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REVIEW

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Overweight and obesity in European children: definition and diagnostic procedures, risk factors and consequences for later health outcome

Abstract In this article, information on the definition of obesity in childhood and adolescence, its differential diagnosis and its adverse health effects is provided. This information can be considered as a basis for the evaluation of an obese patient. Review of the international and European literature shows that the co-morbidities of childhood obesity cover a large medical area ranging from secondary hormonal disturbances to orthopaedic problems. Most of them have so far been underestimated. However, the most severe problem of an obese child is his or her psychosocial discrimination. Longitudinal data demonstrate a positive relationship between childhood obesity and increased morbidity and mortality in adulthood.

Conclusion Data from the literature are in some respect sparce since they do not allow to calculate the exact prevalence of adverse effects nor to assess the outcome of an obese child when it is successfully reducing the relative amount of body fat.

Key words Childhood obesity · Body fat · Evaluation of obese children · Co-morbidity of childhood obesity

Abbreviation *BMI* body mass index

Introduction

The two most important problems associated with childhood obesity are the psycho-social and the medical consequences [4, 38]. Psycho-social discrimination of obese children and adolescents can lead to social isolation and has consequences for psychological and behavioural development. Increased cardiovascular risk factor levels and several other adverse consequences for physical health in obese children and adolescents lead to increased morbidity even in early life as well as to an increase in mortality in later adulthood [19, 20, 24].

Until recently, these psycho-social and medical problems of childhood obesity have not been clearly recognised by most physicians and paediatricians. More data are needed to clarify the prevalence of several adverse health effects of childhood obesity. Physicians and paediatricians need to be educated to diagnose obesity and associated co-morbidities. Adequate treatment and, of course, stronger possibilities for prevention need to be found.

Definition and differential diagnosis

Obesity is defined as a condition where a pathological excess of body fat is present in an individual. Since the amount of body fat per se is difficult to determine exactly, for practical use, percentage overweight or the body mass index (BMI) is used to define and to track obesity in childhood [23, 26, 42]. In 1996, a committee of the European Childhood Obesity Group (ECOG)

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published a proposal to use the relative (age-adjusted) BMI for the definition of childhood obesity [23]. As with the generally accepted 90th and 97th percentiles as cut-off points in the assessment of body weight and height, so for BMI the 90th percentile defines overweight and the 97th percentile defines obesity. In 1997, the WHO International Obesity Task Force agreed that the paediatric percentiles identified in late adolescence by a BMI of 25 and a BMI of 30 should constitute the cut off points for the identification of childhood overweight and obesity. Additional information on the amount of subcutaneous body fat, and perhaps a more precise quantification of obesity, can be obtained by measuring skinfold thickness, especially the triceps. Accumulation of body fat in obesity occurs in children at the subcutaneous site, whereas in adolescents, intra-abdominal fat depots also increase. During puberty in girls a gluteofemoral distribution of body fat develops whereas in boys an abdominal distribution is present. Independent of this sexual dimorphism, high variations in the body fat distribution pattern within one gender can occur. Interestingly, this pattern correlates with associated co-morbidities.

Obesity develops when there is a discrepancy between energy intake and energy output. During its development the original steady-state is disturbed and after a period of positive energy balance, a new steady-state at a higher level with an increase in body fat stores is achieved. Up to now the genetic factors responsible for the development of obesity and the underlying pathophysiological relationship are not clear. This is true for the vast majority of obese patients. It is also not clear whether there might be phenotypic subtypes of obesity which could be characterised by specific genetic markers.

The small percentage of secondary obesity in childhood resulting from a defined underlying disease has to be recognised when a obese patient is presented to a physician. In general, all these primary diseases can easily be diagnosed by their specific clinical features. They are summarised as follows. Congenital chromosomal defects associated with obesity are Down syndrome, Klinefelter syndrome and Prader-Willi syndrome. Some congenital genetic defects associated with obesity are Laurence-Moon-Biedl-Bardet syndrome, achondroplasia and some growth hormone deficiency syndromes. Adiposo-gigantism in neonates or infants is a symptom of diabetic fetopathia, Sotos syndrome, Wiedemann-Beckwith syndrome, and Simpson-Golabi-Behmel syndrome. Recently, rare monogenetic defects were reported in members of specific families (Table 1): mutations in the leptin gene [18], in the leptin receptor gene [2], in the prohormone convertase 1 gene [9], in the pro-opio-melanocortin gene [14], and in the melanocortin-4-receptor gene [29]. These defects could be shown to be a rare genetic explanation for early onset obesity. One symptom of all these patients has been an increased energy intake due to hyperphagia. In addition, a mutation in a transcription factor peroxisome proliferator activated receptor γ, important for adipocyte differentiation, has been reported to be associated with a higher body fat content in humans [25].

Table 1 Monogenetic defects as causes for a non-syndromal extreme form of early onset obesity in man (as known until January 1999)

Mutations in the gene encoding for:
Leptin
Leptin receptor
Prohormone convertase 1
Pro-opio-melanocortin
Melanocortin-4-receptor
Peroxisome proliferator activated receptor γ

There are acquired endocrine alterations in childhood such as hypothyroidism, growth hormone deficiency and Cushing syndrome which consequently lead to secondary obesity. Obesity is also a characteristic of pseudo-hypoparathyroidism and pseudo-pseudohypoparathyroidism. Diseases where hypothalamic damage is apparent (cerebral trauma, post-encephalitis, craniopharyngioma) also lead to obesity. Other causes of secondary obesity are immobility (e.g. patients with spina bifida), poor linear growth (skeletal diseases) or the side-effects of drugs (corticosteroids, anti-thyroid drugs, sodium valproate and others).

Once the clinical signs lead to the differential diagnosis of one of the above mentioned diseases, specific clinical and laboratory investigations have to be performed to confirm them. When these rare causes of secondary childhood obesity are excluded by clinical examination, primary or simple obesity is diagnosed. Further laboratory investigations should then be performed to estimate the health risk to the obese child or the adolescent. Possible health risk factors and co-morbidities are summarised in the following paragraphs. The knowledge of the family history about such risk factors will help to detect them since for most of them, clustering within families is suggested.

Risk factors and co-morbidities

In general, health risk and adverse effects of obesity in European children and adolescents are not likely to be different from those reported for American children and adolescents [4]. If the occurrence of co-morbidities is dependent on the degree of obesity, then in extremely obese patients a high prevalence of co-morbidities would be found. The higher prevalence of super-obese children and adolescents in the United States compared to Europe (Livingstone, this volume) would lead us to expect a higher prevalence of secondary health effects in the United States. In this respect it should be mentioned that as in the United States, the number of extremely obese children and adolescents seems to be increasing in Europe [1]. When studying historical references in medical journals it is interesting to see that obesity and extreme

obesity have been reported in children and adolescents in the 18th and even 17th centuries [32]. In this review, a girl with extreme obesity is described who presented all typical adverse effects as far as they could be diagnosed at this time. The girl died at the age of 10 years when she had achieved a height of 175 cm and a weight of 109.5 kg. Six other publications on cases of extreme obesity in childhood were also reviewed.

Possible adverse effects of childhood obesity are summarised in Tables 2 and 3. Besides endocrinological and metabolic disturbances associated with obesity in childhood, obesity-related morbidities also include pulmonary, skeletal, dermatological, and immunological diseases as well as adverse social and psychological effects. The prevalence of most of these secondary complications and adverse health effects is not precisely known. There is a need for controlled studies to obtain information on the prevalence of these complications and their relationship to the duration and grade of obesity. The early or immediate consequences of childhood obesity have to be clearly distinguished from the late or long-term effects on health and the psychosocial situation. They are summarised in Tables 2 and 3 and Fig. 1. An extensive review of the literature on different co-morbidities of childhood obesity and underlying patho-physiological mechanisms and their influence on later health outcome has recently been published by Dietz [4]. In the following paragraphs selected issues of health consequences associated with childhood obesity are discussed with special respect to findings in European studies.

Table 2 Early consequences of childhood obesity

Early consequences of childhood obesity
Physical appearance
Psycho-social consequences (see text)
Orthopaedic
Metabolic disturbances (see text)
Nightly hypoventilation and sleep apnoea syndrome (associated with neurocognitive deficits)
Immune system and infections (higher prevalence of bronchitis and upper airway infections)
Skin alterations (skin infections, acne, striae distensae, wound healing problems)
Physical handicap (decreased physical mobility leading to decreased physical activity)
Increased blood pressure and hypertension (and increased left ventricular mass)
Liver steatosis cholecystolithiasis

Table 3 Late consequences of childhood obesity

Late consequences of childhood obesity
Persistance of early co-morbidities [30]
Obese adults [28]
Cardiovascular disease [19, 20]
Cancer [4]

Early consequences of childhood obesity and associated health risk

Physical appearance

Elevated body height [31] and increased body circumference determine the appearance of obese children and adolescents and influence the self-esteem of the individual [8, 21]. The most severe problem of an obese boy is, besides his adiposo-gigantism, the often obvious pseudogynaecomastia and pseudohypogenitalism. In an obese girl the frequently obvious hirsutism and increased acne are additional problems of her outer appearance. These specific clinical changes are also recognised by the

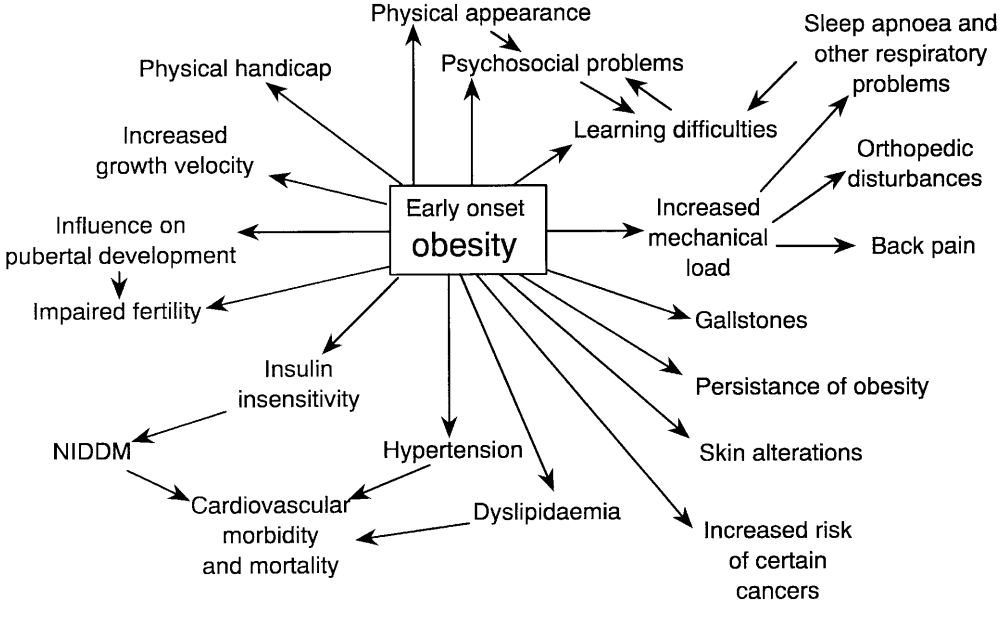

Fig. 1 Health consequences of early onset obesity

environment and schoolmates. It is easy to find reasons to discriminate against these individuals. The psycho-social discrimination in childhood and the resulting changes in the development of an obese child finally influence the socioeconomic status in adulthood [5, 8].

Psycho-social consequences

The main concerns of obese children or adolescents do not lie in the well-known metabolic disturbances of obesity but, at this early stage, is the psycho-social discrimination. Psycho-social discrimination leads in some cases to social isolation. The reason for psycho-social discrimination is complex and can be found on the one hand in the specific culture-related ideal figure. On the other hand, obese children and adolescents have an altered self-esteem resulting from their specific appearance. This leads to alterations in behaviour [8, 21].

Orthopaedic complications

Three relevant orthopaedic findings are genu valga, Blount disease and epiphysiolysis capitis femoris. The occurrence of these alterations is likely to be higher than expected. Genu valga and Blount disease may lead to gonarthrosis. Epiphysiolysis capititis femoris may be silent and be the basis for the development of coxarthrosis.

Metabolic disturbances and elevated blood pressure

Cardiovascular risk factors which are associated with later increased morbidity and mortality can be found in obese children and adolescents. In many studies, childhood obesity has been shown to be associated with increased levels of LDL-cholesterol and triglycerides and decreased levels of HDL-cholesterol [30, 34, 41]. Hyperinsulinaemia and impaired glucose tolerance are also associated with obesity in childhood [17, 39]. Prospective, long-term observations suggest that hyperinsulinaemia in obese children is followed by pathological glucose tolerance and by pancreatic secretory deficiency [16]. The relationship between obesity in adolescence and non insulin-dependent diabetes mellitus has been recently reviewed [27]. A study in Cincinnati showed that 33% of all new cases of diabetes mellitus in childhood were patients with non-insulin-dependent diabetes mellitus [22]. This form of diabetes was especially seen in children and adolescents who have been obese. The ten-fold increase in incidence of non-insulin dependent diabetes mellitus between 1982 and 1994 paralleled the increase in number of obese children and adolescents [22].

Another cardiovascular risk factor is increased blood pressure. Although hypertension is a less common medical consequence of childhood obesity, several studies have shown an association between systolic blood pressure and BMI in obese children and adolescents [4, 6]. It has also been also shown that decreased urinary sodium excretion is associated with hyperinsulinaemia in obese children and adolescents [3], a possible link to the appearance of elevated blood pressure. Compared with age-matched normal-weight children, obese children have a higher blood pressure and an elevated left ventricular mass [11].

The role of body fat distribution in the occurrence of the specific risk factors in obese children and adolescence has been critically reviewed [4, 15, 34, 40, 41]. Recent data clearly show that an abdominal body fat distribution in adolescent girls is associated with an adverse risk factor profile and with hyperandrogenaemia [34, 35]. The relationship of indices of body fat distribution and risk factors in obese boys and younger obese girls is less evident. This is due to the rapidly changing body fat distribution during puberty. This means that it is not possible to compare indices of body fat distribution between 8- and 12-year-old children because of the strong influence of the stage of pubertal development [15]. When investigating the relationships between body fat distribution and risk factors in groups of obese children with the same biological age, it becomes evident that already at early pubertal developmental stages, positive associations between risk factors and body fat distribution in the same way as in adulthood can be detected [15].

Recent data in obese children and adolescents show that weight reduction significantly decreases or even normalises risk factor levels [10, 12, 33, 34, 35]. These positive effects of weight reduction are more pronounced in those with an abdominal body fat distribution. Girls with an abdominal body fat distribution also lose more body fat in the abdominal region than those with a gluteo-femoral body fat distribution [33]. An interesting finding is that the type of body fat distribution is an independent predictor of the achieved weight loss during a standardised weight reduction programme [33]. This weight loss was entirely due to a loss of body fat without changes in fat-free mass [36]. It is not known if obesity in children and adolescents has an impact on adult health if it does not persist into adulthood. Future research to answer this question is needed and prospective studies should be performed to follow the outcome of these individuals.

Metabolic changes seen in obese children and adolescents have been summarised under the so-called multi-metabolic syndrome [7, 30]. The role of decreased growth hormone concentrations and increased growth hormone binding protein levels in the pathogenesis of the multi-metabolic syndrome is not yet clear [13]. The lack of metabolic effects of growth hormone in obesity seems to be directly related to the observed metabolic disturbances. In-vitro data, found with cultured human adipocytes under chemically defined conditions, strengthen this hypothesis [37].

Other early complications of childhood obesity

There are several other early complications of obesity which have been extensively reviewed elsewhere [4]. Taken together, they all show that the knowledge of these co-morbidities of an obese child is essential for the estimation of the health risk and the development of late co-morbidities (Table 3).

Conclusion

Although data from the literature show that a variety of health risk factors are associated with obesity in European children, the exact prevalence of the co-morbidities is not known and no information is available on the importance of the degree and duration of obesity. In order to obtain such data, cross-sectional studies and data from health surveys in a defined population are necessary. Furthermore, there is a need for more studies to track these early complications by longitudinal studies of obese children similar to that recently published by Vanhala et al. [30].

References

1. Barth N, Ziegler A, Himmelmann GW, Coners H, Wabitsch M, Mayer H, Remschmidt H, Schäfer H, Hebebrand J (1997) Significant weight gains in a large clinical sample of extremely obese children and adolescents between 1985 and 1995. Int J Obes Relat Metab Disord 21: 122–126
2. Clement K, Vaisse C, Lahlou N, Cabrol S, Pelloux V, Cassuto D, Gourmelen M, Dina C, Chambaz J, Lacorte JM, Basdevant A, Bougneres P, Lebouc Y, Froguel P, Guy-Grand B (1998) A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction Nature 392: 398–401
3. Csabi G, Molnar D, Hartmann G (1996) Urinary sodium excretion: association with hyperinsulinaemia, hypertension and sympathetic nervous system activity in obese children and control children. Eur J Pediatr 155: 895–897
4. Dietz WH (1998) Health consequences of obesity in youth: childhood predictors of adult disease. Pediatrics [Suppl] 101: 518–525
5. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH (1993) Social and economic consequences of overweight ain adolescence and young adulthood. N Engl J Med 329: 1008–1012
6. Györgyi C, Molnar D (1993) Obesity with hypertonia in children. Gyermekgyogyaszat 44: 393–397
7. Györgyi C, Kozari A, Farid G, Molnar D (1995) Multimetabolic syndrom in obese children. Orvosi Hetilap 136: 595–597
8. Hill AJ, Silver EK (1995) Fat, friendless and unhealthy: 9-year-old children's perceptions of body shape stereotypes. Int J Obes Relat Metab Disord 19: 423–430
9. Jackson RS, Creemers JWM, Ohagi S, Raffin-Sanson ML, Sanders L, Montague CT, Hutton JC, O'Rahilly S (1997) Obesity and impaired prohormone processing associated with mutations in the prohormone convertase 1 gene. Nat Genet 16: 303–306
10. Knip M, Nuutinen O (1993) Long-term effects of weight reduction on serum lipids and plasma insulin in obese children. Am J Clin Nutr 57: 490–493
11. Koehler B, Malecka-Tendera E, Drzewiecka B, Gasior Z, Wackerman-Ramos A, Majer K, Ladarew-Lach I, Dobrowolska-Wiciak B, Girczys W, Lacheta M, Muchacka M (1989) Evaluation of the cardiovascular system in children with simple obesity. Part II. Echocardiographic assessment. Mater Med Pol 2: 131–133
12. Korsten-Reck U, Bauer S, Keul J (1994) Sports and nutrition – an outpatient program for adipose children (long-term experience) Int J Sports Med 15: 242–248
13. Kratzsch J, Dehmel B, Pulzer F, Keller E, Englaro P, Blum WF, Wabitsch M (1997) Increased serum GHBP levels in obese pubertal children and adolescents: relationship to body composition, leptin and indicators of metabolic disturbances. Int J Obes Relat Metab Disord 21: 1130–1136
14. Krude H, Biebermann H, Luck W, Horn R, Brabant G, Gruters A (1998) Severe early-onset obesity, adrenal insufficiency and red hair pigmentation caused by POMC mutations in human. Nat Genet 19: 155–157
15. Luhmann K (1997) Körperfettverteilung und kardiovaskuläre Risikofaktoren bei übergewichtigen Kindern und Jugendlichen vor und nach einer Reduktionsdiät. Dissertation. Universität Ulm
16. Malecka-Tendera E, Koehler B, Wiciak B, Ramos A (1994) Impaired glucose tolerance in obese children – a long-term prospective study. In: Ditschuneit H, Gries FA, Hauner H, Schusdziarra V, Wechsler JG (eds) Obesity in Europe 1993, John Libbey, London, pp 153–158
17. Molnar D (1990) Insulin secretion and carbohydrate tolerance in childhood obesity. Klin Pädiatr 202: 131–135
18. Montague CT, Farooqi S, Whitehead JP, Soos MA, Rau H, Wareham NJ, Sewter CP, Digby JE, Mohammed SN, Hurst JA, Cheetham CH, Earley AR, Barnett AH, Prins JB, O'Rahilly S (1997) Congenital leptin deficiency is associated with severe early-onset obesity in humans. Nature 387: 903–908
19. Mosberg HO (1989) 40-year follow-up of overweight children. Lancet 2: 491–493
20. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH (1992) Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 327: 1350–1355
21. Phillips RG, Hill AJ (1998) Fat, plain but not friendless: self-esteem and peer-acceptance of obese pre-adolescent girls. Int J Obes Relat Metab Disord 22: 287–293
22. Pinhas-Hamiel O, Dolan LM, Daniels SR et al. (1996) Increased incidence in non-insulin-dependent diabetes mellitus among adolescents. J Pediatr 128: 608–615
23. Poskitt EME and the European Childhood Obesity Group (1996) Committee report. Defining childhood obesity: the relative body mass index (BMI). Acta Paediatr 84: 961–963
24. Power C, Lake JK, Cole TJ (1997) Measurement and long-term health risks of child and adolescent fatness. Int J Obes Relat Metab Disord 21: 507–526
25. Ristow M, Müller-Wieland D, Pfeiffer A, Krone W, Kahn R (1998) Obesity associated with a mutation in a genetic regulator of adipocyte differentiation. N Engl J Med 339: 953–959
26. Rolland-Cachera RC, Sempe M, Guilloud-Bataille M, Patois E, Pequignot-Guggenbuhl F, Fautrad V (1982) Adiposity indices in children. Am J Clin Nutr 36: 178–184
27. Rosenbloom AL, House DV, Winter WE (1998) Non-insulin dependent diabetes mellitus (NIDDM) in minority youth: research priorities and needs. Clin Pediatr 37: 143–152
28. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T (1993) Do obese children become obese adults? A review of the literature. Prev Med 22: 167–177
29. Vaisse C, Clement K, Guy-Grand B, Froguel P (1998) A frameshift mutation in human MC4R is associated with a dominant form of obesity. Nat Genet 20: 113–114
30. Vanhala M, Vanhala P, Kumpusalo E, Halonen P, Takala J (1998) Relation between obesity from childhood to adulthood and the metabolic syndrome: population based study. BMJ 317: 319
31. Vignolo M, Naselli A, Di Battista E, Mostert M, Aicardi G (1988) Growth and development in simple obesity. Eur J Pediatr 147:242–244

32. von Eschenmayer (1815) Beschreibung eines monströsen Fett-Mädchen. Tübinger Blätter für Naturwissenschaften und Arzneikunde I: 261–285
33. Wabitsch M, Hauner H, Böckmann A, Parthon W, Mayer H, Teller W (1992) The relationship between body fat distribution and weight loss in obese adolescent girls. Int J Obes Relat Metab Disord 16: 905–911
34. Wabitsch M, Hauner H, Heinze E, Muche R, Böckmann A, Parthon W, Mayer H, Teller W (1994) Body-fat distribution and changes in atherogenic risk-factor profile in obese adolescent girls during weight reduction. Am J Clin Nutr 60: 54–60
35. Wabitsch M, Hauner H, Heinze E, Muche R, Böckmann A, Benz R, Mayer H, Teller W (1995) Body fat distribution and steroid hormone concentrations in obese adolescent girls before and after weight reduction. J Clin Endocrinol Metab 80: 3469–3475
36. Wabitsch M, Braun U, Heinze E, Muche R, Mayer H, Teller W, Fusch C (1996a) Body composition in 5–18 year old obese children and adolescents before and after weight reduction assessed by deuterium dilution and bioelectrical impedance measurement. Am J Clin Nutr 64: 1–6
37. Wabitsch M, Braun S, Hauner H, Heinze E, Ilondo MM, Shymko R, De Meyts P, Teller WM (1996b) Mitogenic and anti-adipogenic properties of human growth hormone in human adipocyte precursor cells in primary culture. Pediatr Res 40: 450–456
38. WHO (1998) Obesity. Preventing and managing the global epidemic. Report of a WHO consultation on Obesity, Geneva 3–4 June
39. Zwiauer K, Schmidinger H, Klicpera M, Mayr H, Widhalm K (1989) Oral glucose tolerance test in grossly obese children and adolescents. Klin Pädiatr 202: 118–122
40. Zwiauer K, Widhalm K, Kerbl B (1990) Relationship between body fat distribution and blood lipids in obese adolescents. Int J Obes Relat Metab Disord 14: 271–277
41. Zwiauer KFM, Pakosta R, Mueller T, Widhalm K (1992) Cardiovascular risk factors in obese children in relation to weight and body fat distribution. J Am Coll Nutr 11[Suppl]: 41S–50S
42. Zwiauer K, Wabitsch M (1997) Relativer Body-mass-Index (BMI) zur Beurteilung von Übergewicht und Adipositas im Kindes- und Jugendalter. Empfehlungen der European Childhood Obesity Group. Monatsschr Kinderheilkd 145: 1312–1318