TEN YEARS AGO Claude Bouchard et al. (1) reported in the HERITAGE Family Study that aerobic exercise training programs result in large heterogeneity in \( V_{O2max} \) response. The mean \( V_{O2max} \) response ranged from 293 ml/min in mothers to 486 ml/min in sons, with highest responses up to 1 l/min. However, there were low responders, nonresponders, and even those who responded negatively. HERITAGE Study has shown that in addition to \( V_{O2max} \), this variability in response to regular exercise is present in almost any measured parameter including heart rate during submaximal exercise, systolic blood pressure during steady-state exercise, and HDL cholesterol (2). There are also a handful of older and more recent smaller studies showing no or even negative correlations between changes in aerobic performance and \( V_{O2max} \) (e.g., 4–6).

Exercise physiologists often consider \( V_{O2max} \) as a measure of aerobic performance. The variability in responses to exercise training is seen more or less as a “statistical nuisance” that dilutes the mean increases seen after exercise programs. Because, in addition to increase athletic performance, exercise training is regarded and used as a tool to prevent and mend metabolic and various other diseases, the reasons of individual response differences should be carefully considered and studied. It is quite astonishing that this large variability in responses has not really caught the attention of researchers. What is the link between the improvement of oxygen transport system and the improvement of aerobic performance? This question was presented in a study by Vollaard et al. (8) in the Journal of Applied Physiology, especially considering how muscle metabolic control is related to training-induced improvements in \( V_{O2max} \) and aerobic performance.

And how did Vollaard et al. (8) study these relations? They simply trained young healthy men at 70% \( V_{O2max} \) for 6 wk in a laboratory setting and determined among other parameters training-induced changes in \( V_{O2max} \) and aerobic performance. Furthermore, they took muscle biopsy samples to quantify substrates and enzyme activities of energy metabolism. Biopsy sampling was performed just before and directly after a 10-min submaximal cycling test before and after the training period. As one may expect, exercise training improved the mean values of various physiological parameters (including \( V_{O2max} \)) and aerobic performance. However, individual adaptations varied from negative values to substantial increases. Most importantly, low responders to one variable were not consistent low responders to other variables, suggesting that also the balance of adaptive mechanisms vary from one person to another. \( V_{O2max} \) and aerobic performance correlated strongly at baseline and after training but the change in \( V_{O2max} \) and the change in performance were not related at all (\( r^2 = 0.05 \)). Furthermore, Vollaard et al. (8), by combining maximal and submaximal physiological and biochemical parameters, demonstrated that changes in muscle metabolism may explain changes in performance but not in \( V_{O2max} \).

What are the implications of these results? As Vollaard et al. (8) state, standardizing training intensity to a certain percentage of \( V_{O2max} \) in training studies will yield large interindividual variation in performance and in metabolic responses. Therefore, \( V_{O2max} \) should not be considered a universal standardization parameter in aerobic exercise training research. More importantly, the individual variation in responses may have considerable ramifications to the use of exercise as a tool in health care. It is conceivable that general exercise advice, e.g., in the prevention of obesity, the metabolic syndrome, or Type 2 diabetes, does not yield the best possible results. For instance, the new 2008 Physical Activity Guidelines for Americans (7) recommend for adults up to 5 h a week of moderate intensity or 2 h 30 min a week of vigorous-intensity physical activity, or an equivalent combination of both. This and even a smaller amount of work will purportedly yield health benefits but individuality in exercise responses very possibly requires individually fitted training programs for the best possible results. Individual exercise procedures become even more important when exercise is used to treat diseases so that the patients would gain more with less effort. Perhaps it is time to start considering and studying the most practical methods how to determine individual exercise programs with best individual gain.

What are then the mechanisms and regulatory processes that participate and lead to the diversity in adaptations? Vollaard et al. (6) propose that calcium-mediated signaling could do the trick in aerobic performance, and HIF1α or VEGF signaling by promoting vascular remodeling might be central for improving oxygen related processes. Obviously this is a very complicated entirety that will be difficult to unravel only by exploiting human exercise studies. Such studies can provide only limited amounts of required tissue samples. One solution to avoid this problem is to develop a suitable experimental model. A project to create rat genetic models for the wide variation that exists for the adaptational component of aerobic capacity is presently going on at the University of Michigan (3). The idea is to produce rats with high response to training and rats with low response to training. Finally, the selectively bred strains are expected to develop contrasting phenotypes and gene networks that determine exercise responsiveness to training. If true, these animal models, in addition to human experiments, could be used to resolve the mechanistic basis for the gene by environment interaction for exercise training response.

REFERENCES


