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LETTER TO THE EDITOR

Adaptation to Maximal Effort

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In their recent paper (Acta Genet Med Gemellol 33:565-569, 1984), DeMeersman and coworkers reported some interesting observations concerning the effects of exercise-training on the response of maximal oxygen uptake in one set of MZ triplets [6]. The association of the Human Performance Laboratory at Virginia Commonwealth University with the well established twin scientists of the Department of Human Genetics at the Medical College of Virginia (Richmond) raises great hope for this field in need of vigorous and careful experimental research. This paper is quite correct from the experimental point of view. Their interpretation of the findings is not quite clear, however, and it prompts me to make the following observations:

- a) Three individuals with the same genotype are involved: one trained for about 10 hr, a second for 90 hr, and a third for 150 hr, over a period of about 4 months. The first member of the siblings is characterized as untrained while the other two are said to be exposed to training of different frequencies. This is not quite true as it is clearly stated that training was performed 5 times a week in the two trained triplets. In other words, the difference is not in the frequency of training but rather in duration. This is not a trivial point, as we have shown earlier that frequency of training was the single most important dimension of training provided you have reached a threshold in intensity [5].
- b) In the present design, changes in maximal aerobic power per se are best represented by $\max \text{VO}_2$ in 1 O_2 as body weight may have decreased with regular training. From Table 1, one finds that the sedentary triplet did not improve over 4 months, while the 90 hr triplet gained 0.49 l O_2 (or 16%) and the 150 hr triplet improved by 0.53 l O_2 (or 18%).
- c) Our interpretation of the data is that both triplets who trained made similar gain in maximal aerobic power. The difference between increases of 16% and 18% is trivial

and well within the error of measurement of max VO_2 . In other words, a difference in duration or total volume of training over 4 months did not have any effects on the trainability of max VO_2 for a given genotype.

- d) In reality, what was tested in this case study was the response of two training durations for one genotype. Under these circumstances, one should not use the important notion of genotype-training interaction [1-5,7,9] as only one genotype and not quite two training regimens were tested. In our opinion what was really shown here was that training of unlike duration could induce similar gain in max VO_2 for one genotype. This does not necessarily apply to other genotypes as we have shown that there are low-responders (and sometimes non-responders) and high-responders to exercise-training [1,2,9].
- e) The only way to account for the data in max VO_2 , provided there is no error variance, is to take into consideration the mean genetic effect in the population (heritability for the trait), the mean environmental and lifestyle effect and the true genotype-training interaction effect as defined elsewhere [2,3].

In other words, variation in cardiorespiratory variables cannot be accounted for by a simple additive model of heredity plus environment as was proposed earlier by Klissouras [8] and as concluded by the authors. Thus, I would have to disagree with DeMeersman and coworkers unless it is a problem of terminology which, I suspect, may be the case.

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