SOMATIC AND MOTOR DEVELOPMENT OF CYPRIOT ELEMENTARY SCOOLBOYS

PhD thesis

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Budapest, 2013

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1. INTRODUCTION

1.1.Health and its determinants

Defining health remains a major challenge, despite the progress made in treating diseases and increasing the average life duration in western societies. The World Health Organization (1948) described health as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity. Health is a human condition with physical, social and psychological dimensions, each characterized on a continuum with positive and negative poles. Positive health pertains to the capacity to enjoy life and to withstand challenges. Negative health pertains to morbidity, and in the extreme, with premature mortality (Bouchard and Shephard 1994, Paffenbarger at al. 2001). Naturally, the role of behavioural and environmental factors can not be neglected in this respect. By the statistics of Mokdad and associates (2004) poor diet and physical inactivity are the major behaviours associated with greater health risks.

There are two major (but not totally independent) challenges in the modern societies that influence the health status of the population. The first is general physical in-activity and the second is the epidemic of overweight and obesity. To determine causal relation in their interaction is almost impossible.

The lifestyle of most people in industrially developed countries is significantly influenced by mass media. Paradoxically, the mass media, and particularly television, can play an important role in the management of low level of habitual physical activity and obesity. A number of studies have confirmed that obesity is directly related to the number of hours spent watching television and other screens. The inactive behaviour of television viewing is often combined with the frequent, very attractive commercials advertising food and drink (Bar-Or 1998, James 2002, Parízková and Hills 2005). Unfortunately, the power of advertising is such that first of all young people and children are captive audiences and readily associate with the unfavourable practices that are presented, most commonly of highly processed and energy-dense foods. The other side of the problem is evolutionary. Evolution teaches us that those carrying genetic alleles favouring motor skills, strength, speed, stamina, and other physical attributes at relevant genes were more likely to have enjoyed greater reproductive fitness (condition) because

of their greater probability of attracting mates and staying alive long enough to have children (Bouchard at al. 2007). From the research of paleontologists, anthropologists, anatomists, and molecular biologists the main events in the evolution of our species can be briefly outlined with an emphasis on those that have implications for physical activity (Tattersall 2002) and the consequences are:

- The human organism can adapt to a wide range of metabolic demands imposed by work or exercise.
- A low level of physical activity is associated with risk for common diseases and premature death.
- Evolutionary history teaches us that early humans could not have survived without the ability to perform very demanding physical work.

And what are the facts nowadays? According to the indirect calculation of (Radák 2008) the daily energy expenditure utilized for physical activity decreased remarkably. This energy expenditure in a caveman can be assessed between 3.500-4.000 kcal in a day, and only 400 kcal in the modern man.

2. REVIEW OF RELATING LITERATURE

Introduction

Indicators of normal growth, maturation, performance and health status are affected by inheritance, direct biological regulatory factors and the wide range of environmental effects. Biological inheritance represents the influences of the parental generation on the offspring generation that are mediated by genes. The role of specific genes and mutations in normal variation in growth and development of children and adolescents remains to be elucidated, in part because of the measures of growth and development that are complex, multi-factorial phenotypes. The genetics of growth and maturation has been examined primarily through studies of different kinds of relatives, most often twins, siblings, parents and offspring, and spouses. The available indicate a major role for the genotype in body size and body composition and in the timing, tempo, and sequence of maturational events. The genotype also has a role in indicators of performance and responses to training, but genetic effects are not as strong as those for measures of growth and maturation. Much can be learned from the study of children who have a specific genetic deficiency. Such studies reveal that a partial or complete invalidation of a single gene can results in early death, failure to thrive, or a predisposition to several diseases. Advances in genomics and other technologies will make possible the investigation of the genetic and molecular basis of normal growth and maturation in the coming decades. We do not want to shrinkage the biological importance of genetics into this short paragraph but taking into account the strict length limitation of the thesis stress: The up –to-date details of the available genetic studies can be found among others in the summary publications of (Van Praagh 1998), (Bouchard and co-workers 2000), (Malina and associates 2005), (Parízková and Hills 2005). This chapter focuses on the hormonal regulation of growth and maturation, the effects of regular physical activity and finally we summarize the effects of some environmental factors.

2.1 Endocrine regulation of growth, maturation and performance

The integrated nature of growth and maturation is maintained by a constant interaction of genes, hormones, nutrients, and factors in the environment. Their separation can be made naturally, exclusively on the didactic points of view. Growth, maturation, and performance are unified processes, and factors that influence these processes are interrelated and interdependent. Many advances have occurred in the past two decades concerning and understanding of the biology of hormones and their mechanisms of actions. Even though the endocrine glands produce key hormones that play critical roles in the growth and maturation processes, hormone-like molecules are synthesized in other tissues and organs, and these substances contribute to growth and maturation as well. In conclusion, hormonal activities are essential for the maintenance of normal growth, the timing of maturation and also the physiologic proportions of active and non-active tissues (Wilson at al. 1998).

2.1.1 Growth hormone (GH)

The synthesis and secretion of GH by the anterior pituitary is regulated primarily by the hormones produced within the hypothalamus: growth hormone – releasing hormone (GHRH), which stimulates its production, and somatostatin or somatotropin release-inhibiting factor (SRIF), which inhibits its production. These two modulators of GH synthesis and secretion bind to specific cell surface receptors in the anterior pituitary somatotropic cells, activate the adenylate cyclase cascade, and increase cyclic AMP levels leading to stimulation or an inhibition of GH synthesis. Growth hormone is a peptide hormone of 191 amino acids. It circulates in the blood after its secretion in a free form or bound to a GH binding protein.

The levels of the binding protein are low at birth but increase rapidly throughout childhood (Grumbach and Styne 1998, Malina at al. 2005). The values of the binding protein are generally stable during adolescence. The levels are low in under-nourished children, high in overweight and obese children (Parízková and Hills 2005) and decline with weight reduction (Root 1994). Target tissue for GH harbour a specific cell surface GH receptor that bind the hormone to initiate cellular response.

Growth hormone influences protein, carbohydrate, and fat metabolism. It decreases the rate of carbohydrate uptake and utilization by tissues and enhances mobilization of lipids from adipose tissue depots. These effects are obviously important during growth but are also essential throughout life, especially for normal body composition and distribution of body fat. GH is essential for postnatal growth, which becomes quite evident early in life. The GH content of the pituitary increases progressively postnatal, reaching the maximum between 12 and 18 years of age. This level is maintained during adult life but decreases with aging.

The GH content of the anterior pituitary is not necessarily related to GH con-centration in circulation. GH is released in a pulsatile manner, that is, in a series of intermittent bursts during the course of the day. Children have slightly more bursts during 24 hours than adults, so the circulating level of GH during a day is greater in children than adults (Martha et al. 1989). The frequency and amplitude of GH pulses both vary with age, and the frequency and amplitude of bursts increases as individual passes from childhood through adolescence.

2.1.2 Insulin-like growth factor (IGF-1)

IGF-1 displays a wide range of metabolic effects. The prevailing component of IGF in the plasma is bound to a specific binding protein (BP). It is presumed that BP modulates the biological activities of IGF-1. BP is regulated by growth hormone (GH) and has a high affinity for IGF-1. Increased linear growth with a normal or high IGF-1 level is apparent in spite of low growth hormone secretion during the pre-pubertal period. In a study of children with simple obesity and normal children with shorter stature, it was revealed that peak levels of GH in the growth hormone releasing factor test were significantly lower than their obese pairs (Yasunaga 1998). The results of this study lead to the hypothesis that increased dietary intake and hyper-nutrition during growth cause hyperinsulinemia that increases GH receptor and IGF-1 secretion. IGF-1 has paracrine and autocrine effects near or at the site of synthesis. IGF-1 stimulates protein synthesis and increases cell proliferation in many tissues of the body. The growth factor IGF-1 is most active in cartilage. The net result is growth in length of individual bones, and in turn, stature, without accelerating skeletal maturation (Pette 2001).

Because GH is produced intermittently during the day, circulating levels vary. Circulating levels of IGF-1, on the other hand, tend to be reasonably stable during the day. Mean circulating concentrations of IGF-1 reach about 50 $ng \cdot ml^{-1}$ at birth but with considerable individual differences. They remain quite stable during infancy but rise gradually during childhood, attaining mean values of about 140 $ng \cdot ml^{-1}$ in boys and 170 $ng \cdot ml^{-1}$ in girls by 6 years of age. IGF-1 levels increases considerably during the adolescent growth spurt, reaching an average of 415 $ng \cdot ml^{-1}$ in girls 13 to 14 years of age and 430 $ng \cdot ml^{-1}$ in boys 15 to 16 years of age. The surge in IGF-1 during the adolescent growth spurt appears to parallel the increase in the number of bursts of GH at this time. Concentrations of IGF-1 increase as growth in stature accelerates. Peak levels of IGF-1 tend to occur about a year or so after peak height velocity and then decline slowly in late adolescence (Esoterix Inc 2000). Given the sex difference in the timing of adolescent events, the increase in circulating concentrations of IGF-1 occurs earlier in girls that in boys.

2.1.3 Thyroid hormones

The thyroid gland is located in front and either side of the trachea below the larynx. Weight of the gland increases gradually through infancy and childhood, followed by considerable enlargement during adolescence. It attains about 15 g in women and 20 g in men. Levels of thyrotropin and free thyroxine fall between birth and 1 month postnatally. Subsequently, blood levels of TSH remain rather stable until maturity. (Nelson at al. 1993, Esoterix 2000). In contrast, free thyroxine declines slightly through puberty and reaches a plateau at maturity. About 99% of the produced thyroxine and triiodothyronine are bound to several serum proteins, and the physiological action of thyroid hormones is mediated by nuclear receptors localized in the nucleus of cells of most tissues. These receptors are members of a super-family of nuclear receptors, and they bind more readily to triiodothyronine than thyroxine. The hormones enter into the nucleus, bind to the chromosome-linked receptors, and activate or suppress gene expression at specific genes.

Both thyroid hormones are calorigenic, that is, they stimulate oxygen uptake and energy expenditure. Hence, the major metabolic effect of thyroid hormones is a general increase in oxygen consumption in most tissues, especially those that compose the bulk

of body weight (skeletal muscle, heart, liver, and kidneys). They increase mitochondrial oxidative metabolism, which is partly explained by the augmented activity of Na/K ATP-ase. In contrast, no acute effect of the thyroid hormones is apparently produced in several tissues, including the brain and smooth muscles of the gastrointestinal tract (Vuori 2010). Much of the knowledge of specific growth and maturation effects of the thyroid gland is derived from clinical observations of individuals with abnormal thyroid function. Hypothyroid children, those with thyroid hormone deficiency, typically present growth failure (Cameron 2003). The rate of growth is reduced, linear growth of bones is impaired, infantile body proportions persists, skeletal and sexual maturation are delayed, and muscular development is deficient (Borsos 2000, Cameron 2006). If thyroid deficiency is severe and persists, somatic, sexual and mental retardation may result. Hypothyroid children, those with an excess of thyroid hormones, often show excessive growth initially, but loss of weight eventually occurs because of increased metabolic demands and resulting inadequacy of energy intake to meet the increased metabolic demand.

The single most frequent cause of hypothyroidism worldwide is iodine deficiency. However, iodine deficiency is very rare in the economically developed countries where iodine is routinely added to food products. Other causes of hypothyroidism include chronic autoimmune thyroiditis, congenital defects, and others. Hypothyroidism is occasionally inherited as an autosomal recessive trait (Tishkoff and Kidd 2004). The clinical manifestations of hypothyroidism are quite heterogeneous. If present in the first month of life, it leads to irreversible mental retardation and stunted physical growth (Vasan at al. 2005). The prevalence of hypothyroidism in new-born babies ranges from about 1 in 3,000 to 4,000 in the United States and Canada (Malina at al. 2005) and remarkably greater in many Asian countries. If treated immediately after neonatal detection, infants grow and develop normally.

2.1.4 Parathyroid hormones

The parathyroid glands are small bodies, usually four, located on the dorsal portion of the thyroid gland. On average, each parathyroid gland weighs about 40 g. However, a small percentage of the population has only three glands, whereas as much as 15 % of the population has five or six. The parathyroid glands do not seem to be under the

influence of a trophic hormone from the anterior pituitary. Parathyroid hormone (PTH) is an essential element in the regulation of calcium and phosphate metabolism. Thus, it is needed throughout life and is especially vital for the normal bone and dental growth and maturation. Because 99% of the body calcium is in bone, PTH is intimately involved in skeletal growth (Christiansen 2001).

Parathyroid hormone functions to maintain a reasonable stable plasma calcium concentration. This level, which is found within narrow limits, must be maintained despite variable calcium intake and excretion and calcium requirements for bone growth and maturation. The primary action of PTH is to stimulate an increase in circulating calcium through direct action of the hormone on bone and kidneys. PTH stimulates osteoclastic activity and thus bone resorption in the short term. However, long-term exposure to high levels of PTH causes a decrease in osteoclastic activity and compensatory in-crease in osteoblasts (Ji 2001). PTH also reduces renal clearance rates of calcium and increases intestinal absorption of calcium.

Calcitonin from the thyroid gland has effects opposite to those of PTH. Thyroid cells that produce calcitonin have a different embryonic origin than other thyroid cells (Malina at al. 2005). Calcitonin causes a decrease in circulating calcium and is secreted by the thyroid in response to a high plasma calcium concentration to either inhibit bone resorption or increase the rate of calcium deposition in bone (Rosenfield 2002).

Calcitonin and PTH function as a dual hormonal feedback system in the maintenance of plasma calcium concentrations. As calcium levels in body fluids become too high, calcitonin is secreted and calcium is thus lowered. When calcium levels are too low, PTH is secreted and blood and extra-cellular fluid calcium are elevated. Because the two hormones function to regulate the amount of circulating calcium and the ex-change of circulating calcium with calcium in bone, both PTH and calcitonin can be reasonably assumed to be essential to normal skeletal growth and maturation (Rowland 2005).

An important component of the pathway regulating calcium levels is vitamin D. Vitamin D is synthesized in the skin by the action of sunlight from the sterol precursor or is consumed in the diet. The product is vitamin D_3 (cholecalciferol), which is then metabolized into 25 (OH) D in the liver and subsequently into 1 α 25 (OH) $_2$ D_3 and

24R, 25 (OH) $_2$ D_3 in the kidney (Sperling 2002). The latter two compounds are considered steroid hormones, and both are necessary to observe the full spectrum of the biological effects of vitamin D. However, 1 α 25 (OH) $_2$ D_3 plays a particularly important role in the regulation of calcium levels. It increases the absorption of calcium in the small intestine and the rate of release of calcium and phosphate from bone. The rate of production of 1 α 25 (OH) $_2$ D_3 by the kidney is influenced by several hormones, but PTH and calcitonin are the most important, particularly in the long-term regulation of plasma calcium concentration. Short-term control is driven primarily by parathyroid hormone and normal levels of 1 α 25 (OH) $_2$ D_3 (1, 25-dihydroxycholecalciferol) (Kulin and Muller 1996, Rowland 2005).

2.1.5 Regulatory effects of adrenal medulla and cortex

The adrenal glands are small organs located over the upper pole of each kidney. Each weighs about 4 g and is composed of two distinct glands:

- An outer gland, the adrenal cortex (about 90% of the size of the adrenals).
- An inner gland, the adrenal medulla.

The growth pattern of the adrenals is characterised by a marked decrease (!) in weight shortly after birth and a further gradual reduction during the first 3 to 6 months of life. Weight of the glands then rises through childhood and shows growth spurt during adolescence (Malina at al. 2005). The size of the gland at birth is thus regained during the second decade. This growth pattern is one of the exceptions to Scammon's curves (1930) of systemic growth.

The adrenal medulla is innervated by the sympathetic nervous system and secretes adrenaline in response to nervous stimulation. Noradrenaline is mainly released from sympathetic nerve endings and converted enzymatically to adrenaline primarily in the adrenal medulla. Catecholamines exert a myriad of biological influences on tissues and organs and regulate levels of a number of molecules. For instance, catecholamines influence the force and rate of contraction of the cardiac muscle, systolic and diastolic blood pressure, gastrointestinal motility, bronchodilatation, insulin secretion, adipose tissue lipolysis, glucose metabolism, fatty acid metabolism, and thermogenesis. Additionally, adequate regulation of catecholamine production and also degradation and

of epinephrine and nor-epinephrine metabolism is important for normal growth and maturation (Molitch 1995, Veldhuis JD 2002).

Hormones of the adrenal cortex, on the other hand, are directly involved in the regulation of growth and maturation, in addition to being essential to many other body functions. The adrenal cortex produces and secretes steroid hormones.

Adrenal cortical steroids influence a variety of body functions and are thus essential to growth and maturation. Because the gonads and the adrenal cortex have a common embryonic origin, one might expect an interaction between the two glands. Indeed, a normally functioning adrenal cortex is necessary for complete sexual and reproductive maturity. Excess secretion of adrenal cortex hormones, or pharmacologic doses of glucocorticoids for systemic inflammation during childhood, can result in stunted growth in stature as the proliferation of cartilage cells at the growth plates is decreased. This condition is apparently caused by increased protein catabolism in bone and elsewhere in the body. The net result is the stunting of linear growth and also growth in weight (Eliakim at al. 2000, Malina at al. 2005).

Adrenarche, the increase in adrenal androgen and oestrogen production, occurs about 2 years before gonadarche, the onset of gonad functions marked by increase in the size of external genitalia and breasts. However, adrenarche is not essential for gonadarche to take place, and they are almost independent events (Cutler at al. 1990). The role of adrenal androgens and oestrogens in pubertal events is not yet clear, but gonadal steroids play the predominant role. Secretion of the main glucocorticoid, cortisol increases gradually with age during growth, and the increase is proportional to the age-associated increase in body size, except perhaps in infancy. How-ever, the relationships between serum cortisol levels or circadian cortisol ranges and body mass, body composition, or growth rate in healthy children and adolescents are thought to be secondary to other factors and do not appear to be causal (Knutsson at al. 2000, Brownell at al. 2009). Nonetheless, when cortisol production rates are related to maturity status in adolescent boys, the rates increase, on the average, with advancing sexual maturity. However, when cortisol production is adjusted for body weight in adolescents of contrasting maturity status the differences in cortisol production across maturity categories are reduced considerably (Beunen at al. 2002).

2.1.6 Hormones of gonad glands

Gonadotropins

The testes and ovaries are, respectively, the male and female gonadal glands. According to the early observation of (Boyd 1952) age-related change in the weight of the testes is periodical and almost linear in respect of the ovaries during growth. The testes and ovaries each have two functional aspects, a hormone-secreting component and a gamete-producing component. The hormone-producing and gamete-producing functions of the gonads are regulated by two gonadotropic hormones from the anterior pituitary: FSH and LH. Follicle-stimulating hormone in females stimulates the growth of ovarian follicles but not their complete maturation and oestrogen secretion. Luteinising hormone promotes maturation of an ovarian follicle, ovulation, development of corpus luteum, and stimulation of further production of oestrogens by the ovary. In males, FSH promotes growth of the seminiferous tubules and stimulates the production of sperm, and LH stimulates the intestinal cells of Leydig to enlarge and produce testosterone.

Changes in serum concentrations of FSH and LH with chronological age and maturity status are significant. Blood levels of FSH are much higher in infant girls (7.2 $mIU \cdot ml^{-1}$) than in infant boys (1.5 $mIU \cdot ml^{-1}$). During childhood, values stabilise approximately at 2.0 $mIU \cdot ml^{-1}$ in girls and 1.0 $mIU \cdot ml^{-1}$ in boys. Blood levels of the gonadotropins increase with puberty in both sexes. Blood levels of LH are quite variable be-tween birth and 1 year of age with no consistent difference between boys and girls (Esoterix Inc 2000). From about 1-1.5 year of age to the onset of puberty, levels of LH are about 0.07 $mIU \cdot ml^{-1}$ in both sexes. With the onset of puberty (stage 2 breasts and pubic hair in girls and stage 2 of genitals and pubic hair in boys), blood values of LH increase about 10-fold in girls and 20-fold in boys. LH levels then continue to rise in girls such that by early adulthood, they reach about 4 to 5 $mIU \cdot ml^{-1}$. The rise in boys is more gradual, but adult levels are also, on average, about 4 to 5 $mIU \cdot ml^{-1}$ (Malina at al. 2005). Thus, gonadotropic hormones are detectable in the blood of pre-pubertal children before overt signs of sexual maturation become apparent. Childhood concentrations are rather stable, with much overlap between girls and boys. From about 9 or 10 years of age, blood levels of gonadotropins rise in both sexes. Blood concentrations are slightly more related to stage of sexual maturity than to chronological age. Late in puberty in girls and

just before menarche, the gonadotropic hormones develop a cyclical pattern related to menstrual cycle.

Sex steroids

In males, testosterone is synthesised in the testes and adrenal cortex from cholesterol precursors. Although testosterone is the major and most abundant androgen in males, the most potent androgen is dihydro-testosterone, which is derived from an enzymatic conversion of testosterone. Dihydro-testosterone has about three times more androgenic activities than testosterone. Dihydro-testosterone appears to be formed from the testosterone precursor mainly in tissues that are sensitive to circulating androgens. Di-hydro-testosterone is thought to be the major growth-promoting androgen necessary for the maturation of male secondary sex characteristics. The rate of enzymatic conversion of testosterone to dihydro-testosterone is related to body size and probably to muscle and adipose tissue masses (Malina at al. 2005, Foley at al. 2007, Jimenez Pavon at al. 2010, Metcalf BS 2009).

In females, estradiol is the most potent oestrogen, and it is produced by the ovaries. Small amounts originate from the peripheral conversion of precursors secreted by the adrenal cortex. In addition to estradiol, other oestrogenic hormones are produced. These hormones are biologically less potent, but, nonetheless, contribute to the sexual maturation of girls. Most of the other oestrogenic hormones result from the peripheral conversion of ovarian and adrenal oestrogenic precursors. Testosterone and other androgens are also present in females. Androgen precursors are secreted in small amounts by both the ovaries and adrenal cortex. Both testosterone and dihydro-testosterone are found in the blood and peripheral tissues of females but in small quantities competed with males. Most studies that consider steroid hormones in growing children use testosterone and estradiol as the markers of androgenic and oestrogenic activity, respectively. Ideally they should be measured repeatedly over several days under standardised conditions because blood concentrations of the hormones can be influenced by several factors (Malina at al. 2005, McLure SA 2009). However, such data are not available from birth to maturity. Recent studies have shown that testosterone administration increases whole-body protein metabolism in pre-pubertal boys in vivo isotopic experiments (Frost HM 1997). The anabolic activity was accompanied by elevated GH, IGF-I, and testosterone levels. In contrast, chronic

exposure to oestrogens in hypo-gonadal girls did not affect whole-body protein metabolism despite an increase in plasma IGF-I concentrations (Li C Basarab at al. 2004, Maj A at al. 2008). The absence of an anabolic effect of oestrogens may contribute to the sex differences in body size that arise during puberty and the adolescent growth spurt.

Effects of the steroids

The effects of androgens and oestrogens on growth and maturation are many. The principal sites of action for testosterone and dihydro-testosterone are the primary and secondary sex characteristics of males. The oestrogens act to bring about corresponding changes in females, including the growth and maturation of the primary and secondary sex characteristics and the gynoid profile of adipose tissue distribution.

Both androgens and oestrogens promote generalised nitrogen retention and thus increased anabolism. Androgens are more potent in this regard than oestrogens. The effects of testosterones, for example, specifically underlie the dramatic adolescent growth spurt in muscle and fat-free mass in males. The protein anabolic actions of oestrogens are less than those of androgens, so the female gain in muscle mass during adolescence is primarily an effect of adrenal androgens.

Androgens also promote bone growth and skeletal maturation. They stimulate some longitudinal growth of long bones in interaction with GH and IGF-I and also promote considerable growth in bone thickness. The greater skeletal growth of boys than girls is related to testosterone secretion. Protein retention enhanced by androgens promotes the formation of cartilage and bone matrix and the deposition of calcium and phosphorus. The effects of oestrogens on bone are generally similar to those of androgens, except for their influence on linear growth. Oestrogens increase bone matrix formation, maintain positive calcium balance, and accelerate skeletal maturation. More-over, oestrogens are the main steroids involved in the final phase of skeletal maturation in the sense that they initiate and complete epiphyseal closure (Smith and Korach 1996, Armstrong and van Mechelen 2000). Thus, oestrogens can be viewed as a primary determinant of final stature of a child. Androgens also contribute to this process as they are aromatised to oestrogens in tissues (Augat 1998).

Oestrogens promote the accumulation of fat throughout the body in females and specifically enhance the accumulation of fat about the hips, buttocks, breasts, and medial aspects of the calf. The rise in concentrations of dehydroepiandrosterone, an adrenal steroid, in boys and girls between 7 and 12 years of age, is perhaps related to the accumulation of fat in both sexes at this time (the so-called pre-adolescent fat wave). As the process of sexual maturation and the adolescent growth spurt continues, males experience a fat loss on the extremities. This loss may be related to increasing androgen out-put, especially testosterone, which has a fat-mobilising effect, and the hormones probably act in concert during male adolescence (Reiter and Grumbach 1982).

The increasing concentrations of the sex steroids during puberty influence the production of other hormones related to growth, specifically GH and IGF-I. The rise in sex steroids at this time leads to an increase in the secretion of GH, which in turn stimulates the production of IGF-I. This effect is especially pronounced in boys compared with the corresponding effect in girls. The interactions between the gonadal steroids and growth hormones become especially apparent at the time of puberty (Kerrigan and Rogol 1992). Thus, some of the sex differences in growth and body composition during adolescence are related to the increased secretion of GH and IGF-I in males consequent to the increased production of sex steroids at this time (Klein KO at al. 1998, Yilmaz D at al. 2005, Huang KC at al. 2004).

2.1.7 Insulin and glucagon

The hormonal secretions of the Islets of Langerhans are insulin (secreted by the β cells), glucagon (secreted by the α cells), somatostatin (produced by the δ cells) and the pancreatic polypeptide (produced by the PP cells). Insulin and glucagon are the primary interest. The actions of the two hormones are mutually antagonistic. Insulin is a blood sugar-lowering hormone, whereas glucagon is a blood sugar-raising hormone.

Insulin is essential in carbohydrate metabolism. It enhances the rate of glucose uptake in skeletal muscle, adipose cells, and other tissues stimulating the transport of glucose and amino acids through cell membranes. The action of insulin decreases the concentration of blood glucose and increases glycogen stores in skeletal muscle and liver and the reliance on glucose as a substrate to meet the cellular energy needs. If blood glucose is

excessive and glycogen stores are high, insulin may stimulate the transformation of glucose into fatty acids, which are in turn converted to triglycerides. The latter is, however, a minor pathway in humans under common dietary circumstances. Insulin can inhibit glucose production by the liver and block fatty acid mobilisation through inhibition of adipose tissue lipolysis. The insulin-mediated disposal of blood glucose is thus particularly important in the maintenance of glucose homeostasis and overall substrate balance (Martin F at al. 1996, Lang J at al. 1997). Glucagon has the opposite effect of insulin.

Because insulin acts primarily on carbohydrate metabolism, it is important to normal growth and maturation. Insulin and GH interact in complex manner, and insulin is essential for full expression of the effects of GH. Although insulin is capable of promoting protein synthesis in the absence of GH, GH has only a slight effect on protein synthesis in the absence of insulin. The effects of GH, IGF-I, and IGF-II on protein synthesis and, in turn, on growth are considerably greater in the presence of insulin. In contrast, insulin and GH have opposite effects on fat. The former stimulates the conversion of carbohydrates into fat and depresses lipolysis, whereas the latter stimulates the mobilisation of fat (Bommert K at al. 1993, Takahashi M at al. 1991).

The δ cells of the pancreas produce somatostatin, which is also secreted by hypothalamic nuclei and is a potent inhibitor of GH-releasing hormone and, in turn, of GH from the anterior pituitary. Somatostatin is also produced in other areas of the brain, in the stomach, and in the gastrointestinal tract. Many issues concerning the various physiological roles of somatostatin are still unsolved, but it is a potent inhibitor of insulin and glucagon secretion from the islet cells. Thus, pancreatic somatostatin has paracrine effects on pancreatic α and β cells. Insulin is also a growth-promoting hormone. It is a potent mitogenic factor associated with cellular hyperplasia and cell hypertrophy. At least 100 genes are known to be regulated by insulin. Insulin, in some instances, increases the expression of specific genes and, in other cases, decreases the expression of genes. This role of insulin can be tissue specific and may be enhanced or diminished in the presence of other hormones or growth factors. Many cellular processes and substrates are affected by insulin, such as transcription of genes encoding metabolic enzymes, hormones, transcription factors, and others. In typical case, the

insulin-sensitive genomic sequence for a gene has an insulin response element in the promoter region. An insulin-stimulated change in the phosphorylation state or a receptor or other molecules or the presence of transcription and other factors may be required for the insulin effect (Wollheim CB at al. 1981, Sadoul K at al.1995, Borer 2003).

2.1.8 Leptin

Leptin is a hormone produced mainly by adipocytes. It is also expressed at much lower levels in the stomach and placenta. Initially, leptin was thought to be the missing factor that predisposed some people to be in positive energy balance. Indeed, leptin is an important regulator of long-term food consumption, and leptin appears not to play an important role in the short-term regulation of appetite and satiety. For instance, leptin levels are not altered by a single meal (Argente at al. 1997). Leptin exerts its effects on energy balance through the hypothalamic leptin receptor. Leptin reaches the central nervous system after having crossed the blood-brain barrier by a mechanism that remains to be elucidated. Leptin binds to the leptin receptors that are present at high levels in several hypothalamic neurons. Activated leptin receptors generate a response cascade whose long-term net effects are a reduction in food intake and an increase in metabolic rates. Leptin does not act in isolation to modulate energy balance. It interacts with key molecules involved in other regulatory loops that are components of highly redundant system. For instance, hypothalamic neuropeptide Y, a strong stimulant of appetite. A series of animal experiments have established that some of the actions of leptin on food intake are the results of its inhibitory effect on neuropeptide Y activity in the hypothalamus. α melanocyte-stimulating hormone has a potential to decrease appetite as a result of its binding to the melanocortin receptor 4 in the brain. Leptin increases the express-ion of proopiomelanocortin (POMC), the gene transcript that encodes several peptides, including α melanocyte-stimulating hormone. In other words, leptin potentiates the action of a number of anorexigenic factors and antagonises the action of orexigenic agents (Augustine RA and Grattan DR 2008).

Leptin is also thought to be involved in the regulation of metabolic rate even though the mechanisms are still unknown. Administration of leptin to animals undergoing caloric restriction attenuates the fall in metabolic rate commonly observed with negative energy balance (Sánchez J at al. 2009). Leptin also favourable influences glucose and lipid

metabolism. Overall, leptin is a potent molecule that influences many functions and is thus necessary for normal growth and maturation.

The critical role of leptin in growth is well illustrated by the clinical picture of the few patients who have been found to have inactivating mutations in the leptin or leptin receptor gene. Two kindreds with patients homozygous for leptin deficiency resulting in a complete absence of leptin have been described (Schwartz at al. 1996, Zlokovic at al. 2000). The patients exhibit severe obesity, increased food intake, and hypogonadotripic hypogonadism. A mutation in the leptin receptor gene leads not only to the same features as in the leptin deficient patients but also to growth retardation and hypothyroidism (Clement at al. 1998). Normal leptin and leptin receptor genes and normal leptin levels are, therefore, necessary for somatic growth.

After the discovery of leptin, researchers soon realised that a main function of leptin could be to protect body fat stores against severe depletion. During caloric restriction or times of under-nutrition, the adipose mass progressively decreases, which leads to a diminution of leptin production and leptin release from the adipose organ. Low leptin levels tend to reset food intake at a higher level and keep the metabolic rate in check to protect energy stores. Such a system confers clear evolutionary advantages and has implications for growth and maturation in children living under impoverished conditions.

Leptin is also required for the normal development of reproductive function and probably also for the onset of pubertal events. The mice characterised by the absence of leptin because of an autosomal recessive mutation in the OB gene are infertile. When treated with recombinant leptin, these mice become fertile. Moreover, treating normal young mice with repeated injections of recombinant leptin hastens the first signs of puberty. These observations together with the clinical profile of the adult patients with leptin or leptin receptor deficiencies strongly suggest that normal leptin levels play import-ant role in the onset of puberty (Elmquist at al. 1998).

Leptin is also involved in the regulation of hypothalamic-pituitary-adrenal axis. It is inversely related to levels of ACTH and cortisol. For instance, leptin deficiency is associated with high blood glucocorticoid levels and increased risk of type 2 diabetes.

Leptin enhances hematopoiesis and immune functions and undoubtedly impacts other pathways and functions as well.

2.2 The effects of regular physical activity

Physical activity is an environmental factor that is often viewed as exerting a favourable influence on growth and maturation. Additional environmental factors include socioeconomic status of the family, illness history, nutritional status, family size, climate, and others. Physical activity should be recognised as only one of many environmental factors that may affect these processes. Current concern for the increasing prevalence of hypo-activity or sedentarism in children and adolescents (Tomkinson at al. 2003, Olds at al. 2004, Mészáros at al. 2009, Melzer at al. 2004) makes a discussion of the potential role of physical activity highly relevant.

Regular physical activity is often assumed to be important to normal growth and maturation. Studies spanning nearly a century have suggested that regular physical activity, including training for sport, has a stimulatory influence on somatic development of children and adolescents. In one of the comprehensive reviews of "exercise and growth" the following was suggested. *"There seems to be a little question that certain minima of muscular activity are essential for supporting normal growth and for maintaining the protoplasmic integrity of the tissues. What these minima mean in terms of intensity and duration of activity has not been ascertained"* (Rarick 1960). At the same time, concern was also expressed and is still currently expressed about potentially negative influences of physical activity, specifically of intensive training for sport during childhood and adolescence (Buckler and Brodie 1977, Drinkwater at al. 1984, Laron and Klinger 1989, Zsidegh at al. 2007). For the present, note that regular physical activity is not equivalent to intensive training for sport.

The mechanical and energetic aspects of activity are potentially important in the context of growth and maturity. Most discussions of physical activity refer to a child's estimated level of habitual physical activity, that is, the level of physical activity that characterises the lifestyle of the individual. It is usually quantified in terms of amount of time in activity (hours·week^{-1}), an activity score, or energy expended in light or moderate-to-vigorous activities (Malina at al. 2005). Estimates are ordinarily derived from

questionnaires, interviews, diaries, and heart rate integrators or combination of methods. Presently available techniques for estimating physical activity have measurement limitations (Vuori 2010).

Qualification and quantification of physical activity programs for children and adolescents are necessary. Describing a program as mild, moderate, or vigorous physical activity or describing children as active or inactive is insufficient. Physical activity needs to be defined in more specific terms if the effects of activity on growth, maturation and performance are to be identified and partitioned from other factors known to affect these outcomes. Partitioning requires more details about number of sessions per week, duration of activity sessions or distance covered in each session, intensity of the activity, type of activity, and perhaps estimated energy expenditure.

2.2.1 Activity and body height

Regular physical activity has no apparent effect on attained stature and the rate of growth in height. Longitudinal data on active and hypo-active boys followed from childhood through adolescence and girls followed during childhood show no difference or only small difference between the speeds and growth patterns (Beunen at al. 1992, Mészáros 2010). The issues of subjects selection, probably self-selection, in the active and hypo-active groups is a factor to consider in making comparisons. Although some early studies suggest an increase in stature with regular activity, however, the observed changes are usually quite small, and selection of subjects and maturity status at the time of training or at the time of making the comparisons was not controlled.

On the other hand, regular activity does not have a negative effect on growth in height. This finding is relevant because the short stature and a slower rate of growth of young athletes in some sports are accepted as evidence that training may stunt growth (Malina at al. 2005). In addition to completeness of the available data, several import-ant factors are not considered drawing such a conclusion – small size as a selection criterion in some sports, inter-individual variation in biological maturity, and parental size (a proxy for genetic potential), among others (Pápai 2002, Pápai at al. 2007).

2.2.2 Weight, body composition and physical activity

Differences between the body weights of active and hypo-active children are generally small and statistically not significant. The data vary among studies, for example, boys in the Canadian sample tend to be heavier than active boys, especially during adolescence (Mirwald and Bailey 1986, Beunen at al. 1992). Exactly the similar patterns were observed in Hungarian pre-pubertal boys (Mészáros 2011).

Components of body mass can be potentially influenced by regular activity. Presently available data are derived primarily from the two-component model (body weight = fat free mass + fat mass). Some data suggest that regular physical activity is associated with a decrease in absolute and relative fatness, and increase in fat free mass. Partitioning effect of training on fat free mass from expected changes associated with growth and maturation is difficult, specifically during adolescence. Both sexes have a significant adolescent growth spurt in fat free mass, but boys more so than girls (Buckler and Green 1999, Cederstrema at al. 2007).

The influence of regular physical activity on body fat content relative to mass is essentially apparent in several studies of obese children. For example, a daily program of aerobic activity for two years resulted in marked decrease in skinfold thickness of obese children (Sasaki at al. 1987). Short-term training programs show a significant decrease in estimated percept body fat in obese children 7 to 11 years of age (Gutin at al. 1997). The observed effects of 1-year aerobic physical activity were slightly moderate in the 7 to 8-year-old Hungarian boys (Vajda at al. 2007a, b). The summary effect of the used programs was that depot fat did not increase in study group, in contrast to the controls in which the sum of the skinfolds, relative fat content and BMI increased significantly. The stabilisation of the obesity markers was interpreted as a positive sign, but the authors stressed the increased level of activity was less effective in this respect than the combination of physical activity and diet. It is important to note, the effects of increased physical activity were apparently more manifest in the changes of the physiological variables. Both absolute and relative aerobic power, minute ventilation, oxygen pulse and running performance were characteristically better in the active group. It is an additional information, even the peak physiological performances measured at the end of the program in study group were very moderate.

Data comparing the body composition of active and non-active girls during childhood and adolescence, or comparing changes in body composition associated with a program of regular physical activity are limited. Morris and associates (1997) com-pared the body composition of two groups of 9-year-old and 10-year-old girls, on that followed a 10-month training program and another that followed a normal pattern of physical activity. Girls in the two groups were similar in age, height, weight, body com-position, and stage of sexual maturity at the start of the study. The training program included 30 minutes of high-impact aerobic and strength training activities three times per week. After 10 months, the trained girls had a greater gain in estimated lean mass (2.2± 1.1 kg) and smaller increase in fat mass (0.5±0.8 kg) than the girls who followed their normal pattern of physical activity (1.4±1.4 kg lean mass and 1.0±0.8 kg fat mass). Note that both groups gained in lean and fat masses over 10 months on the average, fatness did not decrease. Also considerable overlap existed between the trained and control girls, but individual differences among the subjects after 10 months were, unfortunately, not reported. Although the results are suggestive, they indicate the difficulties inherent in attempting to partition growth-related from training-related changes in estimated body composition. The question of sex differences in the responses of fat free mass and fat mass to regular programs of physical activity during growth needs further study. Evidence for young adults indicate a significant decline in percentage fat and subcutaneous fat and an increase in fat free mass in males but not in females after 15 weeks of high-intensity training on a cycle ergometer (Tremblay at al. 1988)

2.3 Relationships of anthropometric characteristics and performance

The period between 5 and 8 years of age appears to be a transitional period in the development of strength and motor performance. Basic motor skills are reaching mature form at this time, but there is considerable variation among children. In addition, the application of these skills and techniques to specific test situations must be practised or learned. Therefore, this variation in the performance of specific strength and motor items at these ages is expected. Muscular endurance appears to show an increased pace of development is boys but not in girls. This difference may, in part, reflect a learning effect as the youngsters get adjusted to the test situation. Controlling for learning effects

is a significant logistic problem in longitudinal studies of motor performance: How much of the observed improvement effects growth-related changes and how much reflects learning to perform the tasks (Seger and Thorstensson 2000, American Academy of Pediatrics 2001, Huang and Malina 2002).

Changes in body size, physique, and body composition associated with growth and maturation are important factors that effect strength and motor performance. The relationship, however, vary among performance measures and with age. Relationships between size, physique, and body composition and performance are commonly viewed as correlations that statistically express the relationship between two variables. Correlations do not indicate a cause-and-effect sequence of events. Mediating or moderating variables, as well as covariates, may be present. Correlations less than 0.30 are considered low, and those between 0.30 and 0.60 are moderate.

Correlations between height and weight and performance on a variety of motor tasks are generally low to moderate in children and adolescents 4 to 18 years of age and are of limited predictive utility. Correlations do not differ by sex. Tasks in which body is projected (dashes and jumps) generally show negative correlations with body mass. Tasks in which the body is raised (pull-ups) or supported (flexed-arm hang) off the ground by the arms consistently have a negative correlations with body weight, and some reach into the moderate range. In contrast, correlations between stature and weight and a variety of strength tasks are higher than those for motor performance and generally fall in the moderate range. Thus, the taller and heavier individual tends to be stronger (Malina at al. 2005). Relationship between body size and performance are confounded in part by age. Age, height, and weight are related, so it is necessary to control for relationships among these variables when evaluating their specific contributions to variation in performance. With this procedure, the relationship between height and performance can be evaluated while statistically controlling for age and weight. The same can be done for weight, controlling for age and height, and for age, controlling for height and mass. This procedure is called partial correlation. Partial correlations for age, height, and weight and variety of motor performances do not differ between boys and girls and several trends are apparent (Malina 1994). Age is positively related to strength and motor performance even when stature and mass are controlled.

This positive relation suggests an important role of neuromuscular maturation and experience in performance on muscular strength and motor tasks. After controlling for age and stature, body mass tends to have a negative influence on performance, especially in tasks in which the body is projected, whereas after controlling for age and body weight, correlations between stature and performance tend to be positive (van Mechelen and Kemper 1995). Body dimensions are related to age, height, and weight. A question of interest is the contribution of specific body dimensions to strength and motor performance after the effects of age, stature, and weight are statistically controlled. Results of several such analyses in children from different health and nutritional backgrounds indicate that few variables add significantly to describing variation in strength and motor performance after age, stature, and mass are statistically controlled. Subcutaneous fatness, however, tends to exert a negative influence on performance (Benefice at al. 1999, Ozsváth at al. 2009). Age and also body size therefore appear to be primary factors influencing the strength and motor performance of children. Data for adolescents are a bit more complicated because individual differences in biological maturity associated with the adolescent growth spurt and sexual maturation must be taken into account (Iuliano Burns at al. 2001).

2.4 Effects of other environmental factors

The conditions into which children are born and subsequently reared can influence growth and maturation. These conditions include quality of living conditions, family size or number of siblings, place of residence (urban or rural), and overall socio-economic circumstances. All are related, and accounting for the effects of specific social factors is difficult.

General living conditions associated with socioeconomic status (SES) include variation in educational background of parents, purchasing power for food and, in turn, nutritional status, access to and use of health-care facilities and programs, and overall regularity of lifestyle. The SES of child's family is a significant factor that can affect growth and maturation. Criteria of SES vary considerably among studies and among different countries, so comparisons are difficult (Chumlea at al. 2003). Criteria relevant to one area, cultural group, or country are not necessarily relevant to other. Commonly used indicators of SES in developed, western countries include annual family income,

per capita income, occupation and education of the head of the household, and place of residence. Children from better-off socioeconomic circumstances within a country tend to be, on average, significantly taller and heavier than those from poorer socioeconomic conditions (Malina at al. 2005). In contrast to measures of body size, SES variation in peak height velocity (PHV) and age at menarche is generally smaller. Among Polish and British adolescents, those from better-off socioeconomic circumstances attain PHV and menarche, on average, slightly earlier than those in lesser socioeconomic conditions. In contrast, SES differences in PHV and menarche are not evident among Swedish adolescents (Bielicki 1986). SES variation in growth and maturity is generally specific to a given country. Generalisation from one country to another is difficult because criteria of socioeconomic circumstances vary and do not necessarily have the same meaning. Social welfare programs also vary among countries and may function to balance economic or income differentials among families and in turn SES differentials in growth and maturity status of children.

Family size, the number of siblings or children in a family, may be a confounding factor in evaluating the effects of SES on growth and maturity because larger family sizes occur more often among those of lower SES. In general, among better-of children whose fathers worked in non-manual occupations (higher SES), number of siblings does not affect height except in large families (5 or more siblings). On the other hand, among children whose fathers worked in manual occupation (lower SES), there is seen a gradient for a reduction in the height of children from families with one or two children through families with five or more children. Similar trends are apparent for the heights of adolescents (Billewitz at al. 1983). After taking into account differences in birth weight, maternal height, and age at PHV, adolescents from large families of unskilled manual workers are significantly shorter than average. Age at menarche also shows a variation with family size (Malina at al. 1997, Kemper 2000, Booth at al. 2002, Tomkinson at al. 2003).

Many case studies of children and adolescents clearly indicate the influence of adverse living conditions, particularly psychosocial or emotional circumstances, on maturation and growth. This condition is sometimes labelled “psychosocial dwarfism” or “deprivation dwarfism” in the clinical literature. Size attained is stunted and maturity

severely delayed, often by as much as 2 to 3 years. Children reared under such psycho-social and emotional circumstances ordinarily do not have specific endocrine or metabolic disorders or a familial history of growth retardation, and their diets are often adequate. The mechanisms through which the stressful home environment influences somatic growth and maturation are not known, but most likely include maternal deprivation, isolation of child from others, a disorganised family life, and, occasionally, physical abuse. Nevertheless, the matrix of environmental circumstances associated with "deprivation dwarfism" can lead to impaired or suppressed growth hormone production and inadequate dietary intake or impaired nutrient utilisation. More specific aspects of the home environment also need consideration. Several recent studies, for example, suggest a potential influence of household composition (e.g., presence or absence of the father) and family distress (e.g., death of a parent, divorce, or potential alcoholism) on indicators of growth and maturity (Ge at al. 1996, Ellis at al. 1999, Ellis and Garber 2000). Familial distress is associated with an earlier age at menarche and shorter adult stature (Hulanicka at al. 2001). Although the associations are not strong, they suggest a role for family relationships and quality of life within the household as potential factors, among others, that may influence growth and maturation.

Motor development, performance, and physical activity occur in a social context (e.g., home, play, and school). Each of these contexts places specific demands on the motor competencies and physical activity of infants, children, and adolescents. The specific influences of social conditions have not been systematically evaluated in the con-text of motor development, performance, and activity. Social conditions interact with the child's rate of neuromuscular maturation, physical characteristics and rate of growth, prior to current experiences in motor activities, and habitual physical activity. A bio-social or bio-cultural framework is essential for discussions of social conditions on motor development and performance. The child carries out motor acts in a social context (Malina at al. 2005).

As in the case of growth and maturation, the specific influences of social conditions on motor development, performance, and activity are difficult to indicate. Quality of living conditions, family size or number of siblings, interactions among siblings, area of

residence (urban-rural or inner city-suburban), and overall socioeconomic circumstances, all of which are related, are potentially important factors to consider.

Variation in lifestyle associated with social class is often viewed in the context of socioeconomic status. Indicators of SES per se do not directly influence motor competence and physical activity. Rather, SES more likely influences motor competence and activity through effects on lifestyle, such as rearing practices, opportunity for activity, and access to special instruction, equipment, and facilities. A confounding factor with SES is its association with ethnicity and race in many parts of the world (Malina at al. 2005). Variation in motor development among ethnic groups is also often attributed to class differences in rearing, but class differences are not always consistent across studies (Malina 1980, 1988). Information on social class and motor achievements at ages beyond 2 years are not extensive, and studies indicate no relationship between SES and motor performance of school-age children. In a large sample of Polish youth, parental education and family living conditions commonly used proxies for SES, had only weak influence on a variety of motor performances (Wolanski, 1993). The role of variation in SES of the family on habitual physical activity of children and adolescents has received some attention. Among Taiwanese adolescents 12 to 14 years of age, no SES differences were seen in estimated total energy expenditure and energy expenditure in mode-rate-to-vigorous activities in boys for either weekdays or weekend days. Corresponding data for Taiwanese girls indicated no differences in estimated total energy expenditure by SES but greater energy expenditure in low SES girls in moderate-to-vigorous activities on weekend days. Although these trends are suggestive, SES accounted for relatively little of the explained variance in estimated total energy expenditure (Huang and Malina 1996). In contrast, SES was related to physical activities of other samples of adolescents. In a sample of Canadian adolescents, high SES was related to the student's intention to exercise (Godin and Shephard 1986). Paternal occupation was significantly related to overall frequency of exercise (Gottlieb and Chen 1985).

Specific aspects of the socioeconomic environment may affect motor competence and physical activity, but these are not detected by the commonly used indices of SES. More important, the translation of specific SES or familial characteristics into variation in

motor performance and physical activity is not ordinarily done. What is unique about a high or low SES familial environment that may affect the activity and performance of individuals in these households? SES, for example, is a factor that influences access to many organised sports and club programs, often at relatively young ages, in which children receive specialised instruction and practice under the guidance of trained coaches.

2.5 Facts, evidences, and conclusions by the reviewed literature

1. The list of the hormones involved in the regulation of the processes underlying growth and maturation is lengthy, and recent advances in understanding the biology of hormones and mechanisms of their actions highlight the complexity of these processes. In recent years, several new growth-promoting factors secreted by the liver, adipose tissue, and other organs have been identified. However, their roles in growth and maturation are poorly defined at this time. Growth hormone, IGF-I, and thyroid hormones have growth-stimulating properties and are primarily involved in the maintenance of normal growth during childhood. The events of puberty and the adolescent growth spurt are dominated by the stimulation of the testes or ovaries by gonadotropins secreted by the anterior pituitary and the markedly elevated production of sexual steroids by the gonads. Production of growth hormone increases at this time as a consequence of the rise in sex steroid production during mid-puberty. The onset of puberty is mediated by changes in the central nervous system, specifically the hypothalamus. Recent evidence suggests a role for leptin in sexual maturation. Leptin is necessary for sexual maturation to occur, but it is not by itself the triggering factor of puberty.

2. The growing and maturing child and adolescent adapt to the stresses imposed by physical activity, especially, systematic activity. Regular physical activity does not alter the processes of growth and maturation as they are ordinarily monitored but it is an important factor in the regulation of body weight and specifically fatness. Regular physical activity functions to enhance skeletal mineral and significant factor in the structural and functional integrity of skeletal muscle tissue. Physical inactivity in combination with a chronically excessive energy intake is associated with greater levels of fatness. Although regular physical activity is related to health-related physical fitness,

the relationship is not strong, and indicators of activity account for a relatively small percentage of variation in several indicators of fitness. Results of trainability studies highlight the specificity of training.

3. Children show considerable increase in performance of some skills between 5 and 8 years of age (running speed and the shuttle run) but show a steady, more gradual increase in performance of other skills from 5 years of age through childhood (jumping, throwing, strength). There is much overlap between the sexes during childhood. During adolescence, in contrast, performances of boys, on average, show a marked improvement, so sex differences are magnified. Age, height, weight, physique, and body com-position account for a substantial portion of variation in performance during childhood and adolescence, but considerable amount is not accounted for by these variables. Factors not related to growth status that nay contribute to the variation in performance include motivation, opportunity for practice and instruction, learning, habitual physical activity, and perhaps others in the cultural environment. The stability or tracking of indicators of strength and motor performance is less than estimates for body size, physique, and fat-free mass.

4. Factors associated with the social environments, racial/ethnic background, and climate can influence of growth, performance, and physical activity. These factors are complex and interact with each other. The specific operation of these factors, and undoubtedly others, is difficult to specify and highlights the plastic nature of the processes growth and maturation. Presently available observations also emphasise the sensitivity of the processes of growth and maturation and physical performance and activity to conditions in social, cultural, and physical environments of children and adolescents.

5. The past decade has witnessed remarkable progress in genomics and human genetics. Although the research on molecular genetics of physical activity, health related fitness and health-related outcomes is still in its infancy, we recognised that understanding the effects of DNA sequence variation on inter-individual differences in responsiveness to acute exercise and regular exercise holds great promise. Such data not only would help to develop more concrete public health measures regarding the role of physical activity

in the prevention and treatment of chronic diseases but also would pro-vide an opportunity to individualise preventive medicine.

6. The primary objective for having an active lifestyle throughout the life span is to contribute to a healthy, enjoyable, productive, and long life. It is possible for some minority of people to be quite sedentary throughout most of their life and still have success and satisfaction, remain reasonably free of major diseases, and avoid weight gain during a long life. In the future, we may be able to identify the genetic and biological profile that makes this situation possible, but for the present we do not have the knowledge or procedures to determine whom these people might be. Thus, the public health goal should be facilitate an active lifestyle throughout the life span for the entire population by means of the following:

- Improved education to increase the public's knowledge about why physical activity is important and how to go about developing a physically active lifestyle.
- Changes in the built environment that makes safe activity available to all.
- Adoption of policies that encourage activity during all aspects of life (occupation, education, transportation, retirement).
- A health care system that aggressively promotes disease prevention, health promotion, and quality of life by means of improved health behaviours, including physically active lifestyle.

3. OBJECTIVES

3.1 Significance of the problem

Improving and also maintaining optimal body composition, physical activity and physical fitness in children may provide both immediate and long-term health benefits. Although the link between regular exercise and health has been established in adults, much less scientific documentation for such a relationship exists in children and youth. Still, regular physical activity in children and adolescents can be expected to have long-term salutary outcomes because the adult diseases influenced by activity often have their origins in the pediatric years. The idea that children should expect a similar salutary effect of physical activity is not altogether obvious. Children do not suffer from disease outcomes for which activity provides benefits for adults. Indeed, as Blair and col-leagues (1989) emphasized, morbidity and mortality in the pediatric age group result principally from accidents, infections, hematologic malignancies, and congenital mal-formations, conditions for which no beneficial effect of physical activity should be expected. Moreover, the habitual physical activity patterns of children, consisting of frequent short bursts of exercise, are different from those of adults, and children's cognitive and physical immaturity makes exercise interventions more problematic.

These observations notwithstanding, a strong rationale has been developed for the promotion of physical activity and fitness in children for both present and future health. Much of this is based on the recognition that the clinical markers of chronic disease in adults are expressions of lifelong processes that begin during childhood and adolescence. Other positive outcomes, such as mental well-being and academic performance, are more immediate (Armstrong and van Mechelen 2000). Promotion of physical activity in children has therefore gained acceptance as a sound strategy for improving health, again both in general population and in risk-specific individuals.

This effort has been stimulated by a concern that the amount of habitual physical activity of children – who are surrounded by an increasingly technological society – is on the decline. There are, in fact, no scientific data on which to base that idea (largely because of the difficulties in accurately assessing physical activity levels in populations). Still this trend is suggested by indirect evidence: the rising frequency of

obesity among children, data suggesting a secular decline in field endurance performance, and increase in television watching time and other general sedentary pursuits (Tomkinson at al. 2003).

The role of exercise as a therapeutic intervention for specific disease states is determinative. Evidence exists that improving physical activity in children may prove beneficial for emotional, cardiopulmonary, and musculoskeletal disorders, but most of the research data are fragmentary (Bar-Or and Rowland 2004). It is important to separate the health outcomes of physical activity in youth from those related to physical fitness. Different factors influence physical activity, which is behaviour, and physical fitness, which describes above all the ability to perform a motor task (Bouchard at al. 2007). The potential health benefits from regular physical activity and fitness may not be the same, and each calls for a different interventional strategy (i.e. behavioural modification for improving activity, a period of exercise training for increasing fitness).

In adults, an individual's level of regular physical activity is often considered a surrogate marker of physical fitness (and vice versa), but this does not appear to be true in children. Somewhat surprisingly, most studies have indicated little relationship between habitual physical activity and physical fitness (at least as defined by maximal aerobic power) in children (Morrow and Freedson 1994, Foster C and Hillsdon M 2004, Vuori at al. 2010). Moreover aerobic training programs in pre-pubertal children conducted according to the standard criteria for frequency, duration, and intensity cause only small increases in maximal aerobic power (about 5%). From a health-outcomes standpoint, then, activity and fitness may need to be considered separately, at least in the growing years.

Certain issues have proven troublesome for those wishing to scientifically document the rationale for promoting physical activity for health in children. Quantifying levels of activity is particularly difficult, given the recurrent short-burst types of activity characteristic of this age and the inability of young children to accurately report activity levels. In addition, the effectiveness of improving activity habits on well-being in children is often unclear, because potential adverse health outcomes by which to gauge success will not surface clinically for decades to come.

Levels of physical activity decline quite precipitously during the growing years, which is largely a biological phenomenon (Rowland 1998, Armstrong at al. 2000). Consequently, differentiating normal developmental changes in activity from those reflecting environmental, psychosocial, and other mediating influences can be difficult (Cavill at al. 2001, Sleap at al. 2001, Sallis at al. 1992).

3.2 The aim of the study, questions, hypotheses

3.2.1 The aim and questions

The aim of the longitudinal study was to analyse and evaluate the pattern of somatic (morphological and selected functional, physiological) growth and development in Limassol elementary school boys. Although the statistical and biological relationship between the speed of somatic development and physical performance may have special importance in some questions, we intend to draw above all public health-related conclusions.

We wish to realize the fulfilment of the main goal along the answers and conclusions for the following questions:

1. Is the change of basic body dimensions and calculated anthropometric indices proportionate with the increase in stature between the years of 6 to 10?

2. What distorting effects can be observed in the growth patterns if the subjects are overweight or definitely obese?

3. Is there a statistically significant human biological relationship between the age-related changes of somatic (body composition) and functional characteristics?

3.2.2 Hypotheses

Before the summary of possible assumptions some important facts should keep in view. (a) Although the somatic development of Cypriot boys was analysed in a nation-wide cross-sectional representative sample (Photiou 2008) longitudinal reference is not available. (b) Because the speed and pattern of somatic and functional growth and development are biologically determined (the more or less expressed inter-race differences can not be excluded) the observed sample dependent characteristics arise from the environment. (c) Cyprus can be evaluated as a technologically developed country, consequently, the general trends and all effects of life-style (UN Population

Division 2003, Lissau at al. 2004, Mamen and Martinsen 2010) are necessarily effective in our society too. According to the great number of respective scientific publications instead of zero hypotheses we prefer the research hypotheses.

Premise 1: The basic assumption in respect of our first question is that exclusively the biologically determined regulation of growth and development never results in non-physiological disproportion (Mészáros at al. 2008). The possible list of opposite statements is rich. The great number of modifying and very often distorting effects could be mentioned in this relationship (Boreham at al. 2001, Tomkinson at al. 2003). Both the national (Photiou 2008, Pampakas at al. 2010) and the earlier quoted international results suggest the significant biological effects of body fat content relative to mass. We suppose this indicator will remarkably modify the biologically determined pattern of growth and somatic development. The observed proportion of overweight and obese children will be high in our sample, consequently the overall health prediction of our children will be negative.

Premise 2: The judgment of biological relationships between various indicators is a difficult task, especially in early childhood. The characteristic fast growth and development in these age groups very often overlap the negative (or sometimes already non-physiological) consequences of the unfavourable body composition (Salmon at al. 2005, Cole at al. 2007, Hume at al. 2008). We can not forget, nevertheless, the risk factors are active, consequently the developed status is dangerous. We can not neglect the biological relationships between the depot fat (or the low level of muscle mass) and the observed physical and physiological indicators but the correlations will be weak or moderate in the given pairs of variables.

Premise 3: The increased stiffness of the aorta and large artery entail an increase in pulse pressure through the compliance and reducing impact of pulse wave reflection. The elevated pulse pressure shows to trigger endothelial dysfunction. We assumed that the long-term obesity greatly affects the quality of the circulatory system and observed the signs of this in the early childhood. A fat group selected averages (ASI) increase confirms this process.

3.3 Limitations

According to our technical and personal conditions the longitudinal study is limited to the group of boys exclusively. We are fully aware that the studied samples represent first of all the Limassol elementary school population, and the generalisation of our results is partly limited, among other things by the subjects' numbers. A comprehensive analysis of body composition, performance and health indicator changes gene-rally requires direct data or by chance estimations that characterise the nutritional habits and lifestyle together. The initial young calendar age of our subjects obviously excluded the usage of questionnaires and additionally the very limited available time (determined by the school managements) the possibility of interview. Active "co-operation" of the parents was also limiting factor.

3.4 Delimitation

The used kinanthropometric and physiologic techniques are accepted by the international literature. The investigators (the same team completed the whole series of data collection) have more decades of practice, consequently there is no doubt that technical error and/or differences in reading accuracy can modify the results. According to the respective literature the measured and also the calculated indicators are valid parameters of growth, development and health status. Finally, we accepted without restriction all those suggested cut-off points and critical values or ranges which were published in authoritative journals.

4. MATERIAL AND METHODS

4.1. Methods and subjects

A total (n=158) of elementary school children took part in the longitudinal data collection between 2007 and 2010. According to the prescription of the Declaration of Helsinki the subjects were volunteer boys exclusively. All of them were definitely Greek origin. Beyond the kind co-operation of the pupils and the school-staff members, the written consents of one of their parents were also collected before the investigation. The following settlements were involved to the investigation: Limasol and the different little settlements around of Limasol. The children were healthy at the time of investigation. All of them took part in the curricular physical education classes (2×45 minutes in a week). Although the level of habitual physical activity can definitely influence the body composition taking into account the low rate of extra curricular physical activity of these boys was not taken into consideration grouping criteria.

To complete the aims detailed anthropometric and psychological measurements were carried out.

4.1.1. Anthropometric methods

Anthropometric measurements for the estimation of physique and body build can be evaluated as relatively new methods. Some of their significant advantages (comparing to somatoscopic techniques) are the clear objectivity (a well-practiced investigator can record the body dimensions reproducibly), and their speed using computer programs during data evaluation.

4.1.1.1. The estimation of Conrad growth type

(Conrad 1963) has suggested the characterization of two developmental directions. The various physique patterns were described by two indices based on different body dimensions. Beyond the constitutional characteristics Conrad has analysed also the bone-muscle development of the physique. Both of the indices can be expressed in decimal numbers.

The metric index

This index relates the chest width to the chest depth and is corrected by the actually measured stature. In first view the metric index is one of the characters of the roundness of the chest, however, following its validation the calculated parameter was characteristic for the roundness or linearity of the whole body. The metric index for girls and females can be calculated as follows:

$$MIX = 0.18\ (CHD - 0.19BH + 0.93CHW - 14.63) \qquad R = 0.999$$

Where: MIX = metric index, CHD = chest depth (cm), BH = height (cm), CHW = chest width (cm), R = multiplied correlation coefficient indicating the congruence between the nomographic and calculated values.

The strongly negative values refer to the leptomorphic body build, the slightly negative ones to the athletic physique, and the positive ones to the picnomorph constitution. For the evaluation of growth type in children and adolescence the respective nation-wide representative data are suggested to use.

The plastic index

This index is the arithmetic sum of three body dimensions that are characteristic for bone-muscle development.

Plastic index (cm) = shoulder width (cm) + lower arm girth + hand circumference (cm)

By the numeric values of these two indices a right-angle co-ordinate system can be created, where the vertical axis is scaled by the metric index and the horizontal one refers to the plastic index. The metromorph-normoplastic body build is located at the centre of the coordinated system. The upper-left quarter contains the leptomorph but hypoplastic individuals; the right-upper quarter refers to the leptomorph-hyperplastic body build. The lower-left area is characteristic for the picnomorph-hypoplastic, and the lower-right quarter contains the picnomorph-hyperplastic physique variants. In children the vertical axis is suggested to be positioned at the level of respective plastic index averages.

4.1.1.2. The estimation of relative body fat content

The expression body composition indicates the ratio of various body substances (for instance: water, protein, fat, muscles bone, different minerals etc.) within the whole body. By the more often used techniques estimate the ratio of depot fat and lean body mass or fat free mass (body mass – depot fat and essential fat mass).

Variability among the results of different body fat estimates gives the reason to use such skinfold techniques by which representative references are available, and the technique was validated by densitometric procedure. The calipermetric estimation of relative body fat content, developed by (Pařízková 1961) meets both conditions mentioned.

This procedure requires the measurement of 5 skinfold thicknesses: over biceps and triceps, subscapular, suprailiac and medial calf.

Procedure: the sum of the 5 skinfold values multiplied by 2, and then entered into the table, where the crossing of the multiplied sum and gender indicates the estimated relative body fat content. The originally published table of Pařízková can be found in Table Appendix No. 2.

As one of the estimates of fatness or obesity the Body Mass Index (BMI) was also calculated.

$$\text{Body Mass Index} = \text{body mass (kg)} \times \text{height (m)}^{-2}$$

4.1.2. Measurement of status of circulatory system

The general status indicators of the peripheral circulatory system (pulse, systolic and diastolic pressure) were before and after the treatment recorded with the CardioVision device (is it the MS-2000 IMDP, Las Vegas, NV, USA) in a lying position, on both upper arms and ankles one after another. The pulse wave pattern was displayed with instrument compatible software (Is the software version 2.05D). The physiologically "normal" artery shows a pyramid-shaped, while the sclerotic artery gives a much flatter pattern with a long stretch (Figure 1). On the figure the vertical axis shows the variation in the pulse amplitude between 80-100%, the horizontal axis indicates the blood pressure and pulse amplitude by which arterial stiffness index (ASI) is calculated. The

lowest limit on the ASI scale is 70.0 units (mmHg x 10) considered a normal value. The ASI scale can reach values between 70.1-280 units as an indicator the different level of abnormality. Arterial stiffness is one of the predictors of cardiovascular event. Arterial stiffness is commonly measured by pulse wave velocity between the carotid and femoral arteries (Sorenson at al. 1997, Rediker at al. 1998).

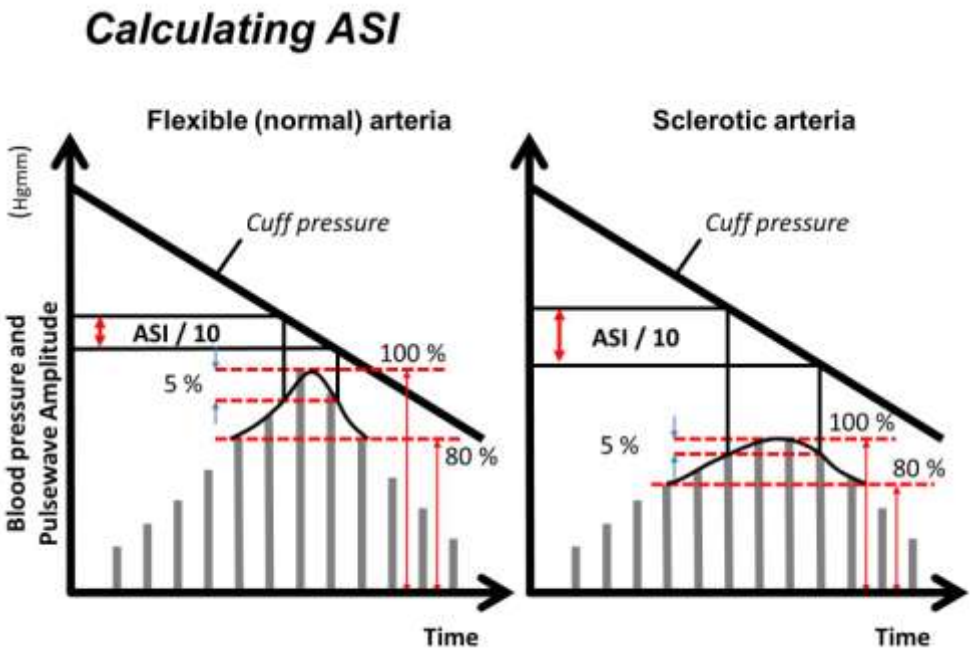

Figure 1. Calculating ASI

Comparison of the pulse wave pattern in a normal and sclerotic artery using the CardioVision device (MS-2000) and software. The relationship between cuff pressure during actual blood pressure measurements and the pulse wave pattern. An Arterial Stiffness Index (ASI) was calculated based on computer-assisted oscillometry.

5. RESULTS

Introduction

Our basic goal in the chapter of results was to present the age-related changes in children's anthropometrical and motoric characteristics (based on the data from the three measurements Table 1). The children selected by fat groups can in turn be a good illustration of direct the negative effects of the unfavourable proportions of muscle and fat on motoric performance which can lead to major cardiovascular problems.

Table 1. Frequency distribution of subjects by their calendar age based on the repeated three surveys

Variable	1. survey (n=158)		2. survey (n=158)		3. survey (n=158)	
	mean	SD	mean	SD	mean	SD
DA	7,95	1,50	8,89	1,51	9,97	1,53

5.1 Height and body mass

Table 2 provides the change in height and body weight over the three year period. The results of the ANOVA indicate a significant increase in both height and body weight. Post hoc tests indicate the, the mean year-to-year increase in height were significant across all age categories. For body weight changes were significant only in the last two age categories.

Table 2. Age-related changes in height and body weight

	Height (cm)		Body weight (kg)	
Age Category	Mean	SD	Mean	SD
6.01 - 6.5	119.03	5.82	23.74	5.07
6.51-7.5	124.93	5.94	26.61	6.03
7.51-8.5	131.61	6.09	30.41	6.94
8.51-9.5	136.09	6.18	33.94	7.21
9.51-10.5	141.23	6.39	37.81	8.09
10.51-11.5	147.79	6.48	42.20	9.45
F	589.60 ($p<0.05$)		769.95($p<0.05$)	

Abbreviations: SD = standard deviation, F = results of the repeated ANOVA.

Table 3 shows the results of the regression analyses. For the entire group as well as the overweight and obese children, the correlations were strong and significant for each parameter associated with body size. The y-intercept for regression was higher in the obese children as compared to overweight and the whole group for height which is expected. For body weight, the y-intercepts were also higher for both the overweight and obese as compared to the whole group. The slopes for height did not differ between the three categories however for body weight; the overweight children had a significantly less steep slope.

Table 3. Body fat-related linear regression coefficients of height and body weight increase

Variable	**Height**			
Sample	**a**	**b**	**r**	**p**
1. Whole	83.998	5.604	0.842	< 5%
2. overweight	85.109	5.343	0.839	< 5%
3. obese	90.723	5.331	0.851	< 5%
Difference	1=2<3	1=2=3		
	Body weight			
1. Whole	-0.178	3.699	0.628	< 5%
2. overweight	4.459	2.826	0.741	< 5%
3. obese	8.367	3.910	0.610	< 5%
Difference	1<2<3	2<1=3		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting.

5.2 Nutritional status

Table 4 lists the change in body mass index (BMI) and relative fat content (fat %) for the six age categories across the study period. The ANOVA indicate significant increase in fat % in each of the categories. BMI did not show significant changes from year-to-year even though there were increases across the age categories.

Table 4. Age-related changes in body mass index and relative fat content

	BMI (kg·m-2)		**Body fat (%)**	
Age (y)	**Mean**	**SD**	**Mean**	**SD**
6.35	16.60	2.33	15.93	5.50
7.28	16.88	2.52	17.47	5.95
8.36	17.39	2.78	19.18	6.09
9.30	18.17	2.98	19.88	6.32
10.26	18.79	3.16	20.92	6.67
11.33	19.15	3.47	21.58	7.01
F	94.72 (NS)		120.74 ($p<0.05$)	

Abbreviations: SD = standard deviation, F = results of the repeated ANOVA.

Table 5 reports the regression and correlation coefficients for both BMI and %fat. The relationships between these fat indices were poor (r<0.30) but yet significant for the whole group and overweight category. For BMI y-intercepts were not significant and only the slopes between the whole group and the overweight children differed. The overweight sample had less vertical than the whole group. For body fat content, the y-intercept was the higher in the overweight and obese groups but had less steep slope in body fat regression.

Table 5. Body fat-related linear regression coefficients of height and body weight increase

Variable	**Body mass index**			
Sample	**a**	**b**	**r**	**p**
1. Whole	12.959	0.553	0.290	< 5%
2. overweight	14.157	0.274	0.291	< 5%
3. obese	19.953	0.276	0.130	NS
Difference	1=2	1>2=3		
	Body fat content			
1. Whole	9.158	1.134	0.290	< 5%
2. overweight	11.463	0.559	0.241	< 5%
3. obese	26.242	0.350	0.18	NS
Difference	1<2	1>2		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting, NS = the correlation coefficient is not significant at 5% level of the random error.

Table 6 lists the means (and standard deviations) for each of the six age categories for metric index and plastic indices. While no significant was found for the metric index, the change in plastic indices (an estimate of bone and muscular development) did have significant findings. The year-to-year differences were significant in all of the age categories.

Table 6. Age-related changes in metric and plastic indices

	Metric index (cm)		**Plastic index (cm)**	
Age (y)	**Mean**	**SD**	**Mean**	**SD**
6.35	-0.85	0.21	59.68	3.75
7.28	-0.87	0.27	61.95	3.66
8.36	-0.93	0.29	64.79	3.97
9.30	-0.96	0.30	66.82	4.17
10.26	-0.99	0.35	69.01	4.32
11.33	-1.01	0.38	71.96	4.68
F	372.15 (NS)		1686.29 ($p<0.05$)	

Abbreviations: SD = standard deviation, F = results of the repeated ANOVA.

Table 7 reports the regression and correlation coefficients for metric index and plastic index. The relationships between fat indicator (metric index) were poor (r<0.30) however the measure of true bone and muscle (plastic index) has moderate-to-strong correlations. For metric index the y-intercepts were lower in the whole group as compared to the overweight and obese groups while the slopes in the whole group were steeper. For the plastic index body fat content, the y-intercept was the higher in the obese groups with no difference in slope for predictability of bone and muscular development.

Table 7. Body fat-related linear regression coefficients of metric and plastic indices increase

Variable	**Metric index**			
Sample	**a**	**b**	**r**	**p**
1. Whole	-0.678	0.029	-0.153	< 5%
2. overweight	-0.488	-0.062	-0.412	< 5%
3. obese	-0.561	-0.004	-0.02	NS
Difference	1<2	1>2		
	Plastic index			
1. Whole	44.551	2.398	0.688	< 5%
2. overweight	45.417	2.153	0.740	< 5%
3. obese	53.567	1.924	0.589	< 5%
Difference	1=2<3	1=2=3		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting, NS = the correlation coefficient is not significant at 5% level of the random error.

5.3. Body Size and Composition

Table 8 list the amount of depot (storage) fat and lean body mass as indicated by body composition measures from year-to-year. The depot fat did not increase significantly for the whole group whereas lean body mass increased significantly over the entire study

period. Post hoc comparisons indicate significant year-to-year improvements for all age categories.

Table 8. Age-related changes in absolute depot fat content and lean body mass

	Depot fat (kg)		LBM (kg)	
Age (y)	**Mean**	**SD**	**Mean**	**SD**
6.35	4.01	2.35	19.73	2.97
7.28	4.94	2.87	21.67	3.43
8.36	6.21	3.51	24.20	3.75
9.30	7.22	4.27	26.72	4.26
10.26	8.43	4.57	29.38	4.67
11.33	9.73	5.09	32.47	5.31
F	151.06 (NS)		734.20 ($p<0.05$)	

Abbreviations: LBM = lean body mass, SD = standard deviation, F = results of the repeated ANOVA.

Table 9 reports the regression results for absolute depot fat and lean body mass. The y-intercept increased significantly for both depot fat and lean body mass across the three sample groups. For the slope characteristics, the least slope was in the overweight group for the depot fat, whereas the obese group had the less slope in lean body mass. For both depot fat and lean body mass, correlations were moderate to strong and all significant.

Table 9. Body fat-related linear regression coefficients of absolute depot fat and lean body mass increase

Variable	**Absolute depot fat**			
Sample	**a**	**b**	**r**	**p**
1. Whole	-3.397	1.151	0.441	< 5%
2. overweight	-0.551	0.626	0.523	< 5%
3. obese	0.839	1.351	0.500	< 5%
Difference	1<2<3	1=3>2		
	Lean body mass			
1. Whole	3.219	2.548	0.722	< 5%
2. overweight	5.010	2.200	0.779	< 5%
3. obese	7.528	0.256	0.660	< 5%
Difference	1<2<3	1=2>3		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting, NS = the correlation coefficient is not significant at 5% level of the random error.

Table 10 reports the relative muscle mass in percentage along with the performance times for the 800 meter run. Results from the ANOVA show significant increase in relative muscle mass percentage however no significant change in the run scores. Post hoc comparisons show that the improvements in muscle occurred in the first three age categories.

Table 10. Age-related changes in relative muscle mass and 800m run scores

	Relative muscle mass (%)		**800m run (s)**	
Age (y)	**Mean**	**SD**	**Mean**	**SD**
6.35	36.16	1.01	337.38	53.89
7.28	38.84	1.55	321.44	48.36
8.36	40.04	1.78	395.19	36.89
9.30	40.30	1.88	283.76	37.41
10.26	40.64	2.15	274.15	38.19
11.33	41.01	2.37	265.62	40.07
F	426.40 ($p<0.05$)		85.60 (NS)	

Table 11 shows the regression coefficients for relative muscle mass and the run performance. In both variables, the correlation coefficients were moderate to strong and significant. For y-intercept comparisons, there were no differences for relative muscle mass and only the obese group had higher values in the 800 meter run performance. The slope comparisons were only different in the obese for relative muscle mass with less of slope in this sample. For the 800 meter run, the steepest slope occurred in the 800 meter run performance for the obese group however was not significantly difference from the overweight group. In comparison, the whole group had less of a slope for regression of these performance indices.

Table 11. Body fat-related linear regression coefficients of relative muscle mass and 800m run score increase

Variable	**Relative muscle mass**			
Sample	**a**	**b**	**r**	**p**
1. Whole	33.261	0.686	0.462	< 5%
2. overweight	31.564	0.954	0.669	< 5%
3. obese	34.815	0.284	0.220	< 5%
Difference	1=2=3	1=2>3		
	800m run			
1. Whole	422.538	-14.199	-0.490	< 5%
2. overweight	431.688	-16.401	-0.620	< 5%
3. obese	488.952	-17.375	-0.468	< 5%
Difference	1=2<3	2=3<1		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting, NS = the correlation coefficient is not significant at 5% level of the random error.

Table 12 lists the blood pressure and heart rate changes across the sample groups and provides regression and correlation coefficients. The results indicate weak yet significant relationships between most these cardiovascular parameters and not significance for diastolic pressure in overweight children and heart rate in obese

children. While the y-intercept for the obese group was higher than the overweight and whole group for systolic blood pressure, diastolic pressure and heart rate intercepts did not differ. The slopes for the diastolic pressure indicate for the obese groups it to be the steepest however no other differences in slope across sample groups and other variables (systolic pressure and heart rate) exists.

Table 12. Body fat-related linear regression coefficients of blood pressures and heart rate

Variable	**Systolic blood pressure**			
Sample	**a**	**b**	**r**	**p**
1. Whole	92.956	2.105	0.391	< 5%
2. overweight	94.453	1.727	0.340	< 5%
3. obese	103.746	1.629	0.232	< 5%
Difference	1=2<3	1=2=3		
	Diastolic blood pressure			
1. Whole	53.297	1.063	0.143	< 5%
2. overweight	57.657	0.297	0.060	NS
3. obese	55.687	1.661	0.230	< 5%
Difference	1=3	1<3		
	Heart rate			
1. Whole	100.863	-1.945	-0.261	< 5%
2. overweight	104.758	-2.520	-0.34	< 5%
3. obese	89.089	-0.253	-0.03	NS
Difference	1=2	1=2		

Abbreviations: a = intercept, b = slope, r = linear regressions coefficient, p = probability of fitting, NS = the correlation coefficient is not significant at 5% level of the random error.

Table 12 states the values provide by the regression analyses on both mean and pulse pressure. The relationships between pressures were weak however mostly significant. The differences lie in mean blood pressure particularly slope in which the obese group

had the steepest values. The overweight group had the highest y-intercept but the less steep slope for regression.

Figure 2 and Figure 3 illustrates the effect on systolic and diastolic pressure based upon weight condition (overweight and obese) around the mean (solid line) pressure in normal weight individuals. Most of these children were above their normal weight peers across the difference age groups. Figure 4 shows the change in mean and pulse pressure for the subjects across the age categories. No differences were evident.

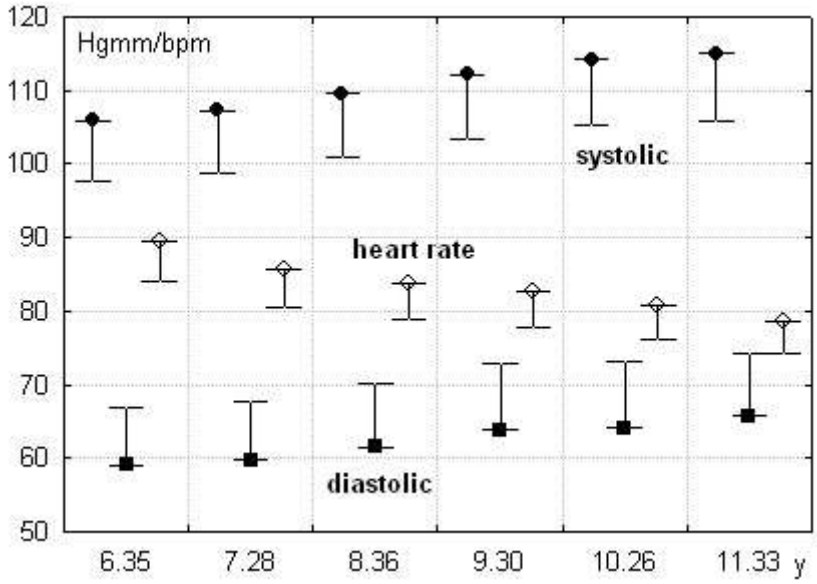

Figure 2. Age-related changes in blood pressure and heart rate

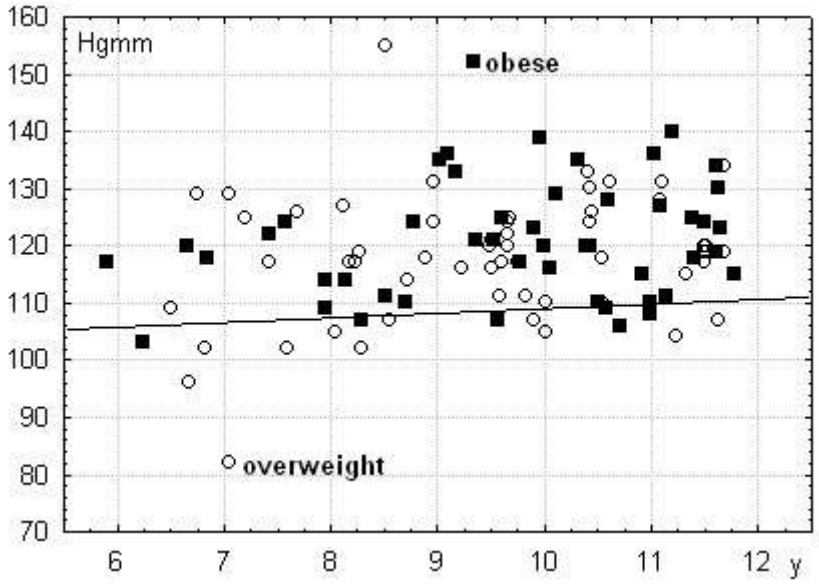

Figure 3. Effects of overweight and obesity on systolic blood pressure

(full line = mean blood pressure of the normal body composition boys).

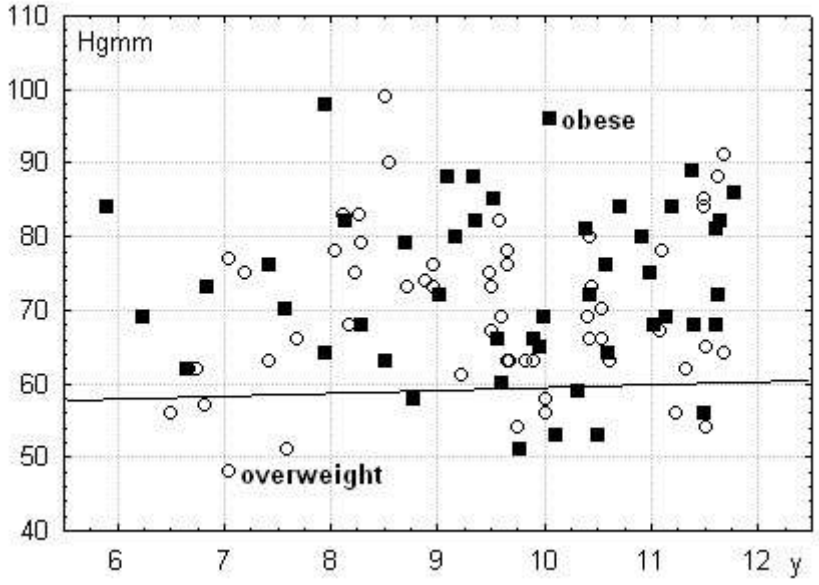

Figure 4. Effects of overweight and obesity on diastolic blood pressure

(full line = mean blood pressure of the normal body composition boys).

Table 13. Mean (SD) measures of body size and composition

	Group	n	Body Mass (kg)	Stature (cm)	% fat	%muscle
Year 1	Normal	56	23.24 (4.35)	123.08 (9.78)	11.99 (2.17)	37.22 (1.88)
	Overweight	55	27.82 (4.60)	128.60 (8.84)	17.32 (1.33)	37.85 (1.74)
	Obese I	19	31.22 (5.83)	129.10 (10.03)	21.46 (1.12)	37.27 (1.83)
	Obese II	28	42.63 (7.68)	137.07 (7.62)	29.32 (2.99)	36.54 (1.98)
Year 2	Normal	56	26.02 (4.81)	128.62 (9.64)	13.50 (2.80)	41.10 (1.75)
	Overweight	55	31.01 (5.65)	134.04 (8.90)	18.59 (3.15)	40.58 (1.49)
	Obese I	19	35.44 (6.63)	134.32 (9.73)	23.55 (3.64)	39.94 (2.06)
	Obese II	28	47.16 (8.73)	142.79 (7.39)	29.58 (3.59)	38.27 (2.03)
Year 3	Normal	56	29.35 (5.28)	135.02 (9.73)	14.53 (3.31)	41.75 (1.37)
	Overweight	55	34.87 (6.32)	140.79 (9.14)	19.90 (3.83)	41.07 (1.75)
	Obese I	19	39.90 (7.25)	141.12 (9.59)	24.28 (4.44)	39.87 (2.51)
	Obese II	28	53.09 (9.74)	149.42 (7.55)	30.91 (3.40)	37.64 (2.22)

Table 14 lists the mean (and standard deviations) for each grouping of children over the three testing periods. The results of a factorial analysis with repeated measures resulted in no interaction between groups and testing periods on stature or percentage fat. There was a significant interaction in body mass and percentage muscle mass (%muscle) however. Body mass increased significantly ($p<0.001$) in all groups (Figure 6).

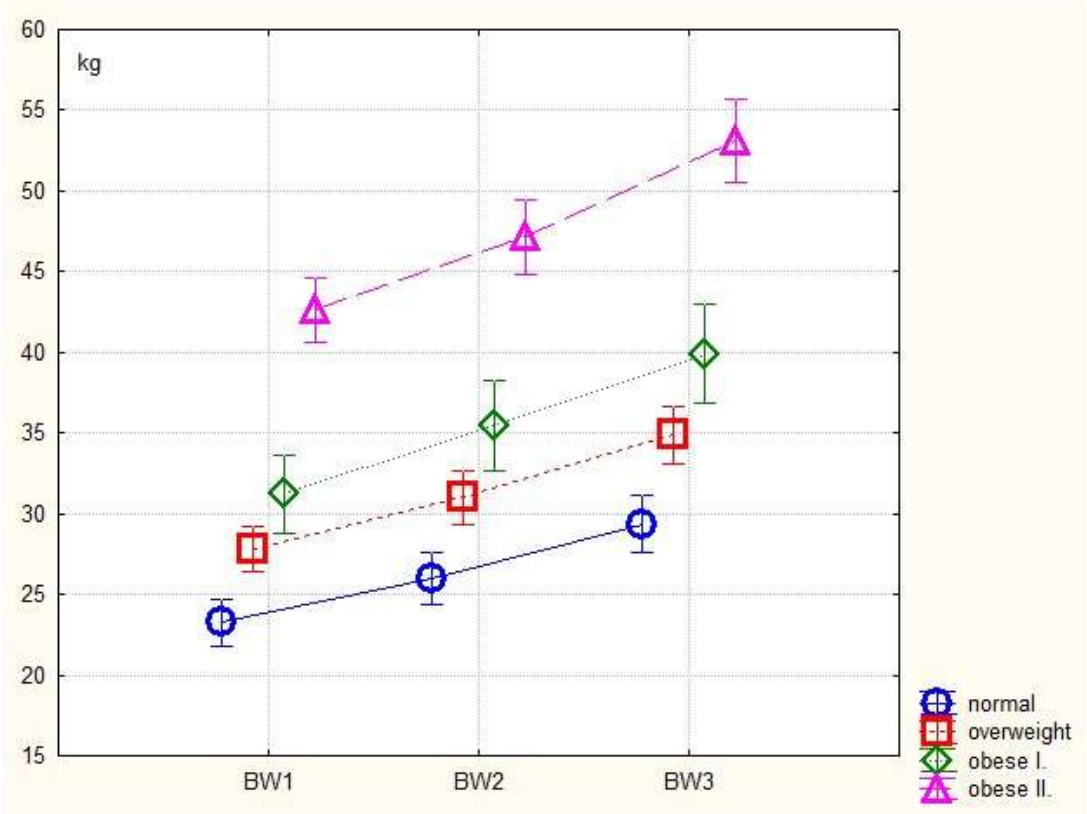

Figure 5. Change in body mass (kg) by group over the study period

Normal weight children had significantly lower in body mass than overweight ($p<0.001$), and obese I ($p<0.001$) children groups at each testing session. The obese II children were significantly higher in body mass than the normal ($p<0.001$), overweight ($p<0.001$), and obese I ($p<0.001$) grouping of children (Figure 6).

Percentage muscle mass increased from the first testing session to the second session; however the change was less in the sequential testing session in some groups (Figure 7).

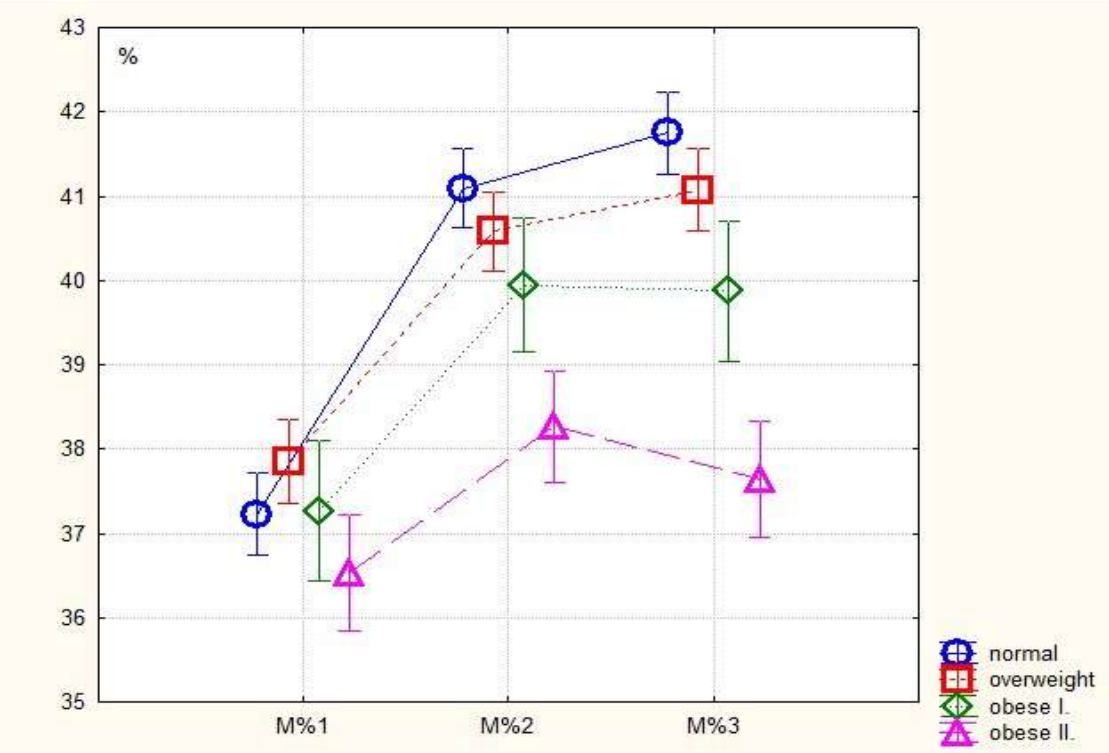

Figure 6. Muscle mass (%) by group over the study period

The normal and overweight groups increased %muscle in all sessions, however for obese I and obese II groups, the improvements were only between sessions one and two. Post hoc comparisons between groups indicate that the obese II grouping had significantly less %muscle than the normal ($p<0.001$), overweight ($p<0.001$), and obese I ($p=0.010$) groups. No other group's comparisons for either body mass or %muscle were significantly different.

Although there was no interaction between groups and testing period on stature, both main effects (group and testing period) were statistically significant. Stature increased significantly ($F=58.19$, $df = 2$, $p<0.001$) over the study period (Figure 8).

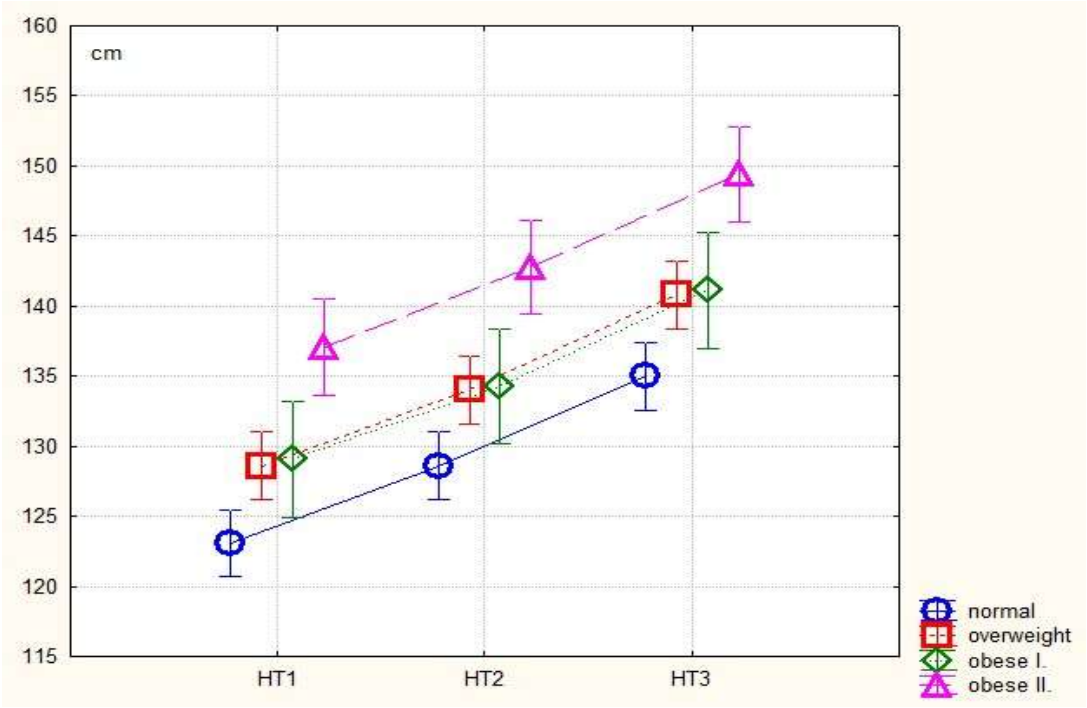

Figure 7. Changes in stature (cm)

Post hoc comparisons indicate stature for the second measure was significantly ($t=60.78$, $df = 157$, $p<0.001$) higher than the first measure and that the third measure was significantly ($t=59.02$, $df = 157$, $p<0.001$) higher than the second measure. Between groups comparisons indicate significantly ($F=15.38$, $df = 3$, $p<0.001$) differences. The normal weight grouping were shorter than the overweight ($p=0.17$). The obese II children were significantly taller than the normal weight ($p<0.001$), the overweight ($p=0.001$) and obese I ($p=0.028$) groups.

As expected, the percentage fat (%fat) changes did not alter the groupings. The factorial analysis did not detect an interaction however main effects for group and time were significant. The %fat increased in all groups significantly ($F=52.11$, $df = 2$, $p< 0.001$) within the study period and %fat was significantly ($F=266.11$, $df = 3$, $p< 0.001$) different between groups (Figure 9).

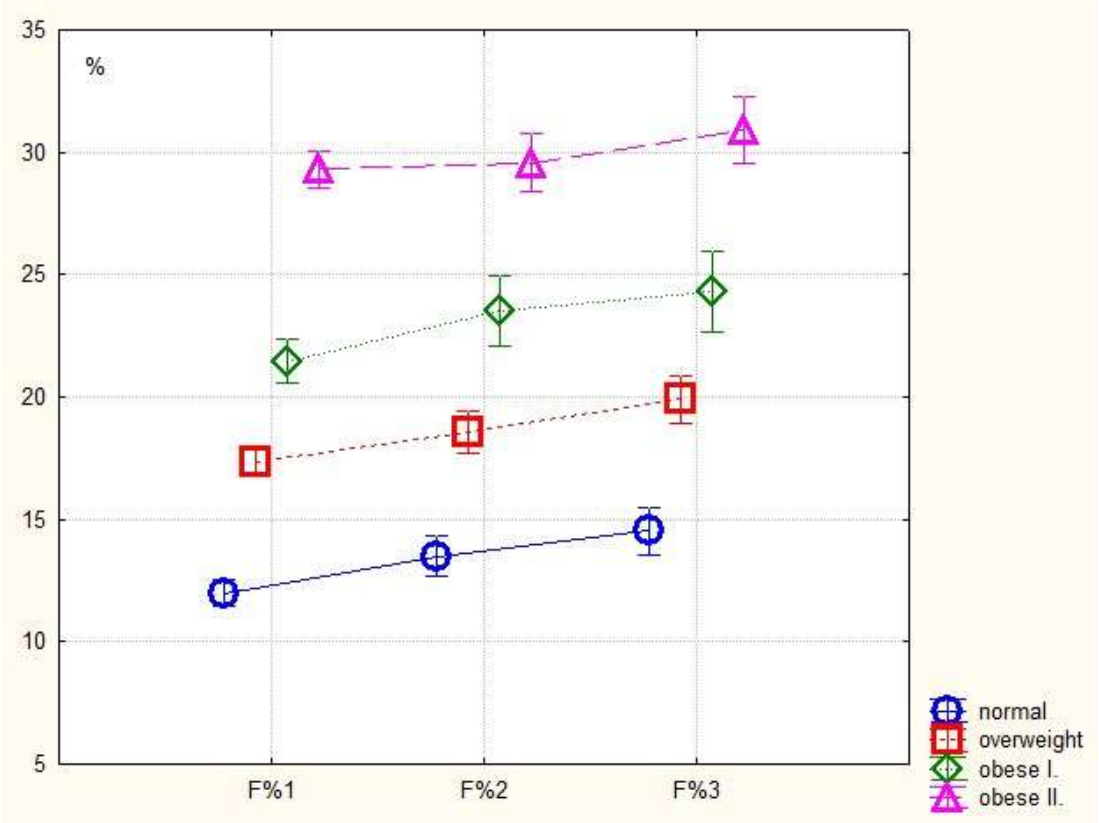

Figure 8. Changes in (%) fat

Post hoc comparisons between measures indicate %fat for the second measure was significantly ($t=6.45$, $df=147$, $p<0.001$) higher than the first measure and that the third measure was significantly ($t=6.08$, $df = 147$, $p<0.001$) higher than the second measure. Post hoc comparisons of groups indicate that all groups were significantly ($p<0.001$) different between each other.

5.4. 800 meter run performance

For the normal weight group, the mean 800 meter run (SD) time was 306.8 (49.9) seconds versus 289.3 (45.1) seconds versus 268.5 (33.0) for trials 1, 2, and 3 respectively. For the overweight group the mean (SD) time was 299.9 (48.8) seconds versus 293.2 (43.7) seconds versus 277.1 (41.6) seconds. For obese 1 group, the trials times were 303.3 (47.0) seconds, 299.4 (53.9) seconds, and 272.9 (40.3) seconds. For obese II group, the trials times were 332.2 (68.9) seconds, 330.4 (58.5) seconds, and 309.2 (44.4) seconds. The results of a factorial analysis with repeated measures resulted

in no interaction between groups and testing periods on 800 meter run performance however main effects were significant. The 800meter run times decreased significantly (F=33.40, df=2, p <0.001) in all of the groups (Figure 10).

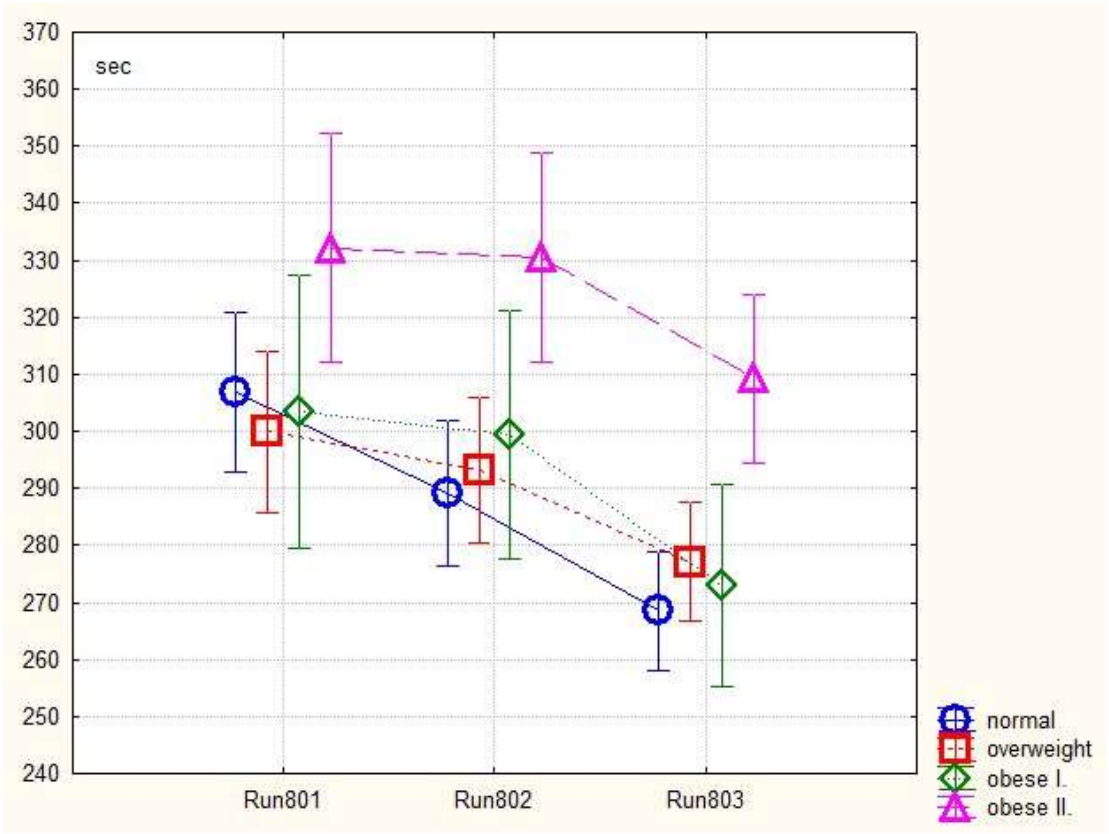

Figure 9. Changes in 800 meter run performance by group

Post hoc comparisons indicate a significant (t=2.71, df =156, p=0.007) faster time between trial one and two (308.4 vs. 298.9 seconds) as well as a significant (t=6.90, df=157, p<0.001) faster time between trial two and three (298.9 vs. 279.2 seconds) for all groups combined. Between groups comparisons indicate a significant (F=5.37, df=3, p=0.002) difference on 800 meter run performance. The obese II group was significantly slower than the normal (p = 0.004) and overweight (p = 0.007) groups. No other group differences were significant.

5.5. Cardiovascular Measures

Table 2 lists the mean (and standard deviation) measures for each group and testing session. The results of a factorial analysis with repeated measures resulted in no

interaction for systolic blood pressure, heart rate, or arterial stiffness index. A significant interaction was evident in diastolic pressure and pulse pressure however. Diastolic pressure was significantly ($p<0.001$) higher in both obese groups as compared to the normal and overweight group. The pattern of change for each testing session was variable among groupings (Figure 11).

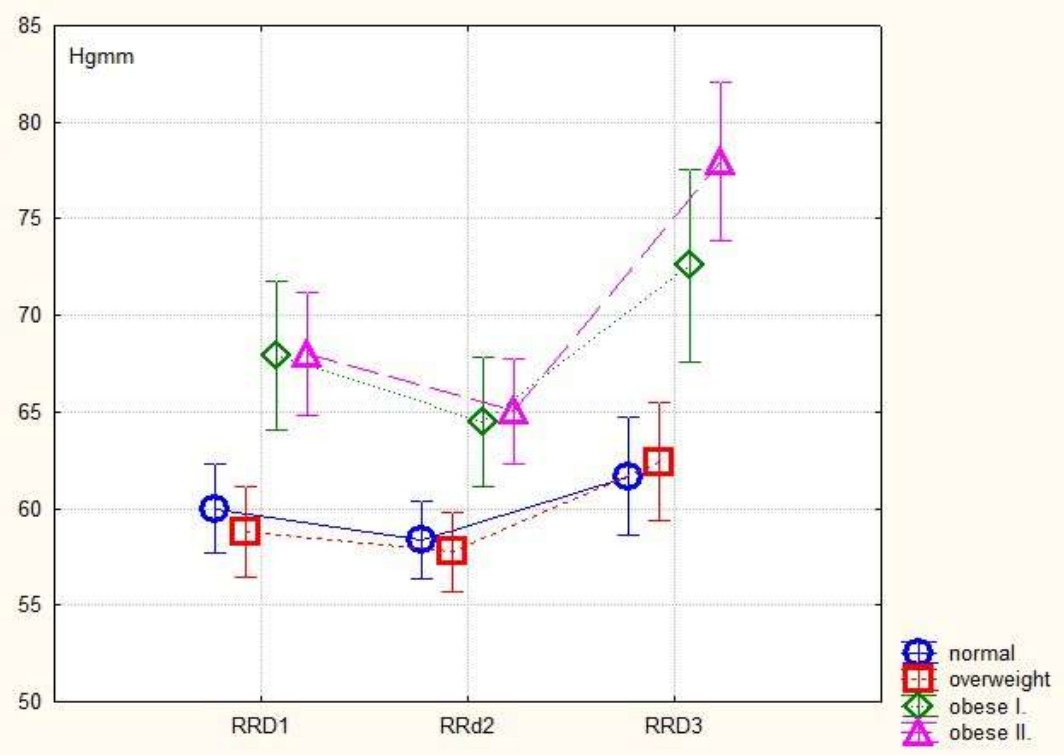

Figure 10. Diastolic pressure (Hgmm) by group over the study period

No increases were significant in the normal weight group, whereas the overweight group significantly increased their diastolic pressure only in the third testing session. In contrast, the obese groups (both I and II) did not significantly increase diastolic pressure over the study period.

Testing the main effects for pulse pressure resulted in contrast in the results. No significant differences were evident between groups in pulse pressure however within group do suggest significant changes within the obese II group (Figure 12).

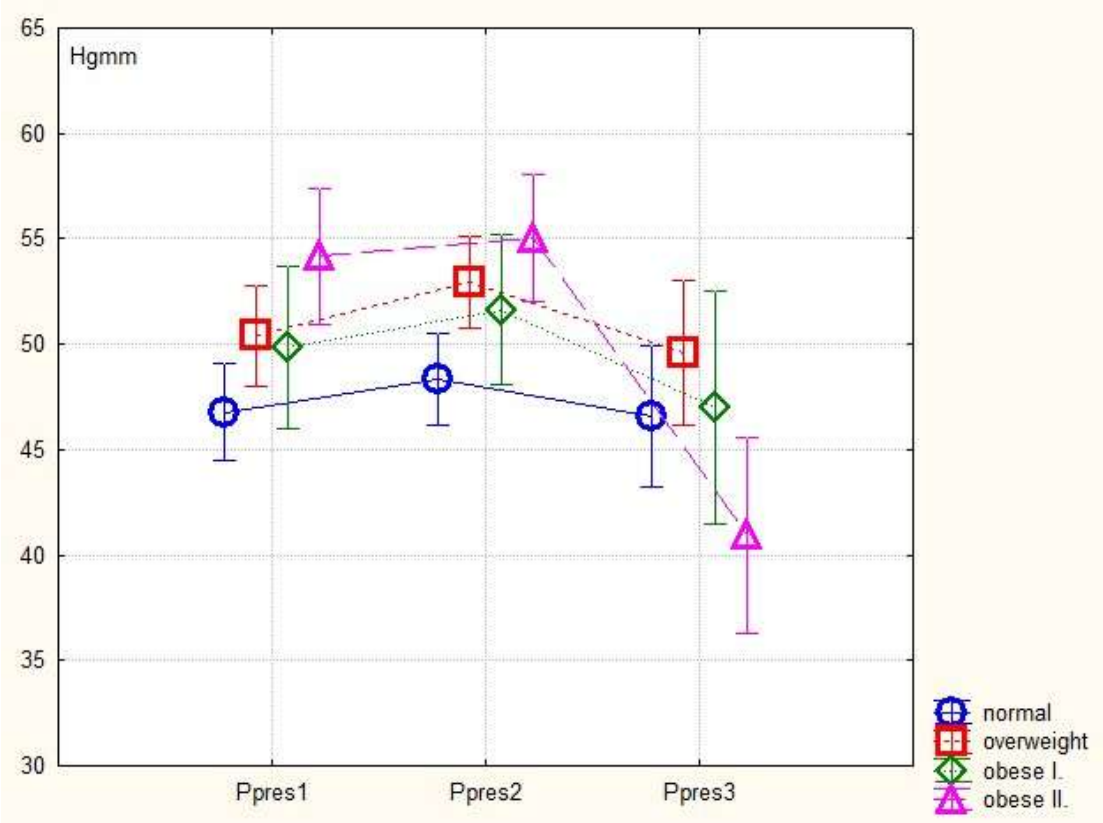

Figure 11. Pulse pressure (Hgmm) by group over the study period

In this group, the pulse pressure decreased and was significantly difference from testing session one (t= 5.00, df= 26, p<0.001) and session two (t=4.25, df = 26, p<0.001). No other differences within other groups were significant.

Table 14. Mean (SD) cardiovascular measures

	Group	Systolic Pressure (mmHg)	Diastolic Pressure (mmHg)	Pulse Pressure (mmHg)	Heart rate (bpm)	Arterial Stiffness Index
Year 1	Normal	106.79 (9.61)	60.00 (8.49)	46.79 (8.42)	87.40 (9.59)	41.14 (11.46)
	Overweight	109.16 (9.78)	58.76 (9.09)	50.42 (8.87)	81.82 (13.38)	43.02 (8.73)
	Obese I	117.74 (6.51)	67.89 (7.08)	49.84 (7.03)	86.53 (12.54)	46.78 (15.93)
	Obese II	121.14 (10.81)	68.00 (8.20)	54.19 (8.93)	86.46 (10.28)	47.29 (16.54)
Year 2	Normal	106.73 (8.60)	58.40 (6.49)	48.33 (8.03)	84.64 (10.77)	44.39 (15.51)
	Overweight	109.43 (6.45)	57.76 (6.68)	52.94 (7.19)	79.14 (11.37)	48.00 (14.20)
	Obese I	115.05 (11.72)	64.47 8.76)	51.63 (5.77)	85.05 (13.47)	53.50 (22.36)
	Obese II	119.82 (9.52)	65.04 (9.00)	55.04 (9.80)	85.65 (11/15)	48.96 (28.22)
Year 3	Normal	108.23 (7.47)	61.67 (11.49)	46.56 (12.05)	80.40 (9.61)	46.49 (11.47)
	Overweight	111.88 (8.40)	62.41 (10.25)	49.62 (11.91)	76.24 (11.62)	45.04 (10.68)
	Obese I	119.58 (10.61)	72.58 (11.48)	47.00 (9.94)	80.42 (10.55)	49.78 (10.96)
	Obese II	118.96 (9.73)	77.96 (11.32)	40.96 (14.44)	80.92 (11.79)	58.08 (25.19)

The group main effect of systolic pressure was significant ($F=23.87$, $df=3$, $p<0.001$). Post hoc comparisons indicate that normal weight children had significantly lower values than obese I ($p<0.001$) and obese II ($p<0.001$) groups of children. The overweight group also had significantly lower systolic pressure than obese I ($p=0.003$) and obese II ($p<0.001$) groups. No changes across time in systolic pressure were significant (Table 15).

Among the other cardiovascular parameters, only main effects for arterial stiffness index were significant. For all groups, the arterial stiffness index increased significantly ($F=4.43$, $df=2$, $p=0.01$) over the study period. Post hoc comparisons indicate that the index was significantly lower in the first measure as compared to the second ($t=2.23$, $df=145$, $p=0.027$) and the third measure ($t=3.40$, $df=151$, $p=0.001$). Comparing between groups results, the arterial stiffness index was significantly ($F=3.95$, $df=3$, $p=0.01$) with

post hoc testing indicated only that the normal weight group had significantly ($p = 0.03$) lower values than the obese II group and no other significant between groupings (Figure 13).

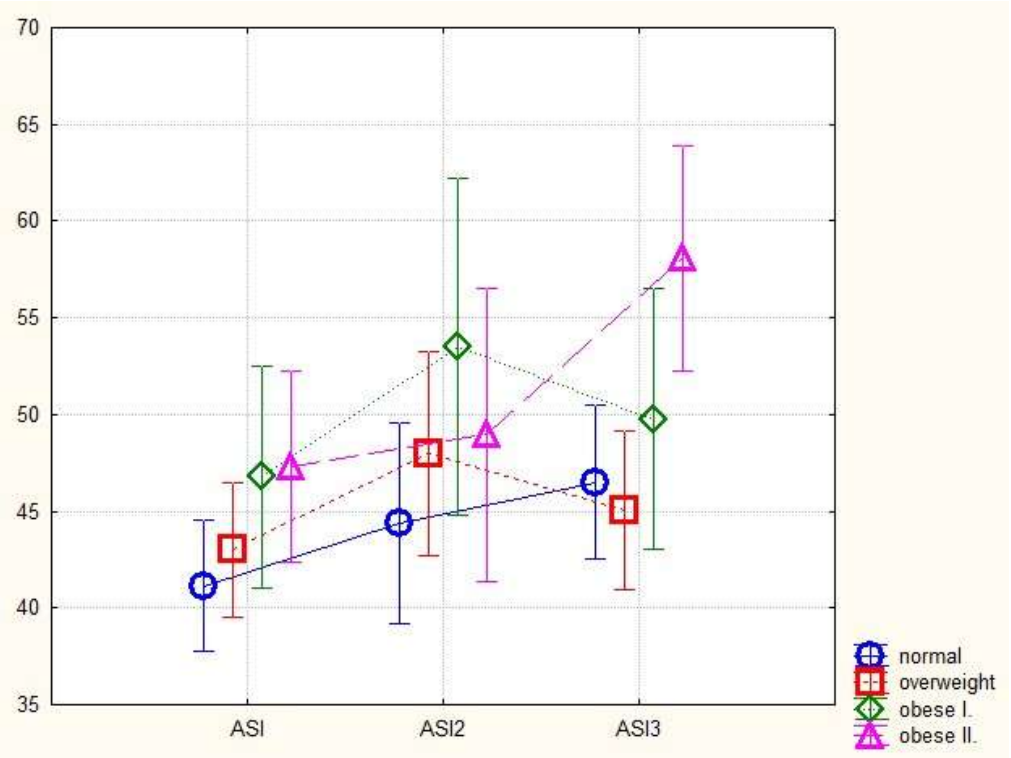

Figure 12. Arterial stiffness index (ASI) by group over the study period

6. DISCUSSION

Introduction

In some European countries economic and social changes affected many people adversely. Concurrently, we also have to face processes that are characteristic of consumer societies, a gradual decrease of habitual physical exercise and its consequences manifested by unfavourable changes of body composition and physique.

During the 20th century body size in the human populations changed rapidly first in the industrialized countries and later in the majority of the developing countries too. This change of the body measurements and proportions is called as secular trend or secular growth change and the most remarkable phenomenon of it is the increase of adult height in each following generations (Eveleth and Tanner 1976). The relationships between stature and weight have changed in different ways in various national groups. Few sets of data allow conclusions about possible secular trends in body composition, but subcutaneous fat thicknesses have increased, especially at the upper percentiles. Also strength, which reflects muscle mass, has increased absolutely, although it has decreased relative to stature. Undoubtedly the secular trend is due to various factors; the identification of causes is necessarily speculative. Changes in nutrition alone could not account for the trends which exceed the original socioeconomic differentials. In the United States, there have been per capita increases in the intake of protein and fat from animal sources, decreases in carbohydrates and fat from vegetable sources, and little change in caloric intake. It is not clear that these changes constitute better nutrition.

The secular trends could reflect environmental improvements, specifically changes in health practices and living conditions leading to improvements in mortality rates and life expectancy (Lohman at al. 1998). The environmental improvements which affect mobility and transportation methods increase levels of physical activity by increasing walking and enabling non-motorized access to areas of high traffic. These changes are thought to develop physical capacities (ability to sustain work) and promote habitual physical activity behavior (choose to walk rather than rely on public transportation). In children, these environmental improvements may play a role in the development of muscularity and reduction of fat storage.

6.1. Summarizing our data it can be stated that the results of this study verified the original hypotheses:

1. The increases in body mass were significant for all groups which are expected with increases in stature over the study period. The components of body composition (fat% and %muscle) did differ between the groups across the study period. The %muscle increase was significant for the obese groups but only between the first two years, while the other groups (normal and overweight) continued to increase over the entire study three year study period. Cessation of gaining muscle might be an artefact for this study population however considering that the obese children were also taller and heavier than the other groups at the first measure, continued natural growth might be slowed by hormonal feedback loops. Over the ages (6-10 years) in boys, we do not expect early sexual maturation which would add testosterone as the growth determinant. With adequate (and possibly excess) nutrition in the obese groups, the development of fat deposits might be a natural reaction to reduction in human growth hormone (HGH) (and thus insulin-like growth factors) levels in the last year. Since this study did not monitor these anabolic hormones, this explanation of the lack of muscular development in the third measure for obese groups is just speculative. Of course, if the normal and overweight groups engaged in physical training which develops skeletal muscle, then the gains over the obese groups are expected. In these young children, physical training however is not expected to provide significant gains over control (a group that does not engage in this activity). Therefore lower %muscle in the obese children might be due to their physical inactivity, a behavior that has been linked to skeletal muscle atrophy. In our opinion the significantly taller mean stature of overweight and obese boys between 7 to 11 years of age needs a detailed explanation. The taller height of over-feed individuals is a relatively new phenomenon in the literature of human biology. Hernandez and co-workers (1994) firstly, and later among others Mohácsi and associates (2003), reported the taller stature of obese children and adolescents. The two possible explanations were the extra energy consumption and the related advanced biological development (acceleration) of these subjects. The effect of advanced biological development seems to be self-evident, because after 12 years of age (following the rapid phase of puberty) the differences between the averages were not significant. Nutrition acts on growth mainly through two mechanisms. The first is the,

direct manner, due to the presence and actions of energetic substrates and molecules with structural functions. The second is an indirect manner, through the endocrine system. The role of nutrition on the hormonal regulation of growth is nowadays less known. In addition, the mechanism through which nutrients regulate or modify hormonal actions or tissue growth factors remains unclear. Regarding a possible role in the interaction of growth and nutrition, more attention has been focused on IGFBP-3 and IGFBP-1. According to the current data, IGFBP-3 would be the principle binding protein of IGFs; its concentration would be regulated by HGH, most likely not directly, but through IGF-1. This would explain the reason why IGFBP-3 is lower in clinical cases of malnutrition, although HGH levels are elevated and its levels are normal or relatively elevated in obesity where IGF-1 level is elevated. Body build of our subjects was estimated by the growth type indices. Its application is more often by the investigators of former East Germany (Jaeger at al. 2004).

2. The performance in the 800 meter run improved in all groups over the study period. While this "weight-bearing" activity might be influenced by higher body mass, the increase in the performance suggests that the gains in stature had a beneficial effect on running. It's likely that the stride length of the children increased as their stature improved. This longer stride would result in a faster velocity and therefore improve the time to complete this endurance event. Therefore if improvements in fitness capacity are of concern in obese children, the data suggest that a similar gain with growth is expected in all groups. For this endurance event, the lack of muscular development did not seem to influence the performances. Other fitness measures, such as a push up or pull up might have resulted in differences in the development of fitness over the study period since this task are dependent upon muscular development.

3. The increase in systolic and diastolic pressure over the study period should be explained. The differences between systolic pressure in the obese and non-obese (normal and overweight) might be due to differences in stroke volume at rest. The amount of blood ejected into the systemic arteries is mostly due to heart size. The obese groups were taller and heavier with might suggest larger heart size using the law of proportions. However no differences were evident in heart rate between the groups which were to validate this statement. Therefore, the higher systolic pressure might be viewed as a potential sign of early stage hypertension. The higher diastolic pressure in

the obese groups confirms that these children might be showing indices of hypertension, a condition associated with obesity. If cardiovascular disease is linked to obesity, even in children, the arteries might be susceptible to “stiffness” evaluated in this study. For this comparison, the obese groups as well as the overweight children showed higher arterial stiffness indexes (ASI). This measure has been linked to peripheral vascular disease in adults, a form of cardiovascular disease which causes reduced circulation in the limbs. More information is needed to determine if higher ASI in children suggest this condition.

7. CONCLUSIONS

The current obesity epidemic is affecting most countries in children as well as adults. The expected biological development in children may be altered with obesity if this condition influences cardiovascular load, endocrine function and target tissue for hormones, and the muscular system. This study has used the period of pre-adolescence to determine if growth in the children tracks as expected and if these growth patterns influence blood pressure, and human performance. The findings suggest that although changes in growth (particularly height) progresses as expected, the levels of adiposity (fat depots) might be influence by more than just biology. The muscularity of boys should begin to influence there function and along with bone development alter their body composition in a positive fashion. What is interesting is that body fat % relative to size did not begin the reduction evident with growth in boys. All groupings of boys (normal weight to obese III) increased their fat% over the three years. Perhaps these changes reflect a more global problem of "increasing adiposity" due to overconsumption of food and lack of physical activity. This study did not monitor food intake nor assess the levels of physical activity. Perhaps the biological development of muscular and the "energy imbalance" of adiposity are both working on these children during their pre-adolescent years. While not detrimental to performance (in the 800m run), these changes might place the child at higher risk for diabetes and hypertension. Future studies in the growth of children should include measures of glucose tolerance, cholesterol/triglyceride levels, as well as blood pressure and arterial stiffness. These risk factors associated with the metabolic syndrome has been shown to be linked to development of cardiovascular disease. Also in future studies, waist circumference should also be measured to obtain a measure linked to visceral fat in the abdominal area. This region is now known to increase the risk of cardiovascular disease in adults and along with high body mass index, cause for concern in the prevention of obesity related diseases.

8. SUMMARY

Childhood obesity has reached epidemic proportions and is considered a serious public health problem. Childhood obesity is a source of serious concern because obese children tend to remain obese adults, and obese children are at increased risk for potentially devastating consequences both during childhood and adulthood. The aim of the longitudinal study (tree times measurements) was to analyse and evaluate the pattern of somatic (morphological and selected functional, physiological) growth and development in Limassol (n=158) elementary school boys between 2007 and 2010.

The increases in body mass were significant for all groups which are expected with increases in stature over the study period. The components of body composition (fat% and %muscle) did differ between the groups across the study period. The %muscle increase was significant for the obese groups but only between the first two years, while the other groups (normal and overweight) continued to increase over the entire study three year study period. Cessation of gaining muscle might be an artefact for this study population however considering that the obese children were also taller and heavier than the other groups at the first measure, continued natural growth might be slowed by hormonal feedback loops.

The performance in the 800 meter run improved in all groups over the study period. While this "weight-bearing" activity might be influenced by higher body mass, the increase in the performance suggests that the gains in stature had a beneficial effect on running. It's likely that the stride length of the children increased as their stature improved. This longer stride would result in a faster velocity and therefore improve the time to complete this endurance event.

The increase in systolic and diastolic pressure over the study period should be explained. The differences between systolic pressure in the obese and non-obese (normal and overweight) might be due to differences in stroke volume at rest. The higher diastolic pressure in the obese groups confirms that these children might be showing indices of hypertension, a condition associated with obesity. For this

comparison, the obese groups as well as the overweight children showed higher arterial stiffness indexes (ASI).

What is interesting is that body fat % relative to size did not begin the reduction evident with growth in boys. All groupings of boys (normal weight to obese III) increased their fat% over the three years. Perhaps these changes reflect a more global problem of "increasing adiposity" due to overconsumption of food and lack of physical activity.

9. ÖSSZEFOGLALÁS

Európai és a tengerentúli tanulmányok alapján a gyermekkori elhízás járványszerű méreteket öltött, amely súlyos közegészségügyi problémák sorát vetíti előre. A vázolt problémán belül a gyermekkori elhízás komoly aggodalomra ad okot a vizsgáló szakemberek körében. Tapasztalatból tudjuk hogy az elhízott gyermekek több mint 80%-a elhízott felnőttként, halmozottan fokozott veszélynek van kitéve. A hosszmetszeti vizsgálat célja az volt, hogy elemezze és értékelje a mintában résztvevő gyermekek (n =158) morfológiai és funkcionális jellemzőit, azok hatását a keringési rendszerre, Limassol és környéke általános iskoláiban, 2007 és 2010 között.

A testtömeg átlagok különbségei szignifikánsak az összes csoportban, ami várhatóan termet folyamatos növekedését vetíti előre a vizsgálati periódus alatt. Ami a két legfontosabb testalkotó átlagait illeti (F% M%) azok csoportonként különbséget mutatnak az teljes vizsgálati időszak alatt. A testtömegre vonatkoztatott zsír mennyisége függetlenül a növekedés és az érés periodicitásától, folyamatosan nőtt a vizsgálati idő alatt minden csoportban. A relatív izom (M%) növekedés szignifikáns különbséget mutat az első két vizsgálati évben (minden zsírcsoportban), míg a második és a harmadik vizsgálat között a különbség nem valódi. A termet tekintetében jelentős a különbség a normál és elhízott csoportok között, amely eltérő hormonális hatás együttes eredményeként értékelhető.

A 800 méteres futó teljesítmények minden csoportban javultak a vizsgálati periódus alatt. Míg az egyik oldalon a termet előnyös növekedése pozitív irányba befolyásolja az állóképességi tevékenységet, addig a másik oldalon a testtömeg előnytelen változása gyengíti a nyereséget. A termet előnyös növekedése döntően a lépéshosszban okoz változást, amely gyorsabb sebességet eredményez, így a gyermek rövidebb idő alatt teljesíti az állóképességi tevékenységet. Ez önmagában rossz hír, hiszen a javuló teljesítmények mögött döntően véletlenszerű hatások állnak, nem pedig a folyamatos és rendszeres fizikai aktivitás hatásai.

A nyugalmi állapotban vizsgált keringési jellemzők tekintetében a szisztolés és a diasztolés nyomások folyamatosan nőttek a vizsgálati idő alatt. Az elhízottak csoportjaiban a két jellemző a kritikus fiziológiás határ közelében található. A magasabb diasztolés nyomás átlagok kapcsolatba hozhatók a gyermekkori elhízással. Ezek a hatások mintegy erősítve egymást már tetten érhetőek a nagyartériák rugalmasságát

minősítő (ASI) számokban. A fent vázolt problémák mögött döntően a túlzott energia bevitel és az alacsony szintű fizikai aktivitás áll.

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11. Publications of Mr. Polydoros Pampakas

1. Mészáros Zsófia, Mészáros János, Uvacsek Martina, **Polydoros Pampakas**, Osváth Péter, Völgyi Eszter, Frenkl Róbert (2007) A szomatikus és motorikus fejlődés különbségei 7-11 éves fiúknál – a szocio-ökonomiai status hatásai. *Sportorvosi Szemle*, 48: 114-119.
2. Mészáros Zsófia, Vajda Ildikó, Mészáros János, **Polydoros Pampakas**, Sziva Ágnes, Osváth Péter, Zsidegh Miklós (2007) Korai gyermekfejlődés: a szocio-ökonómiai státus hatásai. *Magyar Sporttudományi Szemle*, 8: 8-13.
3. Zsidegh Petra, Mészáros Zsófia, Faludi Judit, **Pampakas Polydoros**, Völgyi Eszter, Zsidegh Miklós (2007) Prepubertárs korú fiúk testi és fizikai teljesítmény-fejlődésének megítélése egy integrált paraméter alapján. *Magyar Sporttudományi Szemle*, 8: 17-24.
4. **Pampakas Polydoros**, Mészáros Zsófia, Photiou Andreas, Sziva Ágnes, Zsidegh Petra, Mészáros János (2008) Az emelt szintű testnevelés hatása 7-11 éves fiúk szomatikus és motorikus jellemzőire. *Magyar Sporttudományi Szemle*, 9: 3-8.
5. Mészáros Zs, Mészáros J, Völgyi E, Sziva Á, **Pampakas P.**, Prókai A, Szmodis M. (2008) Body mass and body fat in Hungarian schoolboys: differences between 1980-2005. *J Physiol Anthropol*, 27: 241-245.
6. **Pampakas P**, Mészáros Zs, Király T, Bartusné SzM, Szakály Zs, Zsidegh M, Mészáros J. (2008): Longitudinal differences and trends in body fat and running endurance in Hungarian Primary schoolboys. *Anthropol Anz*, 66: 317-326.
7. Prókai A, Kiss K, Mavroudes M, **Pampakas P**, Zsidegh M, Mészáros Zs, (2008) Depózsír-független teljesítmény-különbségek nem sportoló fiúknál. *Magyar Sporttudományi Szemle*, 9: 20-22.
8. Zsófia Mészáros, János Mészáros, B. Márta Szmodis, **Polydoros Pampakas**, Péter Osváth, Eszter Völgyi (2008) Primary school child development – issues of socioeconomic status. *Kinesiology*, 40: 153-161.
9. Vajda Ildió, Batta Klára, Hegedűs Ferenc, Vajda Tamás, **Pampakas Polydoros**, Bartusné Szmodis Márta (2010) A testi fejlettség, a relatív testzsírtartalom és a Cooper-próbával jellemzett állóképesség nemzedékenkénti különbségei általános iskolás fiúknál. *Magyar Sporttudományi Szemle*, 11: 20-24.

10. **Pampakas P**, Mészáros Zs, Vajda I, Vajda T, Zsidegh M, Mészáros J (2010) Prepubertáskorú fiúk testzsírtartalmának és futóteljesítményének változása: Ciprusi-magyar összehasonlítás. Magyar Sporttudományi Szemle, 11: 17-22. (2010)

11. Mészáros Zsófia, **Pampakas Polydoros**, Zsidegh Miklós, Rikk János, Szmodis Márta, Ihász Ference (2011) Különböző távú futások és a testtömegre vonatkoztatott oxigénfelvétel kapcsolata 9 éves fiúknál. Magyar Sporttudományi Szemle, 12: 11-15.

12. ACKNOWLEDGEMENTS

THE CANDIDATE EXPRESSES HIS GRATITUDE TO THE CO-WORKERS OF THE MINISTRY OF EDUCATION AND CULTURE FOR THEIR KIND PERMISSION THAT WAS REQUIRED FOR THE DATA COLLECTION.

MANY THANKS REFER TO THE RESPECTIVE SCHOOL PRINCIPALS AND TEACHERS AND ALSO FOR THE PUPILS WHO WERE CO-OPERATIVE DURING THE INVESTIGATION.

SPECIAL THANKS TO THE PROFESSORS AND THE WHOLE COLLECTIVE BODY OF THE FACULTY OF PHYSICAL EDUCATION AND SPORT SCIENCES WHO PREPARED ME MENTALLY AND PHYSICALLY.

HOWEVER, I WOULD LIKE TO EXPRESS MY GRATTITUDE TO MY PARENTS AND FAMILY FOR THEIR GENEROCITY DURING MY STUDY IN HUNGARY AS WELL FOR MY POST-GRADUATE EDUCATION I TOOK AT HOME AND ABROAD.