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GROWTH AND DEVELOPMENT AND ATHLETIC PERFORMANCE

RAST IN RAZVOJ TER USPEŠNOST V ŠPORTU

ABSTRACT

Children follow a predictable pattern of growth and development that varies in timing and magnitude but is constant in the achievement of developmental landmarks. This predictability allows athletic performance at any age to be statistically controlled for level of maturity and for future size associated athletic performance values to be estimated. The pattern of linear growth experienced by all children is characterised by rapid growth during infancy, relatively constant growth during childhood, and then accelerated growth during the adolescent growth spurt prior to reaching adult maturity and the cessation of growth in length. Whilst this is a universal pattern it demonstrates a considerable degree of sexual dimorphism generally favouring early growth and maturation in girls and delay in boys. That delay in boys accounts for an extended period of growth leading to greater adult size.

The pattern of linear growth is also not common in all tissues. In particular, soft tissues that are associated with fine and gross motor skills and coordination, cognitive ability, strength, endurance, and other aspects of athletic performance follow patterns that may be associated with but are less predictable than growth in length. For example, increase in size of the central nervous system (brain and spinal cord) demonstrates rapid change during foetal and infant periods and relatively slow growth from mid-childhood onwards, and no adolescent growth spurt in size. However, neural networks continue to increase in complexity throughout childhood and adolescence. In addition, the growth of some tissues, e.g., skeletal muscle and adipose tissue, also demonstrate significant sexual dimorphism resulting in characteristic android and gynoid phenotypes in adulthood and significant differences in athletic ability.

The development of athletic performance during childhood and adolescence will thus be strongly influenced not only by growth in size and increasing complexity of the nervous system but also by rates of maturational change and sexual dimorphism.

Key words: children, growth, development

IZVLEČEK

Pri otrocih rast in razvoj potekata po predvidljivem vzorcu, ki se razlikuje glede na čas in obseg, vendar pa je pri doseganju razvojnih mejnikov konstanten. Ta predvidljivost omogoča statistično spremljanje ravni zrelosti pri otroku katerekoli starosti ter oceno prihodnjih vrednosti športne uspešnosti glede na velikost. Za vzorec linearne rasti, ki ga imajo vsi otroci, je značilna hitra rast v zgodnjem obdobju otroštva, sorazmerno konstantna rast sredi otroštva in pospešena rast v obdobju adolescence pred odraslostjo, ko se rast v dolžino ustavi. Čeprav je to univerzalen vzorec, pa se v njem v precejšnji meri kaže tudi spolni dimorfizem, in sicer je zgodnjo rast in zrelost opaziti prej pri deklicah kot pri dečkih. To zamudo pri dečkih nadomesti podaljšano obdobje rasti, kar se kaže v večji višini telesa v odraslosti.

Vzorec linearne rasti pa ni prisoten v vseh tkivih. Zlasti pri mehkih tkivih, ki so povezana s fino- in grobomotoričnimi spretnostmi, koordinacijo, kognitivnimi sposobnostmi, močjo, vzdržljivostjo in drugimi vidiki športne uspešnosti, se pojavljajo vzorci, ki so lahko povezani z rastjo v dolžino, vendar so manj predvidljivi. Na primer, pri rasti osrednjega živčnega sistema (možgani in hrbtenjača) je vidna hitra sprememba v obdobju zarodka in prvih let življenja ter relativno počasna rast od sredine otroštva naprej, medtem ko v času adolescence ni poskoka v rasti. Vendar pa se živčevje še naprej razvija ter v otroštvu in adolescenci postaja vse kompleksnejše. Poleg tega rast nekaterih tkiv, npr. skeletno mišično in maščobno tkivo, tudi kaže na precejšni spolni dimorfizem, rezultat katerega so značilni androidni in ginoidni fenotipi v odraslosti ter pomembne razlike v športnih sposobnostih.

Na razvoj športne uspešnosti v otroštvu in adolescenci močno vplivajo ne samo vse večji obseg in kompleksnost živčnega sistema, ampak tudi spremembe v stopnji zrelosti in spolnem dimorfizmu.

Ključne besede: otroci, rast, razvoj

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The phrase "growth and development" is often used to describe the complete series of morphological and physiological changes that occur from conception to adulthood. However, the terms *"growth"* and *"development"* refer to very specific aspects of ontogeny; growth is based on a structural concept of increasing size over time whilst "development" or "maturation" is based on a functional concept of increasingly complex behavioural capabilities and actions over time. These capabilities are often only possible because of the changed structure, physiology, or biochemistry with which they are associated. For example, sexual reproduction is not possible without structurally mature secondary sexual organs and a physiologically mature hypothalamic-pituitary-gonadal axis triggering the release of sex steroids.

The US National Library of Medicine (2014) defines athletic performance as the "carrying out of specific physical routines or procedures by one who is trained or skilled in physical activity." The link between performance, training, and skill is central to understanding athletic ability. It is widely accepted that athletic performance in adulthood is influenced by a combination of morphological, physiological, psychological, and socio-cultural factors which in the child and adolescent change over time and are linked to its level of development or maturity.

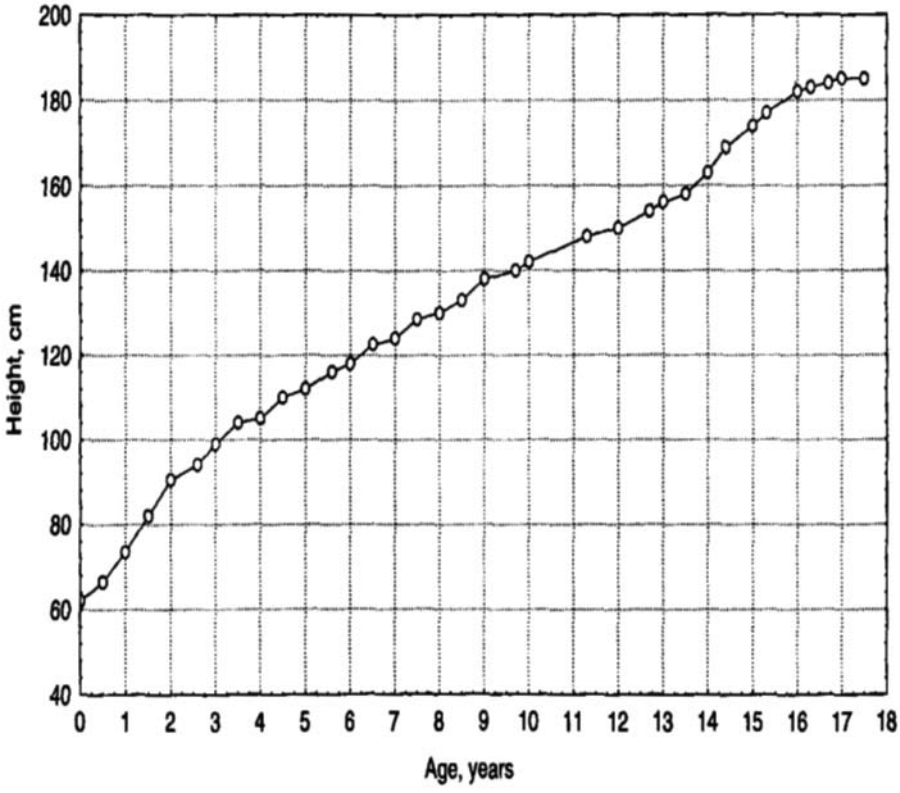

Figure 1: Growth in height of a normal boy

In order to understand the relationship between growth, development, and athletic performance it is necessary to identify and highlight the aspects of the body's structure and function from birth to adulthood that influence performance. (In the last 25 years considerable evidence has accrued

that highlights the importance of the pre-natal period to post-natal growth, development, and the acquisition of morbidity in adult life (e.g. Hanson & Gluckman 2014). There is little doubt that foetal growth and development may also be viewed as critical in relation to athletic performance during childhood and adolescence – particularly in relation to the attainment of critical levels of lean mass and fat distribution as a result of embryological and foetal development. However, at this time, little research has been published that documents the magnitude of these likely associations and we are left to speculate on probable associations.)

Children follow a predictable pattern of post-natal growth and development that varies in timing and magnitude but is constant in the achievement of developmental landmarks. Figure 1 illustrates the pattern of growth in height common to all normal children of both sexes and in all populations around the world. The pattern is characterised by rapid growth in infancy. In fact the *most* rapid growth in length occurs during intra-uterine life, between 20 and 30 weeks of gestation (Tanner, 1989), but thereafter height velocity gradually declines. That decline is interrupted twice; firstly by the juvenile or mid-growth spurt that occurs between six and eight years of age in both boys and girls, and secondly by the adolescent growth spurt (AGS). The AGS is initiated (the "take-off point") at about 10 years of age in girls and 12 years of age in boys living in industrialised nations, and at slightly older ages in children in developing countries (Eveleth &Tanner, 1990; Cameron, 2007). The AGS is characterised by rapid acceleration lasting one to two years prior to the attainment of a peak height velocity (PHV) at about 12 years in girls and 14 years in boys. The timing and magnitude of PHV is extremely variable and is affected by various factors including socio-economic status, nutrition, and ethnicity (Cameron, 1991). Following adolescence growth in height becomes asymptotic and adult height is said to be reached when the velocity is less than 1cm.yr^{-1}. Some sources (e.g. Roche, Wainer, &Thissen, 1975) use height at 18 years as "adult height" but this is inappropriate when considering the growth of children in developing countries who characteristically continue growing into their 20s. The primary landmarks of the growth curve are thus early rapid growth, the mid-growth spurt, the take-off point that initiates the AGS, and PHV linked with the post-natal phases of growth of infancy, from birth to about 3 years, childhood until take-off, and adolescence from take-off to adulthood. Bogin (1999), in a bio-cultural context, recognises a "juvenile" phase of growth between about 6 and 10 years of age characterised by social and behavioural independence and the ability to help in traditional parental duties with regard to the care of younger siblings and food preparation. The biological basis of the juvenile phase may be adrenarche or the maturation of the adrenal cortex and the release of adrenal androgens.

Growth in length (or weight) appears as a smooth curve but, if the frequency of assessment of post-natal growth is increased to daily or weekly measurements growth in length appears to correspond to a series of aperiodic saltatory episodes separated by periods of stasis in which no growth occurs (Lampl et al 1992). In a sample of 31 infants studied by Lampl et al (1992), periods of stasis lasted from 3 to 63 days and saltation occurred during periods as short as 24 hours in which the infants grew up to 2.5cm in length.

The pattern of growth that emerges as the frequency of assessment increases changes our approach to understanding the control mechanism. A continuous linear model suggests a mechanism that acts continuously perhaps with the constant release of a growth promoting hormone. A curvilinear model composed of two or three phases (infancy, childhood, and adolescence) suggests modifications of the controlling mechanism at certain important and perhaps critical periods, at the end of infancy and childhood. A model based on saltation and stasis implies a

discontinuous aperiodic control mechanism that underlies a more general mechanism occurring in three phases. One such aperiodic mechanism is found in the control of the cell cycle in which the cell leaves the cycle during G1 and becomes quiescent (stage G0).

Different tissues reflect different growth rates, perhaps because of different lengths of the quiescent G0 phase of the cell cycle. In the first five years of life, for instance, the nervous system grows rapidly to the extent that the brain reaches 95% of its adult size by about 7 years of age. Conversely tissues of the reproductive system, e.g., breasts and genitalia, do not demonstrate rapid growth until after 10 years of age. The tissues of the lymphatic system, e.g. thymus, grow rapidly in the first 10 years of life to achieve a size approximately 80% greater than they will be in adulthood but then recede during adolescence. These growth rates are in contrast to the curve of general linear growth that is represented by height with its clear childhood and adolescent components.

The juvenile growth spurt is not readily apparent in all tissues or measured dimensions but analysis of growth increments reveals clear mid-growth spurts in soft tissues such as muscle and subcutaneous fat. In both sexes the growth of muscle at least in this analysis of calf, appears to be about the same prior to adolescence although boys have a slight later and longer AGS in this dimension. Alternatively subcutaneous fat at the triceps and subscapular sites demonstrates a clear difference between the sexes; both sexes exhibit a mid-growth spurt in both skinfolds and an AGS for subscapular skinfolds but only girls exhibit an AGS in the triceps skinfold. This pattern of change in soft tissues coincides with a general increase in total body fat for girls during adolescence and particularly following the first menstrual cycle (menarche), and a centralisation of body fat for males. Thus the growth of adipose tissue, represented by subcutaneous fat, demonstrates clear sexual dimorphism. There is greater acquisition of fat in girls and the development of the classic gynoid distribution with fat accumulation in the gluteo-femoral region in contrast to the android abdominal distribution in boys. An index of body size and composition, such as Body Mass Index (BMI) exhibits yet another pattern of growth with a sharp increase in the first year followed by a reduction until a rebound, the "adipose rebound" (Rolland-Cachera et al, 1984) occurs during early to mid-childhood.

Given these different growth patterns it is not surprising that the organism exhibits variation in the proportions of body segments that change with advancing age. These proportional changes in size, caused by tissue specific growth rates (allometric growth), are most marked during foetal growth but are apparent throughout childhood and adolescence.

At the beginning of the foetal period (about 9 post-fertilization weeks) the head is 50% of total body length and the lower limbs make up approximately 20% of total length. By mid gestation (16 post-fertilization weeks) the head has reduced to about 30% of total length and the lower limbs have increased to also form about 30% of total length. These proportional changes or allometric growth continue throughout childhood and adolescence achieving adult proportions at the end of growth in length at about 18 years. The controlling mechanisms for allometric growth are far from clear although the pattern of growth of the limbs during pre-natal growth may well be affected by the unique nature of the foetal circulation diverting the most oxygenated blood to the head and brain whilst the least oxygenated blood passes through the *ductus arteriosus* to the descending aorta and lower limbs. The growth of limb segments has been well documented (Cameron, Tanner and Whitehouse, 1982) and demonstrates that distal segments preceded proximal segments in the age at peak velocity. Considerable individual differences, however, occurred in the order for the upper limb segments. These differences seemed to be related to

the individual's tempo of growth; late developers had a significantly different order to early developers. These findings may have significance for the performance of athletic tasks in which segmental length affects biomechanical outcomes in activities such as running and throwing. Indeed differences in sitting height-subischial ratios and limb proportionality were identified in Olympic athletes by Tanner (1961) as potential explanatory factors for the dominance of sprinters with African ancestry.

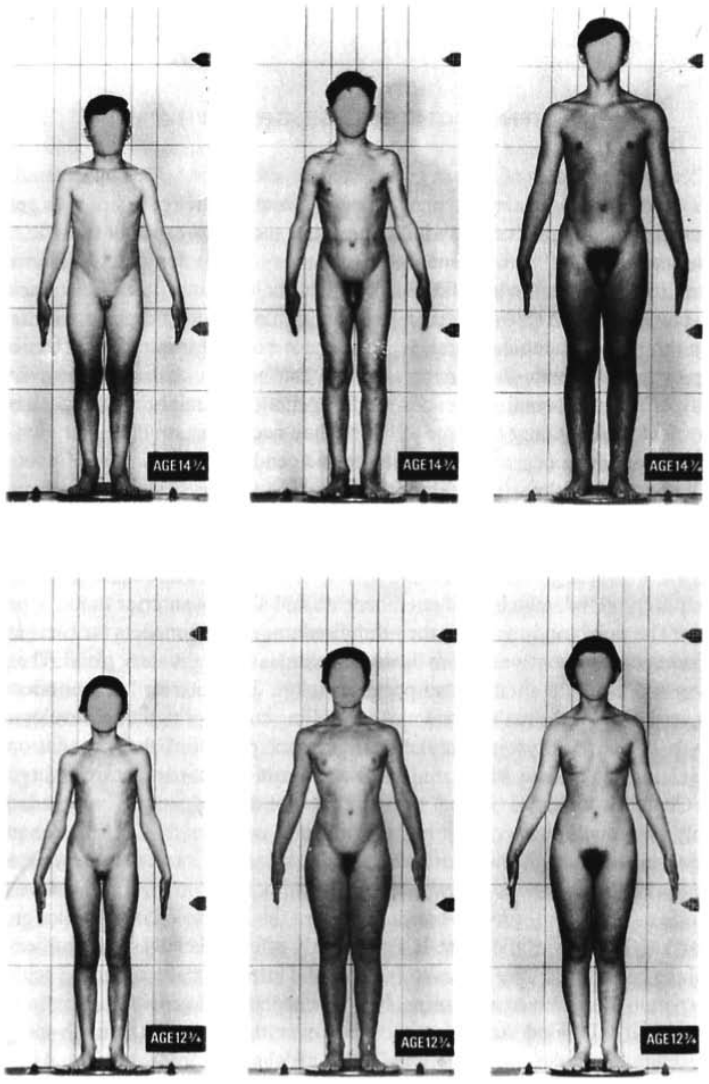

Figure 2: Three boys and three girls demonstrating variation in maturity during adolescence; the three boys are all aged 14.75 years and the three girls are all aged 12.75 years. (From Tanner JM. Growth and Endocrinology of the Adolescent. In: Gardner L. ed. Endocrine and genetic diseases of childhood. 2nd Ed. Philadelphia PA: WB Saunders.)

The adult phenotype is thus the result of increases in the size of a variety of tissues (bone, muscle, fat, CNS/PNS, etc.) as a result of varying rates of growth within (different times) and between (different rates) tissues. This structural outcome is intimately related to the underlying process of maturation.

Figure 2 shows three boys and three girls who are of the same ages within gender; the boys are exactly 14.75 years of age and the girls 12.75 years of age. The most striking feature of this illustration is that even though they are the same age they demonstrate vastly different degrees of maturity within the limits of normal variation. The average girl will enter the adolescent growth spurt at about 10 years of age, experience peak height velocity at 12 years, and reach adult height (a growth velocity of less than 1 cm.yr-1) at about 16 years. The average boy will be delayed by two years compared to the average girl, entering the growth spurt at 12 years, experiencing peak height velocity at 14 years, and reaching adult height at 18 years. The extra two years of pre-adolescent growth is the major reason for the average adult male being taller than the average adult female; two years of growth at about 5 cm.yr-1 result in about 10 cm of extra adult height and a slightly greater peak height velocity accounts for another 2 to 3 cm. The variability associated with this average pattern can be remarkable as Figure 2 demonstrates. It is not surprising that the individual's response to environmental factors affecting the process of growth is dependent on their level of maturity.

In addition to the variability within the sexes there is clearly variability between the sexes with girls in advance of boys. The two year difference in entry to the adolescent growth spurt is the result of a gradually increasing gap in maturity that has its roots in foetal growth evidenced by a two week advancement (Bogin, 1999). The difference between the sexes is a mark of sexual dimorphism which becomes more pronounced during adolescence as the functional requirements for successful reproduction become established. However, the male delay in age of entry to pubertal development is not as marked as that for somatic growth. Mean age at entry to puberty, usually marked by change in breast development in girls and genitalia development in boys differs by approximately 6 months (Marshall &Tanner, 1969, 1970). Comparison of the timing of the AGS with the timing of pubertal development demonstrates that the relationship between somatic growth and maturation differs between the sexes. Boys begin their pubertal development prior to the initiation of the adolescent growth spurt and appear to experience most secondary sexual changes relatively early in adolescence being sexually mature prior to the end of the AGS. Recent evidence also suggests that changes in boys voices (voice breaking) also occurs prior to PHV. Girls have a more synchronised start to both puberty and adolescent growth becoming sexually mature as they complete the AGS. The reasons for this degree of sexual dimorphism and the relationship between the different timing of puberty and adolescent growth between the sexes are mostly speculative. Clearly some advantage must result from this differential timing and that advantage must be related to the success of reproduction. Bogin (1999) has suggested that the early sexual maturity of late adolescent boys, that is not accompanied by the adult size and body composition of men, allows them to experiment sexually without representing a physical challenge to the adult males. Similarly the pubertal timing of adolescent girls results in simultaneous physical and sexual maturity and an increased attractiveness to slightly older, and more capable, male partners than relatively immature boys of similar age.

Sexual dimorphism in the timing and magnitude of secondary sexual development is accompanied by changes in skeletal dimensions and body composition that result in broader shoulders in males and greater total body fat content in females. In addition the distribution of fat becomes

classically android in boys and gynoid in girls. In the extreme this patterning is associated with increased risk factors for a variety of non-communicable diseases of lifestyle (e.g. obesity, CVD, CHD) which collectively are described as the metabolic syndrome.

So, how are these growth changes related to athletic performance? My proposal is that athletic performance is about function i.e. it is about speed, strength, endurance, coordination, and motor control. Successful function is based on efficiently and effectively utilising structure and that requires maturity to better decide when and how speed, strength, and endurance should be utilised. Thus structure and maturity precede function and it follows that effective athletic performance takes place when appropriate structure is present at a specific level of maturity.

It is not surprising therefore, that functional, physiological changes which directly impact on athletic performance are closely associated in timing and magnitude with these structural changes. Specifically during adolescence, the heart and lungs become bigger absolutely and in relation to body size, systolic blood pressure rises, heart rate slows, respiratory rate slows, blood haemoglobin increases, thus providing greater O_2 carrying capacity, and there is a more efficient neutralisation of chemical products of exercise e.g. lactic acid. These changes result in increased strength and endurance.

It is also not surprising that the level of maturity is strongly associated with athletic performance. Investigations of the relationship between maturity and athletic performance have consistently used maturity as a discrete variable to group participants into early, average, and late maturing groups. The reasons for this are clearly because of the dramatic differences in phenotype between maturity groups and the direct impact of phenotype on athletic ability.

In a recent review in the Research Quarterly of Exercise and Sport Malina posed the "Top 10 questions related to growth and maturation of relevance to physical activity, performance and fitness" (Malina, 2014). These were grouped into six areas of concern: exercise and growth, issues related to body weight, movement proficiency, individual differences, tracking of activity and fitness, and biological maturation. In addition to the unanswered questions of how much regular physical activity is essential to support normal growth and maturation, and whether physical activity affects the magnitude and tempo of human growth, Malina asked what is the impact of individual differences in biological maturation on physical fitness and performance during childhood and adolescence? Does maturity-associated variation (in fitness and performance) persist into adulthood? What is the influence of individual differences in biological maturation on physical activity?

Data is limited to answer any of these questions because the research design is logistically very difficult requiring longitudinal studies over many years synchronised with repeated standardised tests of fitness and performance and assessments of both habitual and programmed physical activity. However, the available data suggests that male athletes tend to be of advanced and average maturity status within an age group and female athletes tend to be of average and late maturity status within an age group. Late maturers may be at an advantage as adults with data from the Belgian longitudinal studies suggesting that early strength and motor performance differences between maturity groups are eliminated by 30 years of age in men (Lefevre et al, 1990) and that later maturation is associated with better muscular function at 45-49 years (Beunen et al, 2009).

The well documented "relative age effect" or "month of birth effect" that operates in the selection of young sports people, particularly footballers, demonstrates the selection bias for early developers (Fragoso et al. 2014; Hancock et al. 2013; Hirose, 2009). Recent research used a sample of Serbian youth soccer players (Ostojic et al. 2014) to determine the percentage of players from different maturational groups who achieved elite level of adult soccer competence. Competence was assessed by the percentage who eventually played for clubs competing in the top five international soccer leagues (La Liga, English Premier League, Bundesliga, Serie A, Ligue 1). Over 60% of late developers achieved elite level competence compared to only 10% of early developers and 40 % of average developers. The fallacy of selecting early developers at young ages on the basis of size (i.e. "the bigger they are the better they are") is demonstrated by this analysis.

Such is the complexity of the association between human growth and athletic performance that this review must leave many questions unanswered because the research community have not yet adequately addressed them. In his review Malina (2014) highlights many of these questions which, in my opinion could be addressed by appropriate longitudinal research designs.

In summary, this discussion presents the following points: the pattern of human growth and development varies in timing and magnitude; the achievement of developmental landmarks is universal and predictable; early and average developing boys and late and average developing girls perform better than late developing boys and early developing girls during childhood and adolescence; late developing boys may perform better as adults. The implications are that selection during childhood for potentially elite performance in adulthood must assess growth (i.e. size) in relation to maturity and skill and that future research ought to focus on identifying valid and reliable predictors of future performance controlling for current maturational status.

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