letters to the editor

Blood glucose turnover during exercise above and below the lactate threshold

To the Editor: The recent article of Coggan et al. (1) largely confirmed our previous publications [in 1986 (3) and 1989 (2)] that blood glucose turnover during exercise is dramatically increased when the work performed is above the lactate threshold (LT). We concluded (2), "When work rate protocols are designed using a fixed percentage of the [maximal oxygen uptake] $\dot{V}O_{2\,max}$. . . , the result will invariably be a mixed sample population with subjects exercising both below and above the lactate threshold." This was echoed by Coggan et al., who noted, "hormonal and metabolic responses during exercise may differ considerably . . . when exercise is performed at the same percentage of the $\dot{V}O_{2\,max}$."

Remarkably, Coggan et al. (1) overlooked these and other works and, in so doing, missed important mechanisms, namely, those related to reduced oxygen availability, that could explain the increased glucose uptake in their subjects. For example, we demonstrated in 1986 (3) that by manipulating the subject's fractional concentration of inspired oxygen ($F_{I_{O_2}}$), below-LT work could become above-LT work without changing the work rate itself. Glucose turnover was markedly increased when subjects breathed hypoxic gas ($F_{I_{O_2}}$ = 0.15) compared with hyperoxic gas ($F_{I_{O_2}}$ = 0.8), even though the work rate was the same under both conditions. Our data are not explained by differences among the subjects' mitochondrial oxidative capacity (as proposed by Coggan et al. to interpret their results), since in our experiments both exercise protocols were performed by the same subjects. In our 1989 study (2), we showed that blood glucose turnover for above-LT compared with below-LT exercise was increased to a much greater extent than was the metabolic rate ($\dot{V}O_2$). Again, both the above- and below-LT protocols were performed by the same subjects. Our previous studies entirely predicted the results obtained by Coggan et al.: for a given work rate, blood glucose turnover will be increased if the subject is exercising above his or her LT.

The authors' failure to even comment on the possible role of oxygen availability (in addition to mitochondrial oxidative capacity) as a mechanism for the association of increased serum lactate with high glucose turnover in their subjects is surprising. Coggan et al. have ignored a large body of experimental and clinical evidence originating with Pasteur over a century ago and ranging to the classic, more contemporary, work of Randle and Smith (5) and Idstrom et al. (4) that supports the idea that glucose uptake and metabolism in muscle and other tissues can be profoundly mediated by oxygen availability.

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