Commentaries on Viewpoint: Do oxidative and anaerobic energy production in exercising muscle change throughout growth and maturation?

MANIFESTATIONS OF A COMMON UNDERLYING CAUSE

TO THE EDITOR: Ratel et al. (5) presented the view that children’s glycolytic capacity increases with maturation. This clearly manifests itself in the maturational increase of anaerobic power. Conversely, the apparent concurrent decrease in oxidative capacity, as presented in the Viewpoint, is not coupled with a corresponding decrease in aerobic performance capacity. Also, children demonstrate lower (size normalized) muscle contractility (single-contraction force and velocity) (2), which cannot be explained by inferior glycolytic or even phospholytic apparatus. The totality of these performance disparities cannot be explained by the energy kinetics or histochemical evidence of child-adult differences. Rather, those differences can be reconciled by proposing that children are appreciably more limited in their capacity to utilize higher-threshold, glycolytic, type-II motor units. Lower reliance on glycolytic motor units and greater dependence on oxidative ones can explain a whole range of metabolic and performance child-adult differences, notably the following.

1) Children’s seeming oxidative superiority (5), e.g., anaerobic threshold at higher %V\textsubscript{\text{O}}\text{\textsubscript{2}}\text{max}.
2) Children’s inferiority in both anaerobic and contractile capacities, and lower peak-lactate levels (1, 2).
3) Children’s lower fatigability (4) and faster recovery from high-intensity exercise (1).
4) Children’s glycolytic/phospholytic enzyme deficiencies (5) (a disuse effect, due to type II motor units being little used).

While true maturational changes in muscle oxidative and glycolytic capacities cannot be ruled out, they can explain neither the scope nor the extent of observed child-adult differences. Although direct support for the maturation-dependent motor-unit utilization theory is currently scant (e.g., 3), the hypothesis does account for all the above and other diverse observations.

REFERENCES


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TO THE EDITOR: In their Viewpoint, Ratel and colleagues (3) concluded on the large set of factors that could lead to misleading interpretation when comparing oxidative and anaerobic energy production in exercising muscle change throughout growth and maturation. Some other factors could also be addressed. First, spontaneous physical activity has been reported to decline with growth, with a particular decrease in moderate to vigorous physical activity (2). This data suggest matching “sedentary” children and sedentary adults according to physical activity level (and pattern) when comparing their metabolic responses to exercise. Second, a lower decrement in plasma volume has been reported with children after a maximal graded test (1). In most of the studies that have investigated metabolite concentrations in the blood stream, these differences in transient changes in plasma volume are not taken into account. Blood metabolite concentrations must be corrected according to these changes to accurately compare children and adults responses to exercise. The last point concerns the force or power output production that is considered as an indicator of anaerobic metabolism. Children demonstrated specific anatomical characteristics that could affect force production independently from metabolic factors. For example, Morse and colleagues (2) demonstrated that moment arm leg of the lateral gastrocnemius was 25% smaller in boys while force production normalized to cross sectional area was 21% higher. Well-designed protocols, associated with new non-invasive methodology, will allow some of the queries addressed by Ratel and colleagues (3) to be answered.

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THE IMPORTANCE OF EXERCISE INTENSITY WHEN STUDYING DEVELOPMENTAL ENERGY METABOLISM

TO THE EDITOR: The Viewpoint by Ratel and colleagues (3) serves as a valuable reminder of the myriad of potentially confounding factors and methodological considerations which need to be considered when studying developmental changes in aerobic and anaerobic energy metabolism during exercise. However, an additional factor, which may account for a number of discrepancies in the literature and those discussed by Ratel et al., is the intensity of the imposed exercise bout.

Using the noninvasive technique of 31P-magnetic resonance spectroscopy (1, 2, 4), we have demonstrated age- and sex-related differences in muscle metabolic responses to manifest only during exercise above the intracellular threshold (IT) for
pH. That is, during exercise below the IT (i.e., “moderate” intensity exercise where cellular pH remains close to baseline), the dynamics of the muscle phosphates (PCr, Pi/PCr, and ADP) are strikingly similar between children and adults and between sexes. However, during exercise above the IT (i.e., “high” intensity exercise where cellular pH fall below baseline) children, and men compared with women, are characterized with a lower perturbation of the muscle phosphates and pH, suggestive of a greater oxidative contribution during exercise. These findings are generally consistent across both incremental (2, 5) and square-wave (1, 4) exercise protocols, and highlight the importance of exercise intensity when studying developmental exercise metabolism. We believe such findings are, in part, related to different muscle recruitment patterns during the transition from moderate to high-intensity exercise, such that younger individuals recruit fewer higher-order type II muscle fibers.

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AEROBIC DECONDITIONING AND GAIN IN MUSCLE MASS DURING PUBERTY RATHER THAN IMMATURE MUSCULAR GLYCOLYTIC ACTIVITY PRE PUBERTY

TO THE EDITOR: Children’s play is dominantly interval-sprint-like with activity bursts up to 15-s duration (1). Despite a decreasing volume of daily physical activity (4) maturation increases muscle mass and maximum short-term power substantially during puberty (5). Corresponding effects on anaerobic enzymes and maximum short-term performance related to muscle mass appear equivocal (2,5), but ratios of oxidative to anaerobic enzymes decrease (5), and the dynamics of oxygen uptake slows down (5). Although more or less unchanged if related to body mass, maximum oxygen uptake related to muscle mass decreases with increasing relative muscle mass during puberty. Also the relative body water content decreases (2). In children and adolescents the reliance on carbohydrate oxidation measured via indirect calorimetry is identically described as a function of the blood lactate concentration (BLC) up to an exercise intensity at which aerobic metabolism is almost fully reliant on carbohydrate oxidation (3). Above that exercise intensity the BLC is higher in adolescents than in children (3). Modeling maximum short-term exercise reveals that differences in performance and BLC reflect changes in relative muscle mass, relative water space, and oxygen uptake kinetics but no difference in glycolytic rate per unit muscle mass (2). All described changes in physical activity, performance, body composition, muscular enzymes, and dynamics of BLC and respiration are fully consistent with aerobic deconditioning combined with a maturation-related gain in muscle mass during puberty rather than a muscular metabolic specificity of immature glycolytic activity pre puberty.

REFERENCES

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