TO THE EDITOR: We are grateful to our colleagues in providing valuable commentary to our Viewpoint (3). We respond to the key points as we see them. Our aim was to present potential cerebral pathways involved in exercise tolerance, regulation and termination. As stated by Dr. Lutz (see Ref. 1), this model is by no means exclusive and we hope it serves further exploration. We appreciate Micklewright et al.’s (see Ref. 1) recognition that methodological limitations currently mean that we need to draw on neurocognitive evidence to begin the exploration of this area. We certainly agree with Professor Meeusen (see Ref. 1) that we do not have the tools available yet to fully investigate this area. Therefore as per the recommendations within the commentaries, it is important to continue with interdisciplinary research examining neurophysiological responses in conjunction with the measurement and manipulation of internal and external factors. As proposed by Micklewright et al. it is important to consider external factors altering motivational status (see Ref. 1) but also environmental factors altering perceived exertion (2).

Professor Meeusen (see Ref. 1) correctly interprets our discussion proposing that the brain integrates various signals and emotions but points out that the brain may not make decisions to avoid catastrophe. We would like to clarify that it was not our intention to prove nor refute the tenet of the Central Governor Model but rather provide potential pathways involved in the interpretation of physiological signals and other factors present in exercise environments, which might influence what decisions are made about exercise regulation. As stated by Drs. Rauch and Pires (see Ref. 1), these brain processes are likely to be different depending on the situation of the sporting event and exercise model in question. Dr. Pires (see Ref. 1) outlines how changes in metabolic costs measured at the PFC suggest a greater involvement of the PFC in more demanding exercise bouts. Professor Cheung (see Ref. 1) provides evidence that highly fit participants have a greater ability to tolerate physiological sensations and provide better performances in conjunction with attenuated insular activation. This adds weight to the concept that repeated tolerance of adverse sensations can aid in the ability to tolerate them (3, 4).

Dr. Perrey (see Ref. 1) confirms that the orbitofrontal cortex and anterior cingulate are involved in the cost benefit analysis of exerciser tolerance and adds that the PFC may become disengaged, leading to exercise cessation. Dr. Rauch (see Ref. 1) adds that the point of fatigue may be attributable to subcortical structures with reduced PFC processing, particularly at higher exercise intensities. We agree that there is likely a strong subcortical component to this regulatory process, e.g., dopamine release. However, if indeed the PFC is involved in motivational processes as discussed by Dr. Lutz (see Ref. 1), at which point does this motivation become redundant in the face of this alternate system? Future research needs to develop an understanding of both neural correlates of exercise regulation and fatigue but also the psychological elements that go with this. As it stands, our understanding of psychological processes affecting exercise are predominantly limited to theoretical constructs rather than neurophysiological responses and it is difficult to connect the two.

DISCLOSURES
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AUTHOR CONTRIBUTIONS
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