusion responses to the stress of induced hypoglycemia. Neuroimage 53, 584–592.

Tenenbaum, G., Connolly, C.T., 2008. Attention allocation under varied workload and effort perception in rowers. Psychol Sport Exer 9(5), 704–717.

Thomas, M., Sing, H., Belenky, G., et al., 2000. Neural basis of alertness and cognitive performance impairments during sleepiness. Effects of 24 h of sleep deprivation on waking human regional brain activity. J Sleep Res 9, 335–352.

Tomporowski, P.D., 2003. Effects of acute bout of exercise on cognition. Acta Psychol 112, 297-324.

van Duinen, H., Renken, R., Maurits, N., Zijdewind, I., 2007. Effects of motor fatigue on human brain activity, an fMRI study. Neuroimage 35, 1438–1449.

Van Orden, G.C., Holden, J.G., Turvey, M.T., 2003. Self-organization of cognitive performance. Journal of Experimental Psychology: General 132, 331–350.

Vealey, R. S., 2007. Mental skills training in sport. *Handbook of Sport Psychology, Third Edition*, 285-309.

Veling, H., Aarts, H., & Papies, E. K., 2011. Using stop signals to inhibit chronic dieters' responses toward palatable foods. *Behaviour research and therapy*, *49*(11), 771-780.

Vestberg, T., Gustafson, R., Maurex, L., Ingvar, M., & Petrovic, P. (2012). Executive functions predict the success of top-soccer players. *PloS one*, *7*(4), e34731.

von Leupoldt, A., Sommer, T., Kegat, S., et al., 2008. The unpleasantness of perceived dyspnea is processed in the anterior insula and amygdala. Am J Respir Crit Care Med 177, 1026–32.

Walton, M.E., Bannerman, D.M., Alterescu, K. Rushworth, M.F., 2003. Functional specialization within medial frontal cortex of the anterior cingulate for evaluating effort-related decisions. J Neurosci 23, 6475–6479.

Walton, M.E., Kennerley, S.W., Bannerman, D.M., Phillips, P.E., Rushworth, M.F., 2006. Weighing up the benefits of work: behavioral and neural analyses of effort-related decision making. Neural Netw 19, 1302–1314.

Williamson, J.W., Fadel, P.J., Mitchell, J.H., 2006. New insights into central cardiovascular control during exercise in humans: a central command update. Exp Physiol 91, 51–58.

Wills, T.A., Stoolmiller, M., 2002. The role of self-control in early escalation of substance use: A time-varying analysis. J Cons Clin Psychol 70, 986–997.

Yerkes, R.M., Dodson, J.D., 1908. The relation of strength of stimulus to rapidity of habit-formation. J Comp Neurol Psychol 18, 459–482.

Zwierko, T., Florkiewicz, B., Fogtman, S., Kszak-krzyzanowska, A., 2014. The ability to maintain attention during visuomotor task performance in handball players and non-athletes. Central Eur J Sport Sci Med 7(3), 99–106.

Figure's caption

Fig. Neuro-functional (a) and dynamical (b) schematic representations of executive functioning during fatiguing exercise.

Notices. At the beginning of exercise, exercise-related arousal up-regulates PFC activity, improving executive functioning and self-regulatory performance. The parallel physical demand (e.g., pain, thirst) remains minimal as motor functions are fully efficient. As exercise duration increases, however, the physical demand progressively augments (e.g., inhibit muscle pain, monitor the body) and competes for PFC resources. As a consequence of the limited capacity of information processing, executive functioning get increasingly hindered. Exercise becomes 'fatiguing' when self-regulatory performance is back to square one. Counteracting the negative effect of fatiguing exercise on executive functioning requires factors arousing self-regulatory and/or physical performances (e.g., motivation), or alleviating body sensations (e.g., cooling, hydration).

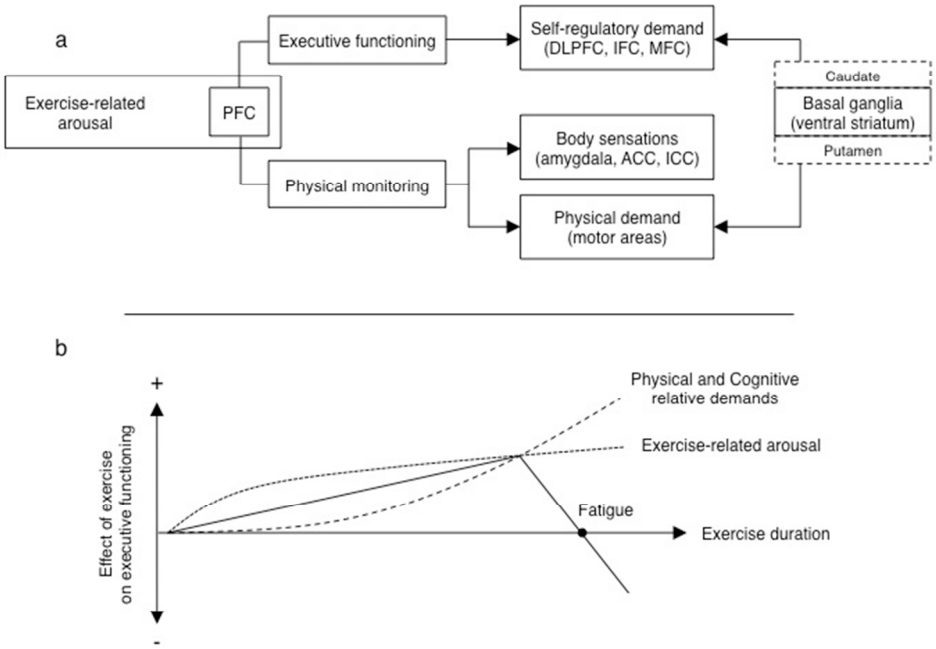


Fig. Neuro-functional (a) and dynamical (b) schematic representations of executive functioning during fatiguing exercise.
Notices. At the beginning of exercise, exercise-related arousal up-regulates PFC activity, improving executive functioning and self-regulatory performance. The parallel physical demand (e.g., pain, thirst) remains minimal as motor functions are fully efficient. As exercise duration increases, however, the physical demand progressively augments (e.g., inhibit muscle pain, monitor the body) and competes for PFC resources. As a consequence of the limited capacity of information processing, executive functioning get increasingly hindered. Exercise becomes 'fatiguing' when self-regulatory performance is back to square one. Counteracting the negative effect of fatiguing exercise on executive functioning requires factors arousing self-regulatory and/or physical performances (e.g., motivation), or alleviating body sensations (e.g., cooling, hydration).

254x190mm (72 x 72 DPI)

TRAVAIL DE THÈSE (en cours)

Schmit, C., Blain, B., Hausswirth, C., Pessiglione, M., & Le Meur, Y. Neural correlates of functional overreaching in endurance athletes.

Résumé en français

L'état de surmenage fonctionnel a été progressivement caractérisé au regard de ses implication physiologiques, mais ses soubassements cognitifs et neuronaux restent encore mal connus. Cette étude a eu pour objet de soumettre des athlètes entraînés et conduits en état de surmenage fonctionnel à un ensemble de tâches cognitives sous imagerie par résonance magnétique. Les résultats de cette investigation montrent une diminution de l'activité du cortex préfrontal latéral au cours de la réalisation de tâches de choix, associée à une augmentation du niveau d'impulsivité. Ces résultats suggèrent des implications dans l'évolution du comportement d'athlète soumis à des périodes d'entraînement intense, et donc la mise en place d'éléments de diagnostiques spécifiques permettant de protéger la performance de ces athlètes. Ce travail est en cours de finalisation.

TABLEAU DE SYNTHÈSE DES TRAVAUX DE THÈSE

Études	Participants	Test – Mesures principales	Résultats principaux	Éléments principaux de discussion
Pacing adjustments associated with familiarisation: Heat vs. temperate environments.	34 triathlètes – 2 groupes PMA ~380W $\dot{V}O_{2max}$ ~63ml.kg^{-1}.min^{-1}	2 CLM de 20km à 21°C ou à 35°C : Puissance moyenne, stratégie s'allure	Améliorations comparables de la puissance moyenne d'un CLM à l'autre entre les deux conditions. Réajustement de la stratégie d'allure en conditions chaudes.	La familiarisation à la chaleur entraîne une amélioration de la performance sous l'effet d'une redéfinition de la stratégie d'allure vers un pattern plus conservateur. Ce changement n'est pas observé en environnement tempéré.
Heat-acclimatization and pre-cooling: a further boost for endurance performance?	13 triathlètes – 1 groupe PMA ~406W $\dot{V}O_{2max}$ ~65ml.kg^{-1}.min^{-1}	4 CLM de 20km à 35°C : Températures centrale et cutanée, perte de poids de corps, fréquence cardiaque, volume plasmatique, RPE, confort thermique, charge d'entraînement	Les effets physiologiques bénéfiques du pré-cooling sur la performance sont atténues après une acclimatisation à la chaleur. Les effets perceptifs semblent, eux, persister.	Le pré-cooling post-acclimatation est faiblement ergogénique, et semble ainsi devoir être intensifié pour des athlètes déjà acclimatés à la chaleur.
Optimizing heat-acclimation for performance peaking: high- vs low-intensity training.	29 triathlètes – 3 groupes PMA ~370W $\dot{V}O_{2max}$ ~62ml.kg^{-1}.min^{-1}	3 CLM de 20km à 35°C : Températures centrale et cutanée, fréquence cardiaque, RPE, confort thermique, perte de poids de corps, charge d'entraînement, performance cognitive	Dégradation du niveau de performance des athlètes ayant réalisé une acclimatation à la chaleur comprenant strictement des entraînement à haute intensité. Baisse non surcompensée après la période d'affûtage.	Si des entraînements à haute intensité peuvent être programmés lors d'un stage d'acclimatation à la chaleur, ceux-ci ne devraient intervenir qu'après une série d'entraînements à basse intensité sous peine de potentialiser l'instauration précoce d'un état de surmenage.
Pushing to the limits: the dynamics of cognitive control during exhausting exercise.	15 individus – 1 groupe PMA ~260W $\dot{V}O_{2max}$ ~45ml.kg^{-1}.min^{-1}	2 temps-limite à 85% PMA : Performance cognitive, RPE, oxygénation cérébrale (NIRS), activation musculaire (EMG), fréquence cardiaque	Amélioration de la performance cognitive en première partie d'effort, puis dégradation de cette performance avant l'épuisement. Absence d'hypofrontalité à l'exercice.	Le déclin de performance cognitive à l'exercice fatiguant est à rapprocher d'une augmentation de l'impulsivité et non d'une dégradation du fonctionnement exécutif. Ce déclin n'est pas associé à un phénomène d'hypofrontalité.
Executive functioning during prolonged exercise: A fatigue based-neurocognitive perspective.	Un rationnel neuronal basé sur le fonctionnement des afférences et efférences pourrait expliquer les variations de performance cognitives à l'exercice.			
Cognitive functioning and heat strain: performance responses and protective strategies.	Une relation en U-inversé est suggérée entre l'état d'hyperthermie et la performance cognitive. Cela suggère que les stratégies de refroidissement pourraient être utiles essentiellement lorsque la température interne dépasse ~38.5°C.			

DISCUSSION GÉNÉRALE

Cette thèse avait pour ambition d'appréhender le phénomène de fatigue à l'exercice physique relativement à ses effets sur la performance, ses dynamiques et ses mécanismes. Pour cela, nous avons croisé les composantes physiologiques et psychologiques de la performance en endurance à des approches à moyen- et court-terme de la compétition sportive. Par ailleurs, nous avons aussi confronté l'ensemble de ces paramètres à des ambiances thermiques différentes, eu égard à l'éventualité d'environnements chauds lors des Jeux Olympiques de Rio 2016.

Ensemble, ces travaux ont abouti à la rédaction d'articles scientifiques qui, dans leur essence, peuvent être rapprochés du fonctionnement du Flush Model de Millet (2011). Initialement, ce modèle propose une régulation de la performance en endurance à partir du ressenti – modulable – de pénibilité à l'exercice de l'athlète. En introduction à nos travaux, nous dégagions du Flush Model deux écueils : un premier renvoyant à la réponse de l'athlète à une approche chronique de la performance ; un second ayant trait à la réponse cognitive de l'athlète à l'exercice. Nos travaux sont discutés ci-dessous en regard de ces points pour possiblement envisager un complément au modèle de Millet (2011).

1. Le Flush Model à l'épreuve des charges d'entraînement

L'état de fatigue accompagnant la planification de lourdes périodes d'entraînement en phase précompétitive peut dépasser la capacité de récupération de l'athlète. Ainsi, quatre des travaux de cette thèse ont permis d'affiner la compréhension de la réponse d'athlètes entraînés aux variations de leurs charges d'entraînement (Schmit et *al*., 2016a ; Le Meur et *al*.,2016 ; Schmit et al., 2015c, 2016b). De façon notable, bien que ces travaux aient été conduits dans des environnements thermiques distincts (*i.e.,* tempérés ou chauds), un message consistant s'en dégage. Spécifiquement, une charge de travail optimale apparaît requise afin d'induire une élévation du potentiel de performance de l'athlète, au-delà de laquelle ce potentiel diminue en raison de l'instauration d'un état de surmenage. En d'autres termes, que l'origine de la surcharge provienne d'une augmentation du volume d'entraînement ou d'une manipulation de la contrainte thermique environnementale, le

déséquilibre de la balance « charge / récupération » de l'athlète reste un phénomène commun à prévenir.

La différence en termes d'évolution de performance entre une augmentation optimale ou, au contraire, délétère de la charge d'entraînement est en effet importante. En particulier, lors d'épreuves de contre-la-montre de 20 km à vélo en chaleur (~35°C, 50% RH), nous avons constaté un gain de performance de 2,1 ± 1,2% après cinq jours d'acclimatation à la chaleur (Schmit et *al*., 2016b) et de 3,1 ± 1,7% et 4,2 ± 1,8% après huit et onze jours d'acclimatisation, respectivement (Schmit et *al*., 2015c ; Le Meur et *al*., 2016). En contraste, lorsque le protocole d'acclimatation n'était pas toléré par les athlètes nous relevions une baisse de performance de 1,7 ± 1,3% (Schmit et *al*., 2016b), voire l'abandon de l'étude par certains participants (Le Meur et *al*., 2016). Ce déclin de performance se révélait par ailleurs comparable à celui constaté en ambiance tempérée lorsqu'une surcharge d'entraînement programmée sur trois semaines résultait en une baisse de 1,9 ± 4,1% de la performance (Schmit et *al*., 2016a). De façon importante, consécutivement à une période d'affûtage, chacune de ces performances se trouvait améliorée, soit en étant encore augmentée (dans le cas d'une tolérance initiale à la surcharge), soit en étant faiblement surcompensée (dans le cas de l'instauration d'un état de surmenage). Ensemble, ces dynamiques respectives de performance en réponse aux variations de charge d'entraînement rappellent donc celles distinguant des athlètes en état de « fatigue aigue » *vs*. de « surmenage fonctionnel » (Aubry et al., 2014 ; Meeusen et al., 2013). Dans ce contexte, les résultats de ces études renforcent i) l'absence d'intérêt d'une période de surcharge conduisant à une baisse du niveau d'efficacité de l'athlète, et ii) l'existence, chez celui-ci, d'un seuil de tolérance discriminant le caractère bénéfique *vs*. nuisible de la surcharge. En effet, au-delà des variations de performance en cohérence avec la littérature sur l'étude du surmenage, plusieurs indices relatifs au dépassement de cette limite ont été recueillis.

Un nombre croissant de travaux s'accorde sur la pertinence d'outils simples du diagnostique de l'état de surmenage *e.g.,* baisses de la fréquence cardiaque (FC), de la lactatémie et des performances cognitives à l'exercice (Decroix et al., 2016 ; Dupuy et *al*., 2014 ; Le Meur et *al*., 2013). Les travaux de cette thèse confirment ces observations, les étendent au contexte spécifique de l'état de surmenage induit par l'exercice en environnement chaud, et agrémentent ces mécanismes de la présence d'une inhibition

corticale chez des sujets surmenés. Plus précisément, la moindre activation d'une partie du PFC (*i.e.,* le gyrus frontal moyen, MFG ; ci-contre) lors de la réalisation de tâches cognitives a permis de caractériser un phénomène de fatigue neurale ; celui-ci s'exprimait au sein d'une plus grande propension des sujets surmenés à privilégier des récompenses immédiates lors de tâches économiques *i.e.,* à engager des choix impulsifs (Schmit et *al*., 2016a). De façon notable, ces observations étaient effectuées spécifiquement à un état de surmenage induit par trois semaines de surcharge d'entraînement en environnement tempéré ; leur extension à une population d'athlètes surmenés à partir d'une période d'acclimatation à la chaleur reste donc à investiguer. Pour autant, le déclin des performances cognitives observé chez les athlètes surmenés par des entraînements en conditions chaudes invite à une réflexion similaire *i.e.,* l'influence d'une fatigue neurale sur la capacité d'attention soutenue (Schmit et *al*., 2016b). Quoi qu'il en soit,

dans une perspective d'aide au suivi de l'état de forme de l'athlète, ces données neurales et comportementales soutiennent l'intérêt de tests cognitifs utilisés en routine et, possiblement, évaluant la capacité d'inhibition de l'individu. Ce type de tâche est en effet spécifique à requérir l'implémentation du PFC (dont le MFG) pour la retenue d'impulses spontanés (*e.g.,* une décentration de la tâche, une activation motrice réflexe) et pourrait ainsi être particulièrement sensible à une intolérance de l'athlète à la surcharge d'entraînement (Aron et *al*., 2004 ; Schmit et *al*., 2015a ; Swick et *al*., 2008).

Diagnostiquer le développement d'un état de surmenage semble indissociable de ce type de mesures (*i.e.,* les variables d'ordre psychologique). En effet, les baisses de FC et de lactatémie à l'exercice évoquées plus haut demeurent certes des symptômes de surmenage, mais sont aussi des observations en cohérence avec une amélioration de la condition physique. Ces mesures peuvent donc, lorsqu'utilisées seules, inviter à un positionnement optimiste sur l'état de forme de l'athlète mais, en réalité, masquer une mauvaise adaptation au stimulus d''entraînement. En période d'acclimatation à la chaleur, ce type d'interprétation peut encore plus facilement être supposé, en raison des variations de FC liées à la fois aux

incidences neurales de la surcharge *et* au phénotype d'acclimatation à la chaleur (*i.e.,* ses caractéristiques cardiovasculaires, thermorégulatoires et métaboliques) (Schmit et *al*., 2016b). Dans ce contexte, sans prise en considération de variables de charge d'entraînement autres que celles énoncées d'ordre physiologique, la gestion de l'état de forme de l'athlète en phase précompétitive semble délicate. Cette idée est par ailleurs accentuée par les doutes émis relativement à l'augmentation du volume plasmatique post-acclimatation en tant que fondement de l'amélioration des performances en endurance (Le Meur et *al*., 2016).

Avec ceci en tête, la méthode de quantification de la charge d'entrainement de Foster (RPE*durée d'entraînement ; 2001) pourrait constituer un complément fiable d'ajustement du travail de l'athlète (indépendamment de la nature de la surcharge appliquée à l'organisme) dans la mesure où elle inclut *et* dépasse la prise en compte des marqueurs physiologiques énoncés ci-dessus. Par exemple, nous relevions une large différence de charge d'entraînement entre les athlètes surmenés par une augmentation de leur volume d'entraînement (+65%) et les athlètes du groupe contrôle (Schmit et *al*., 2016a) ; malheureusement, un tel score suppose aussi un large éventail au sein duquel pourrait se développer le phénomène de surmenage. Aussi, de façon plus précise, nous reportions une hausse de ~7% de la charge lorsque les séances d'entraînement effectuées en chaleur comprenaient de basses intensités d'exercice (et conduisaient à un gain de performance), contre une hausse 17% de la charge pour des séances menées exclusivement à haute intensité (et entraînant une baisse du niveau de performance) (Schmit et *al*., 2015c, 2016b). Ensemble, ces différences de magnitude de la charge d'entraînement suggèrent une réponse de l'athlète hautement dépendante de la nature du stress manipulé pendant la surcharge (*e.g.,* durée, intensité, environnement). Autrement dit, le seuil de variation de la charge d'entraînement de l'athlète à partir duquel pourrait s'observer un déclin de performance lors d'une acclimatation à la chaleur pourrait être spécifique et s'avérer indépendant de celui initiant une contre-performance après une manipulation du volume d'entraînement uniquement. Cette relation de dose-réponse reste largement à investiguer pour optimiser les charges d'entraînement précompétitives à la nature du stress manipulé. Néanmoins, elle demeure consistante dans l'approche systémique de l'individu qu'elle sous-tend et peut, à cet égard, être rapprochée d'un fonctionnement à moyen-terme du Flush Model.

L'essence du Flush Model repose sur sa disposition à cristalliser la performance en endurance autour d'un construit relatif à une large gamme de paramètres d'ordre physiologique comme psychologique : le RPE. Indépendamment de la nature de la surcharge qu'ils impliquent, les travaux présentés plus haut dénotent une évolution contrastée du rapport 'RPE/intensité d'exercice consentie' en fin de surcharge. Pour des scores RPE similaires, celle-ci pouvait en effet distinctement résulter en une élévation ou, à l'inverse, une réduction de la puissance de pédalage lors d'épreuves de contre-la-montre à vélo. Dans le premier cas (« fatigue aigüe »), l'équilibre de la balance charge/récupération de l'athlète était effectivement respecté et, pour un même ressenti, autorisait une stratégie d'allure plus agressive sur tout ou partie de la course (Schmit et *al*., 2016a ; Schmit et *al*., 2015c, 2016b, données non publiées). Relativement au Flush Model, ces observations supposent pour une intensité d'exercice donnée un remplissage plus lent de la cuvette (dans le cas d'une stratégie d'allure croissante) et/ou un niveau d'eau initial inférieur après la surcharge (dans le cas d'une stratégie d'allure plus agressive dès le départ) (Figure 5). Ces évolutions théoriques pourraient refléter le développement à moyen-terme des fonctionnements cardiovasculaire, thermorégulatoire, perceptif et/ou cognitif plus efficients. En contraste, lorsque la surcharge n'était pas tolérée par l'athlète (« surmenage fonctionnel »), le même score RPE se traduisait spécifiquement par des allures de course plus faibles (Schmit et *al*., 2016a,b). Comme évoqué, ce comportement était associé à des altérations physiologiques, cognitives et perceptives chez l'athlète à l'exercice. En termes de modélisation, ces altérations pourraient être interprétées de la manière suivante : la programmation d'une charge d'entraînement trop importante pour l'athlète (c'est-à-dire supérieure à sa capacité de récupération) pourrait entrainer l'instauration latente d'un niveau d'eau plus élevé dans la cuvette, et finalement une baisse du niveau de performance (Figure 5).

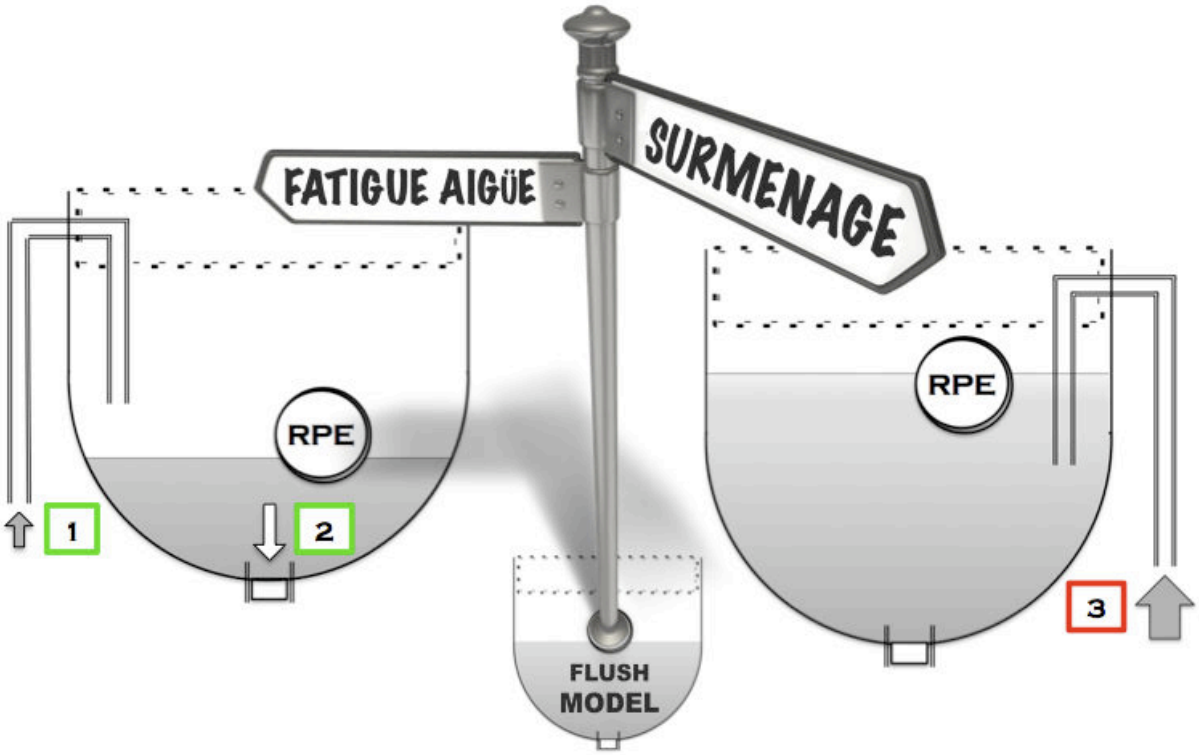


Fig. 5. Illustrations des modalités de réponse à la surcharge d'entraînement à partir des principes de fonctionnement du Flush Model (Millet, 2011).
Notes. RPE = '*rating of perceived* exertion' ; 1 représente un débit ralenti de remplissage de la cuvette ; 2 représente la vidange du niveau d'eu initial de la cuvette ; 3 représente un niveau d'eau initial augmenté.

En somme, ces points présentent et questionnent la limite fine et les implications mécanistiques d'une adaptation délétère à une surcharge d'entraînement. Ces implications restent à approfondir pour identifier de possibles congruences entre les différentes natures de surcharge ; néanmoins, des observations comportementales communes s'en dégagent. Dans ce cadre, des outils simples de suivi de la charge de l'athlète combinant les caractéristiques de l'entraînement (durée, intensité, mode) à la réponse de l'athlète à celui-ci (*e.g.,* échelles perceptives, FC, tâches cognitives) peuvent faciliter la prévention du développement d'un état de surmenage en phase précompétitive, et maximiser les chances de survenue d'un pic de performance (Drew & Finch, 2016). En phase compétitive, cette approche psycho-physiologique propre au modèle de Millet (2011) est aussi à envisager, bien qu'à compléter.

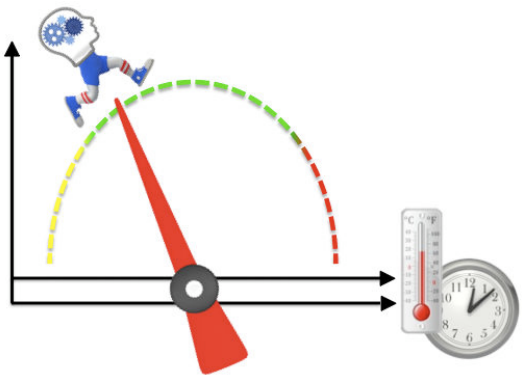
2. Le Flush Model : Implications cognitives d'une approche systémique

Comparativement aux approches linéaires de la fatigue, le Flush Model pose l'idée de la redéfinition par l'athlète de sa stratégie d'allure en cours d'épreuve. Millet (2011) fonde cette régulation sur la base de l'expérience perceptive de l'athlète au cours de l'épreuve ; ce point est rapidement évoqué ci-dessous relativement à nos travaux. Cependant, la réponse cognitive à l'exercice constitue aussi un facteur déterminant de la stratégie d'allure de l'athlète (McCarron et *al*., 2013), et est discuté plus loin en écho au Flush Model.

Deux de nos travaux ont conclu à des implications perceptives dans l'observation d'une amélioration du niveau d'efficacité de l'athlète lors d'épreuves de contre-la-montre de 20 km en chaleur (35°C, 50% RH). Dans le premier cas, le gain de performance (~2%) était constaté après une familiarisation initiale à cette épreuve spécifique en chaleur, et associé à une allure plus conservatrice en début de contre-la-montre (Schmit et *al*., 2015b). L'intervalle de 11 ± 4 jours entre les deux performances venait infirmer l'influence d'éventuelles adaptations physiologiques à la chaleur dans l'amélioration de la performance, et rapprochait alors le changement de stratégie d'allure d'un moyen pour l'athlète de ralentir le développement d'un état d'hyperthermie. A noter, un tel « transfert d'expérience » entre deux performances représente un outil ergogénique initialement non-développé par le Flush Model bien qu'au service de la régulation du ressenti de l'athlète (Noakes et *al*., 2005). Dans le second cas, le port d'un gilet réfrigérant CryoVest® en phase d'échauffement au 20 km autorisait un gain de performance (0,4 ± 1,1%) essentiellement manifesté en début d'épreuve avant de s'atténuer (Schmit et *al*., 2015c). De façon importante, cette observation était effectuée après huit jours d'acclimatisation à la chaleur. Spécifiquement, les bénéfices physiologiques induits par ce stage en chaleur étaient tels qu'ils 'shuntaient' ceux relatifs au port du gilet. Malgré cela, la perception d'un plus grand confort thermique restait évidente en condition de refroidissement, et était statistiquement associée à l'augmentation de l'allure de course. Ce constat se rapproche des observations sur lesquelles s'établit initialement le Flush Model *i.e.,* une altération du rapport 'ressenti/intensité d'exercice' *in situ* à la performance. Ensemble, ces travaux suggèrent l'atteinte plus tardive d'une pénibilité critique à l'exercice, par l'intermédiaire d'un remplissage plus lent de la cuvette (suite à la familiarisation) ou d'un niveau d'eau initial inférieur (suite au refroidissement par CryoVest®).

Alors que les fondements perceptifs de la sensation de difficulté à l'exercice sont largement documentés, les soubassements cognitifs de la performance en endurance restent pour leur part pauvres en investigations. Pourtant, l'idée d'une projection de la sensation d'effort sur la distance de course restante jusqu'à la ligne d'arrivée (et donc l'idée d'un calcul cognitif orienté sur le ressenti) est inhérente aux modèles de téléanticipation de la fatigue (Noakes et *al*., 2004 ; Tucker, 2009). Bien que s'appuyant sur ces modèles, Millet (2011) n'aborde toutefois pas cette composante de la performance *i.e.,* la capacité de l'athlète à être – et rester – lucide dans ses choix. Dans ce cadre, trois de nos travaux ont investigué la réponse du fonctionnement exécutif à l'exercice et à la chaleur.

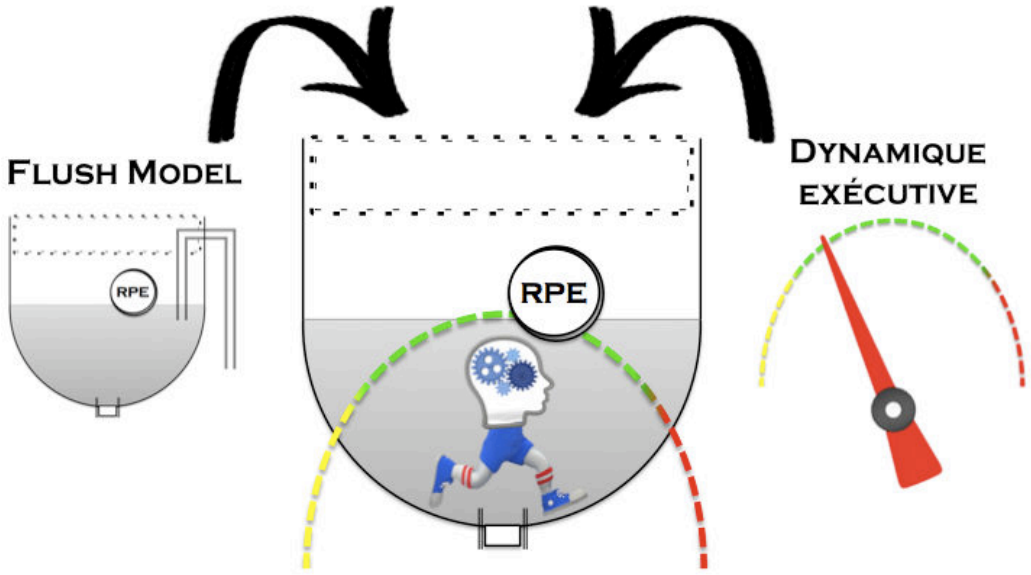
Deux revues et une étude expérimentale ont été conduites, dont le dénominateur commun est une représentation dynamique du fonctionnement exécutif en situation de stress. Plus précisément, afin de se rapprocher des conditions écologiques, les analyses des mesures cognitives effectuées ont été envisagées à l'épreuve du temps, et ce indépendamment du stresseur appréhendé (*i.e.,* l'exercice physique ou la chaleur endogène). De façon intéressante, les conclusions de ces travaux convergent. En particulier, elles raisonnent autour d'une réponse en U-inversé des performances exécutives à l'exercice physique et à l'état d'hyperthermie (Schmit et *al*., 2015a, 2016c ; Schmit & Brisswalter, 2016). Ainsi, les fonctions exécutives sont estimées gagner en vitesse et/ou précision à l'amorce de l'exercice ou lorsque la température interne s'élève, avant de progressivement perdre en efficacité à mesure que l'intervention se prolonge. A l'origine de cette perte d'efficacité, des mécanismes consistants (*e.g.,* l'intégration neuronale des afférences de l'organisme) comme spécifiques à l'intervention (*e.g.,* le développement de traumatismes musculaires liés au parcours, la montée en température du cerveau en ambiance chaude) pourraient être impliqués. Cette nuance dans les causes d'une perte d'efficience cognitive suggère alors, d'une part, des stratégies à systématiser afin de soulager l'état de stress physiologique ou d'augmenter l'état d'activation de l'individu (*e.g.,* les solutions hydriques, nutritionnelles, motivationnelles), mais aussi des manœuvres plus spécifiques (*e.g.,* les solutions thermorégulatrices). Par exemple, l'ingestion de bupropion (un inhibiteur de la recapture de dopamine et d'adrénaline libérées à l'exercice) est identifié pour ses effets ergogéniques



lorsque la performance se déroule en chaleur mais pas lorsque celle-ci a lieu en ambiance tempérée (Watson et *al*., 2005). Les incidences d'une perte d'efficacité à réguler son comportement (*i.e.,* la déficience des composantes technique, tactique, émotionnelle et/ou aérobie de la performance, Badin et al., 2016; Grillon et al., 2015; Marcora et al., 2009; Smith et al., 2014) justifient la mise en place de telles stratégies, ainsi que la nécessité d'une préparation à moyen terme de l'athlète à ces situations de stress (*cf*. partie 1). En revanche, d'un point de vue théorique, la représentation parabolique des performances exécutives à l'exercice que nous proposons contraste avec celle rectiligne de l'échelle 6-20 de pénibilité de l'effort (Borg, 1998). En ce sens, cette discrépance invite à une perspective spécifique – bien que spéculative – du Flush Model relativement à ses corrélats exécutifs.

3. Perspectives

En introduction au Flush Model, nous notions le rôle des efférences (*e.g.,* l'intensité de la commande motrice) et afférences (*e.g.,* le développement de traumatismes musculaires) dans la détermination du ressenti de l'individu à l'exercice. Ces informations sont estimées croitre avec le niveau de sollicitation de l'organisme au cours de l'exercice, dès lors qu'aucune régulation d'allure n'est opérée par l'athlète (Millet, 2011). Par ailleurs, nos travaux suggéraient une réponse dynamique du fonctionnement exécutif à l'exercice (*i.e.,* sous la forme d'un U-inversé) au sein de laquelle une activation physiologique optimale apparaissait bénéfique aux performances cognitives. Dans ce contexte, en raison des chevauchements mécanistiques supposés entre ces deux types de réponse à l'exercice, les performances cognitives à l'exercice pourraient être maximales à des valeurs moyennes de RPE représentatives de ce degré d'activation optimale de l'organisme *i.e.,* ni à des scores RPE maximaux, ni minimaux. Autrement dit, dans le contexte du Flush Model, un ressenti d'effort 'léger', ou proche (*i.e.,* ~13 sur une échelle de 6 à 20, Borg, 1998), pourrait effectivement s'avérer synonyme d'un niveau de performance cognitive maximal, dans la mesure où celle-ci perdrait en efficacité en amont et au-delà de ce niveau d'activation. La nature de cette relation 'RPE – cognition'



reste à investiguer (*e.g.*et demeure donc spéculative ; néanmoins, elle apparait supportée par les implications neuronales du PFC dans le traitement des afférences et efférences à l'exercice (*cf*. introduction) et l'évolution de la réponse biochimique de l'organisme aux durées et intensités d'exercice (McMorris et *al*., 2016), lui conférant un rôle potentiel dans les activités de prise de décision soumises à la fatigue.

Si les études ayant investigué le fonctionnement cognitif à l'exercice par l'intermédiaire de tâches cognitives sont abondantes (Lambourne & Tomporowski, 2003 ; McMorris et *al*., 2009), en revanche, l'analyse de ce fonctionnement dans le cadre écologique des sports d'endurance (*i.e.,* rapportée à la gestion d'allure de l'athlète) reste pauvre. Ce n'est en effet que récemment que l'impact de la charge cognitive sur la stratégie d'allure de l'individu a été caractérisé, pour mettre en lumière un ralentissement de l'allure de course proportionnel à la complexité de l'opération cognitive à résoudre (McCarron et *al*., 2013). Ainsi, avec l'ensemble de ces travaux en tête, se pose la question d'une relation entre la stratégie d'allure et la dynamique d'efficacité cognitive à l'exercice. En d'autres termes, une demande cognitive similaire pourrait ne pas entraîner les mêmes effets sur la gestion d'allure de l'individu selon que cette demande soit requise en début, milieu ou fin d'épreuve. Bien qu'intuitive, cette réponse de variation d'allures à celle des variations d'efficience cognitive demeure à investiguer. L'enjeu de telles études pourrait résider dans l'identification de stratégies d'intervention spécifiques au timing de l'épreuve *i.e.,* spécifiques aux variations de scores RPE. A titre d'exemple, alors que les stratégies d'élévation de la température interne apparaissent à recommander en

début d'épreuve (en raison de leur faculté à bénéficier aux opérations cognitives), les stratégies d'élévation de la température cutanée semblent à proscrire (Schmit et al. 2016c), conférant aux applications thermiques locales un intérêt particulier (*e.g.,* CryoVest®).

4. Conclusion

Les travaux de cette thèse se sont penchés sur la compréhension des versants psychologique et physiologique de la performance en endurance, à l'instar du rationnel du Flush Model (Millet, 2011). Dans la perspective des Jeux Olympiques de Rio, cette approche systémique de l'individu à l'exercice a aussi été confrontée aux problématiques de gestion des charges d'entraînement et de sollicitations cognitives propres à la phase précompétitive, ainsi qu'à celles spécifiques de l'exercice en conditions chaudes. Dès lors, les résultats de ces travaux peuvent servir de support pour complémenter la dimension écologique du Flush Model.

Les études relatives à la gestion des charges d'entraînement se sont intéressées à la réponse de l'athlète à des périodes de surcharge d'entraînement. Confrontées les unes aux autres, ces études concluent sur l'existence d'une limite fine entre l'impact ergogénique *vs.* délétère de la surcharge sur la réponse de performance de l'athlète, limite décelable de façon complexe au sein des réponses de l'organisme à l'exercice, et possiblement indépendante de la nature de la surcharge. En effet, des réponses psychophysiologiques convergentes pouvaient être remarquées chez des athlètes surmenés par une augmentation de leur volume d'entraînement comme par l'accroissement de la contrainte thermique endogène pendant l'entraînement. Dès lors, d'un point de vue pratique, ces études participent à la sensibilisation et la formulation de préconisations aux entraîneurs (*e.g.,* distinguer les variables sur lesquelles rechercher une évolution de celle où cet écart est à prévenir). Sur le plan scientifique, elles invitent à poursuivre les investigations relatives à l'augmentation de la charge *interne* d'entraînement (*i.e.,* la réponse subjective à une charge donnée) afin de déterminer la dose d'entraînement optimale préconisée pour *un* athlète avant la période d'affutage. Ainsi, de futures études devraient permettre de spécifier les magnitudes de variation de charge d'entraînement tolérées selon la nature du stress appréhendé au cours de la période de surcharge (*e.g.,* chaleur, altitude, volume d'entraînement).

Les études de cette thèse ayant investigué la réponse cognitive à l'exercice dessinent, indépendamment du contexte tempéré ou chaud d'investigation, une représentation dynamique du fonctionnement exécutif. Cette réponse était peu formalisée au sein de la littérature. Précisément, bien que des mécanismes différents puissent entrer en jeu, les performances relatives aux fonctions exécutives (*e.g.,* la mémoire de travail, l'inhibition sélective) semblent bénéficier des effets de l'exercice comme de ceux de la chaleur lors des premiers temps d'activité/d'exposition, avant de décliner. Cette caractérisation « instable » de la réponse cognitive constitue une première étape permettant d'envisager plus avant les implications pratiques d'un entraînement cognitif pour la performance en endurance ou l'exercice en chaleur. Les résultats de ces études suggèrent ainsi une grille de lecture à travers laquelle pourraient être interprétés les comportements de terrain, fondée sur la capacité de l'athlète à réguler efficacement (*i.e.,* de façon rapide et précise) son comportement lorsqu'il est confronté à une situation imprévue. Alors que nous avons tenté de rendre fonctionnels ces résultats en les abordant par l'intermédiaire des fonctionnalités du Flush Model (*i.e.,* le remplissage et la vidange de la cuvette.), les futures études relatives à l'étude de l'interaction exercice-cognition pourront certainement prétendre à la définition de stratégies d'entraînements cognitifs (*e.g.,* Romeas et *al*., 2016), comme de moyens permettant de contrecarrer la perte de lucidité inhérente au développement de la fatigue pendant l'effort prolongé ou l'exercice en chaleur.

L'essence du Flush Model repose sur sa disposition à cristalliser la performance en endurance autour du RPE. Les travaux de cette thèse ont tenté de relier cette dimension perceptive de l'exercice à la préparation à moyen-terme de la performance ainsi qu'à ses versants cognitifs – deux volets oubliés du Flush Model. À haut niveau, la considération de ces facteurs de performance est en effet importante, notamment eu égard à l'éventualité de conditions chaudes lors des Jeux Olympiques de Rio 2016. Les problématiques inhérentes à la préparation des prochains Jeux pourront peut-être alors donner naissance à un nouveau complément au modèle de Millet (2011).

RÉFÉRENCES

Abbiss, C. R., & Laursen, P. B. (2005). Models to explain fatigue during prolonged endurance cycling. *Sports Medicine*, *35*(10), 865-898.

Altareki, N., Drust, B., Atkinson, G., Cable, T., & Gregson, W. (2009). Effects of Environmental Heat Stress (35° C) with Simulated Air Movement on the Thermoregulatory Responses during a 4-km Cycling Time Trial. *International journal of sports medicine*, *30*(01), 9-15.

Amann, M., Proctor, L. T., Sebranek, J. J., Pegelow, D. F., & Dempsey, J. A. (2009). Opioid-mediated muscle afferents inhibit central motor drive and limit peripheral muscle fatigue development in humans. *The Journal of physiology*, *587*(1), 271-283.

Ando, S., Kokubu, M., Yamada, Y., & Kimura, M. (2011). Does cerebral oxygenation affect cognitive function during exercise?. *European journal of applied physiology*, *111*(9), 1973-1982.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in cognitive sciences*, *8*(4), 170-177.

Aubry, A., Hausswirth, C., Louis, J., Coutts, A. J., & Le Meur, Y. (2014). Functional overreaching: the key to peak performance during the taper?. *Medicine and science in sports and exercise*, *46*(9), 1769-1777.

Baden, D. A., McLean, T. L., Tucker, R., Noakes, T. D., & Gibson, A. S. C. (2005). Effect of anticipation during unknown or unexpected exercise duration on rating of perceived exertion, affect, and physiological function. *British Journal of Sports Medicine*, *39*(10), 742-746.

Badin, O. O., Smith, M. R., Conte, D., & Coutts, A. J. (2016). Mental Fatigue Impairs Technical Performance in Small-Sided Soccer Games. *International journal of sports physiology and performance.*

Baldwin, J., Snow, R. J., Gibala, M. J., Garnham, A., Howarth, K., & Febbraio, M. A. (2003). Glycogen availability does not affect the TCA cycle or TAN pools during prolonged, fatiguing exercise. *Journal of Applied Physiology*, *94*(6), 2181-2187.

Bassett, D. R., & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and science in sports and exercise*, *32*(1), 70-84.

Blanchfield, A. W., Hardy, J., De Morree, H. M., Staiano, W., & Marcora, S. M. (2014a). Talking yourself out of exhaustion: the effects of self-talk on endurance performance. *Med Sci Sports Exerc*, *46*(5), 998-1007.

Blanchfield, A., Hardy, J., & Marcora, S. (2014b). Non-conscious visual cues related to affect and action alter perception of effort and endurance performance. *Frontiers in human neuroscience*, *8*, 967.

Borg, G. (1998). *Borg's perceived exertion and pain scales*. Human kinetics.

Bosquet, L., Montpetit, J., Arvisais, D., & Mujika, I. (2007). Effects of tapering on performance: a meta-analysis. *Medicine and science in sports and exercise*, *39*(8), 1358.

Buhle, J.T., Silvers, J.A., Wager, T.D., Lopez, R., Onyemekwu, C., Kober, H., Weber, J., Ochsner, K.N., 2013. Cognitive reappraisal of emotion: a meta-analysis of human neuroimaging studies. Cerebral Cortex 24(11), 2981–2990.

Byrne, C., Lee, J. K. W., Chew, S. A. N., Lim, C. L., & Tan, E. Y. M. (2006). Continuous thermoregulatory responses to mass-participation distance running in heat. *Medicine and science in sports and exercise*, *38*(5), 803.

Chambers, E. S., Bridge, M. W., & Jones, D. A. (2009). Carbohydrate sensing in the human mouth: effects on exercise performance and brain activity. *The Journal of Physiology*, *587*(8), 1779-1794.

Chang, Y. K., Labban, J. D., Gapin, J. I., & Etnier, J. L. (2012). The effects of acute exercise on cognitive performance: a meta-analysis. *Brain research*, *1453*, 87-101.

Cheuvront, S. N., Kenefick, R. W., Montain, S. J., & Sawka, M. N. (2010). Mechanisms of aerobic performance impairment with heat stress and dehydration. *Journal of Applied Physiology*, *109*(6), 1989-1995.

Cuddy, J. S., Hailes, W. S., & Ruby, B. C. (2014). A reduced core to skin temperature gradient, not a critical core temperature, affects aerobic capacity in the heat. *Journal of thermal biology*, *43*, 7-12.

Decroix, L., Piacentini, M. F., Rietjens, G., & Meeusen, R. (2016). Monitoring Physical and Cognitive Overload During a Training Camp in Professional Female Cyclists. *International journal of sports physiology and performance*.

Delp, M. D., & Laughlin, M. H. (1998). Regulation of skeletal muscle perfusion during exercise. *Acta physiologica Scandinavica*, *162*(3), 411-419.

Drew, M. K., & Finch, C. F. (2016). The relationship between training load and injury, illness and soreness: a systematic and literature review. *Sports Medicine*, 1-23.

Dupuy, O., Lussier, M., Fraser, S., Bherer, L., Audiffren, M., & Bosquet, L. (2014). Effect of overreaching on cognitive performance and related cardiac autonomic control. *Scandinavian journal of medicine & science in sports*, *24*(1), 234-242.

Ely, B. R., Ely, M. R., Cheuvront, S. N., Kenefick, R. W., DeGroot, D. W., & Montain, S. J. (2009). Evidence against a 40 C core temperature threshold for fatigue in humans. *Journal of Applied Physiology*, *107*(5), 1519-1525.

Evans, J. S. B. (2008). Dual-processing accounts of reasoning, judgment, and social cognition. *Annu. Rev. Psychol.*, *59*, 255-278.

Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, Doleshal P, Dodge C. A new approach to monitoring exercise training. *J Strength Cond Research*. 2001; 15(1): 109-115.

Fuster, J.M., 1997. Network memry. Trends in Neurosciences 20, 451–459.

Gallagher, K. M., Fadel, P. J., Strømstad, M., Ide, K., Smith, S. A., Querry, R. G., ... & Secher, N. H. (2001). Effects of partial neuromuscular blockade on carotid baroreflex function during exercise in humans. *The Journal of Physiology*, *533*(3), 861-870.

González-Alonso, J., & Calbet, J. A. (2003). Reductions in systemic and skeletal muscle blood flow and oxygen delivery limit maximal aerobic capacity in humans. *Circulation*, *107*(6), 824-830.

Green, H. J. (1997). Mechanisms of muscle fatigue in intense exercise. *Journal of sports sciences*, *15*(3), 247-256.

Grillon, C., Quispe-Escudero, D., Mathur, A., & Ernst, M. (2015). Mental fatigue impairs emotion regulation. *Emotion*, *15*(3), 383.

Guy, J. H., Deakin, G. B., Edwards, A. M., Miller, C. M., & Pyne, D. B. (2015). Adaptation to hot environmental conditions: an exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. *Sports Medicine*, *45*(3), 303-311.

Hill, A. V., & Lupton, H. (1923). Muscular exercise, lactic acid, and the supply and utilization of oxygen. *QjM*, (62), 135-171.

Hillman, C. H., Erickson, K. I., & Kramer, A. F. (2008). Be smart, exercise your heart: exercise effects on brain and cognition. *Nature reviews neuroscience*, *9*(1), 58-65.

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in cognitive sciences*, *16*(3), 174-180.

Jacobson, J., & Matthaeus, L. (2014). Athletics and executive functioning: How athletic participation and sport type correlate with cognitive performance. *Psychology of Sport and Exercise*, *15*(5), 521-527

Jenkins, I.H., Brooks, D.J., Nixon, P.D., Frackowiak, R.S.J., Passingham, R.E.., 1994. Motor sequence learning: a study with positron emission tomography. Journal of Neuroscience 14, 3775–3790.

Kayser, B. (2003). Exercise starts and ends in the brain. *European journal of applied physiology*, *90*(3-4), 411-419.

Lambourne, K., Tomporowski, P., 2010. The effect of exercise-induced arousal on cognitive task performance: A meta-regression analysis. Brain Research 1341, 12-24

Lawrence, J. M., Hoeft, F., Sheau, K. E., & Mackey, S. C. (2011). Strategy-dependent dissociation of the neural correlates involved in pain modulation. *The Journal of the American Society of Anesthesiologists*, *115*(4), 844-851.

Le Meur, Y., Hausswirth, C., Natta, F., Couturier, A., Bignet, F., & Vidal, P. P. (2013). A multidisciplinary approach to overreaching detection in endurance trained athletes. *Journal of applied physiology*, *114*(3), 411-420.

Le Meur et al., 2016

Marcora, S. M., Bosio, A., & de Morree, H. M. (2008). Locomotor muscle fatigue increases cardiorespiratory responses and reduces performance during intense cycling exercise independently from metabolic stress. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *294*(3), R874-R883.

Marcora, S. (2009). Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart, and lungs. *Journal of Applied Physiology*, *106*(6), 2060-2062.

Marcora, S. M., Staiano, W., & Manning, V. (2009). Mental fatigue impairs physical performance in humans. *Journal of Applied Physiology*, *106*(3), 857-864.

Marcora, S. M., & Staiano, W. (2010). The limit to exercise tolerance in humans: mind over muscle?. *European journal of applied physiology*, *109*(4), 763-770.

Martin, B. J. (1981). Effect of sleep deprivation on tolerance of prolonged exercise. *European journal of applied physiology and occupational physiology*, *47*(4), 345-354.

McCarron, J., Hodgson, T. L., & Smith, M. F. (2013). Brain drain: Evaluating the impact of increased cognitive load during self-paced running performance. *British journal of sports medicine*, *47*(17), e4-e4.

McMorris, T., Tomporowski, P.D., Audiffren, M., 2009. (Eds) Exercise and cognitive function. Wiley, New-York

McMorris, T., Turner, A., Hale, B. J., & Sproule, J. (2016). Beyond the catecholamines hypothesis for an acute exercise-cognition interaction: a neurochemical perspective. *Exercise-Cognition Interaction: Neuroscience Perspectives, edited by McMorris T. New York: Academic*, 65-104

Meeusen, R., Duclos, M., Foster, C., Fry, A., Gleeson, M., Nieman, D., ... & Urhausen, A. (2013). Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Medicine and science in sports and exercise*, *45*(1), 186-205.

Millet, G. Y., Lepers, R., Maffiuletti, N. A., Babault, N., Martin, V., & Lattier, G. (2002). Alterations of neuromuscular function after an ultramarathon. *Journal of applied physiology*, *92*(2), 486-492.

Millet, G. Y. (2010). Central fatigue is not the source but can explain performance decrement due to afferent feedback. *Journal of applied physiology (Bethesda, Md.: 1985)*, *108*(2), 464-author.

Millet, G. Y., Aubert, D., Favier, F. B., Busso, T., & Benoit, H. (2009). Effect of acute hypoxia on central fatigue during repeated isometric leg contractions. *Scandinavian journal of medicine & science in sports*, *19*(5), 695-702.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive psychology*, *41*(1), 49-100.

Nielsen, B., Strange, S., Christensen, N. J., Warberg, J., & Saltin, B. (1997). Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflügers Archiv*, *434*(1), 49-56.

Noakes, T. D., Gibson, A. S. C., & Lambert, E. V. (2004). From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *British journal of sports medicine*, *39*(2), 120-124.

Nybo, L., & Nielsen, B. (2001). Hyperthermia and central fatigue during prolonged. *J Appl Physiol*, *91*, 1055-1060.

Nybo, L. (2003). CNS fatigue and prolonged exercise: effect of glucose supplementation. *Medicine and science in sports and exercise*, *35*(4), 589-594.

Nybo, L. (2012). Endurance training and competition in the heat. In *Endurance Training*. Iñigo Mujika.

Nybo, L., Rasmussen, P., & Sawka, M. N. (2014). Performance in the Heat—Physiological Factors of Importance for Hyperthermia-Induced Fatigue. *Comprehensive Physiology*.

Ochsner, K.N., Gross, J.J., 2008. Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. Current Directions in Psychological Science 17, 153–158.

Ogoh, S. Tsukamoto, H., Hirasawa, A., Hasegawa, H., Hirose, N., Hashimoto, T., 2014. The effect of changes in cerebral blood flow on cognitive function during exercise. Physiol Rep. doi:10.14814/phy2.12163

Périard, J. D., Cramer, M. N., Chapman, P. G., Caillaud, C., & Thompson, M. W. (2011). Cardiovascular strain impairs prolonged self-paced exercise in the heat. *Experimental physiology*, *96*(2), 134-144.

Racinais, S., Périard, J. D., Karlsen, A., & Nybo, L. (2015). Effect of heat and heat acclimatization on cycling time trial performance and pacing. *Med Sci Sports Exerc*, *47*(3), 601-6.

Raglin, J. S. (2007). The psychology of the marathoner. *Sports Medicine*, *37*(4-5), 404-407.

Romeas, T., Guldner, A., & Faubert, J. (2016). 3D-Multiple Object Tracking training task improves passing decision-making accuracy in soccer players. *Psychology of Sport and Exercise*, *22*, 1-9

Rooks, C.R., Nathaniel, J.T., McCully, K.K., Dishman, R.K., 2010. Effects of incremental exercise on cerebral oxygenation measured by near-infrared spectroscopy : a systematic review. Progress in Neurobiology 92, 134–150

Sawka, M. N., Leon, L. R., Montain, S. J., & Sonna, L. A. (2011). Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. *Comprehensive Physiology.*

Sawka, M. N., Cheuvront, S. N., & Kenefick, R. W. (2012). High skin temperature and hypohydration impair aerobic performance. *Experimental physiology*, *97*(3), 327-332.

Schmit, C., Davranche, K., Easthope, C. S., Colson, S. S., Brisswalter, J., & Radel, R. (2015a). Pushing to the limits: the dynamics of cognitive control during exhausting exercise. *Neuropsychologia*, *68*, 71-81.

Schmit, C., Duffield, R., Hausswirth, C., Coutts, A. J., & Le Meur, Y. (2015b). Pacing Adjustments Associated With Familiarisation: Heat vs. Temperate Environments. *International journal of sports physiology and performance*.

Schmit, C., Le Meur, Y., Duffield, R., Robach, P., Oussedik, N., Coutts, A. J., & Hausswirth, C. (2015c). Heat-acclimatization and pre-cooling: a further boost for endurance performance? *Scandinavian journal of medicine & science in sports*.

Schmit, C., & Brisswalter, J. (2016) Executive functioning during prolonged exercise: A fatigue based-neurocognitive perspective. *Under review* (in *Int Rev Sport Exerc Psychol*).

Schmit, C., Blain, B., Hausswirth, C., Pessiglione, M., & Le Meur, Y. (2016a). Neural correlates of functional overreaching in endurance athletes.

Schmit, C., Duffield, R., Hausswirth, C., Brisswalter, J., & Le Meur, Y. (2016b). Optimizing heat-acclimation for performance peaking: high- vs low-intensity training. *Submitted* (in *Medicine and Science in Sports and Exercise*)

Schmit, C., Hausswirth, C., Le Meur, Y,, & Duffield, R. (2016c). Cognitive functioning and heat strain: performance responses and protective strategies. *Under review* (in *Sports Medicine*).

Seiler, K. S., & Kjerland, G. Ø. (2006). Quantifying training intensity distribution in elite endurance athletes: is there evidence for an “optimal” distribution?. *Scandinavian journal of medicine & science in sports*, *16*(1), 49-56.

Seiler, S. (2010). What is best practice for training intensity and duration distribution in endurance athletes. *Int J Sports Physiol Perform*, *5*(3), 276-291.

Shulman, R. G., & Rothman, D. L. (2001). The “glycogen shunt” in exercising muscle: a role for glycogen in muscle energetics and fatigue. *Proceedings of the National Academy of Sciences*, *98*(2), 457-461.

Smith, M. R., Marcora, S. M., & Coutts, A. J. (2014). Mental fatigue impairs intermittent running performance. *Med Sci Sports Exerc.*

Swick, D., Ashley, V., & Turken, U. (2008). Left inferior frontal gyrus is critical for response inhibition. *BMC neuroscience*, *9*(1), 1.

Tatterson, A. J., Hahn, A. G., Martini, D. T., & Febbraio, M. A. (2000). Effects of heat stress on physiological responses and exercise performance in elite cyclists. *Journal of Science and Medicine in Sport*, *3*(2), 186-193.

Tomporowski, P.D., 2003. Effects of acute bout of exercise on cognition. Acta Psychol 112, 297-324

Tucker, R. (2009). The anticipatory regulation of performance: the physiological basis for pacing strategies and the development of a perception-based model for exercise performance. *British Journal of Sports Medicine*, *43*(6), 392-400.

Tucker, R., Marle, T., Lambert, E. V., & Noakes, T. D. (2006). The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *The Journal of physiology*, *574*(3), 905-915.

Vestberg, T., Gustafson, R., Maurex, L., Ingvar, M., & Petrovic, P. (2012). Executive functions predict the success of top-soccer players. *PloS one*, *7*(4), e34731.

Watson, Phillip, et al. "Acute dopamine/noradrenaline reuptake inhibition enhances human exercise performance in warm, but not temperate conditions." *The Journal of physiology* 565.3 (2005): 873-883.

LE PHÉNOMÈNE DE FATIGUE EN CONDITIONS EXTRÊMES :

LE « FLUSH MODEL » À L'ÉPREUVE DES JEUX OLYMPIQUES

Les modèles explicatifs de l'état d'épuisement lors de performances en endurance se sont récemment distancés des seules implications périphériques pour magnifier le rôle du système nerveux central dans le développement de la fatigue. En particulier, le « Flush Model » (Millet, 2011) a rassemblé les opinions en introduisant la pénibilité perçue à l'exercice en tant que régulateur princeps de la performance. Brièvement, cette sensation d'effort émergerait de l'ensemble des interactions psycho-physiologiques inhérentes à l'accomplissement de l'exercice et demeurerait modulable pour retarder l'atteinte de l'état d'épuisement.

Au sein de cette thèse et dans la perspective des Jeux Olympiques (JO) de Rio, nos efforts se sont concentrés autour de la mise en pratique de ce modèle au service de la performance des athlètes. Les aspects fonctionnels du Flush Model ont ainsi été revisités tant à l'égard des contraintes de l'entraînement que de celles propres à cette compétition (*i.e.,* l'éventualité d'un stress thermique important). Plus précisément, le développement du phénomène de fatigue a été confronté aux problématiques de charges d'entraînement et de performances cognitives à travers une double approche de la compétition.

Une approche aigüe de la compétition renvoie à l'ensemble des stratégies ponctuelles dont dispose l'athlète pour moduler l'évolution de la perception de son effort et optimiser le rapport « difficulté perçue / intensité d'exercice » au cours de l'épreuve. De façon spécifique aux épreuves en endurance et au contexte potentiellement chaud des JO, cinq travaux ont alors été entrepris. Deux d'entre eux ont appréhendé les effets sur la performance en contre-la-montre en conditions chaudes de stratégies à court-terme *i.e.,* la familiarisation à la chaleur, et le port d'une veste réfrigérante lors de la période d'échauffement. Les résultats indiquent une amélioration de la stratégie d'allure des sujets possiblement induite par des adaptations psycho-physiologiques spécifiques à l'intervention. En complément, trois autres travaux ont étudié la réponse cognitive à l'exercice aigu, en conditions tempérées puis de chaleur, afin de mieux comprendre l'évolution des paramètres d'autorégulation comportementale (*i.e.,* les fonctions exécutives) impliqués dans la performance sportive. Les dynamiques cognitives identifiées suggèrent l'utilisation de stratégies ponctuelles et durables susceptibles de préserver le niveau d'efficacité de l'individu à l'exercice.

Une approche chronique de la compétition fait écho aux interventions durables mises en place par l'athlète en phase précompétitive et visant à maximiser son niveau de performance le jour de l'épreuve. Trois travaux ont été entrepris dans cette perspective. Deux d'entre eux ont investi les problématiques de charge d'entraînement et d'acclimatation à la chaleur afin de déterminer les effets sur la performance et les composantes psycho-physiologiques de stages d'acclimatation à la chaleur. Ces études démontrent la nécessité d'un ajustement précis des charges d'entraînement en chaleur afin de ne pas engendrer chez l'athlète l'installation d'un état de surmenage contre-productif. Un travail complémentaire a consisté à analyser, via imagerie par résonance magnétique fonctionnelle, les soubassements neuronaux de l'état de surmenage possiblement rencontré par les athlètes en phase précompétitive. Les résultats de cette étude indiquent une moindre activation du cortex préfrontal lors de la réalisation de tâches cognitives, accompagnée de réponses comportementales tendant vers un plus grand degré d'impulsivité.

Ensemble, ces travaux autorisent une approche complexe du phénomène de fatigue, à partir d'angles de vue aigu et chronique, mais aussi physiologique et central, de ses composantes. Sur la base d'une plus grande compréhension de ce phénomène, et des apports fonctionnels du Flush Model, des stratégies peuvent ainsi être préconisées aux athlètes afin d'optimiser tant leur condition physique en phase précompétitive que leur niveau d'efficacité lors de l'épreuve.

MOTS-CLÉS : Haut niveau ; Performance en endurance ; Cognition ; Refroidissement ; Acclimatation à la chaleur ; Surmenage