

Childhood Obesity

Van der Merwe M-T, MBChB, FCP(SA), PhD

Professor of Endocrinology, Senior Consultant Physician Endocrinologist,
Department of Medicine, Division of Endocrinology, Johannesburg Hospital,
Honorary Secretary, International Association for the Study of Obesity (IASO)

Correspondence: Tel: +27 (0)11 484 1323, Fax: +27 (0)11 643 5139, e-mail: tessavdm@iafrica.com

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Introduction

Childhood obesity has been identified as a major threat to children's health worldwide.¹ The prevalence of obesity in children between the ages of 6 and 17 years is currently estimated at approximately 11% (BMI > 95th percentile), with an additional 14% overweight with a BMI between the 85th and 95th percentiles.² These prevalence rates are a dramatic increase from the 1963 – 1970 prevalence rates, when approximately 4 – 4.5% of 6-17 year olds were overweight. (**Fig. 1**)

Aetiology

Childhood obesity is not restricted to a single ethnic, age or socioeconomic group, although cultural, environmental and genetic factors clearly play a role.² In industrialized countries it is children in lower socioeconomic groups who are at greatest risk.³ In contrast, developing countries show obesity to be more prevalent among higher income sectors of the population, and among urban populations rather than rural ones. (**Fig. 2**) The environmental factors that contribute to the development of obesity early in life include a high proportion of sedentary activities (e.g. TV viewing), lower proportion of physical activity, and a shift in diet towards more fast foods with high fat and calorie content. Among the genetic factors, polymorphism of mutations in any of the following genes may be involved in the pathophysiology of

Fig. 1

Trends in overweight children 1970 - 2000

Overweight defined by IOTF criteria. Children's ages may differ between countries.

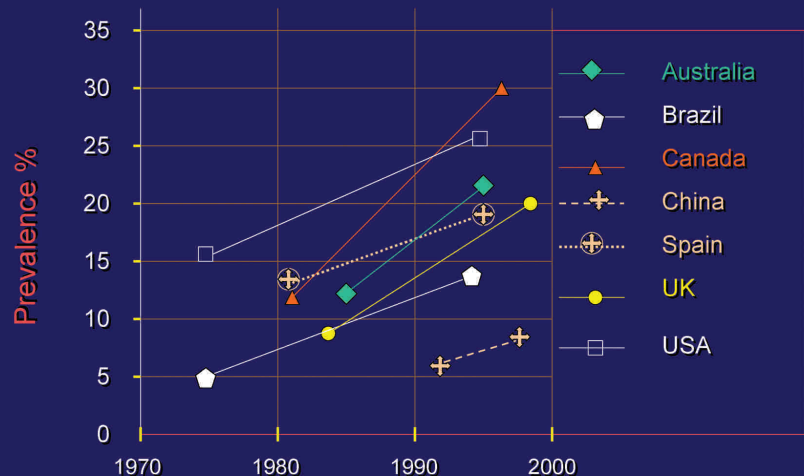
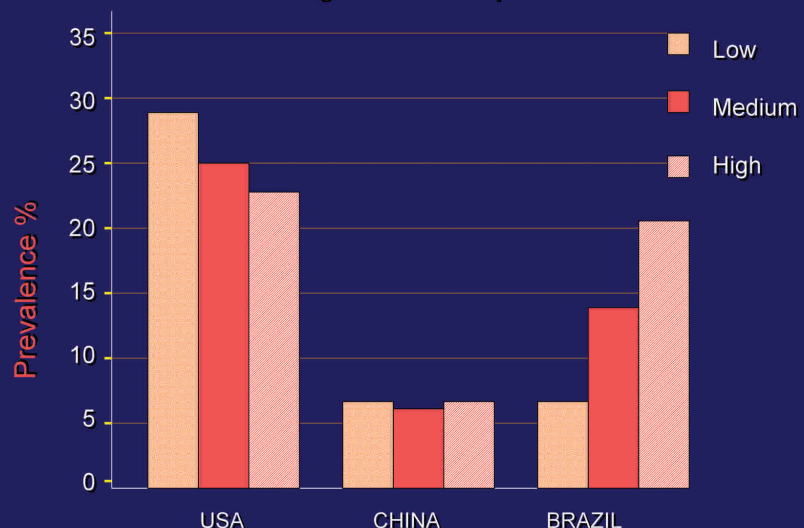


Fig. 2

Prevalence of overweight children by household income



obesity: α -adrenergic receptor, tumor necrosis factor (TNF), pro-opiomelanocortin (POMC), neuropeptide Y (NPY), NPY receptor, melanocortin receptor (M4R), leptin and leptin receptor.⁴ These factors may be responsible for about 40% in the variation in genetic heritability transmission.

Severe childhood obesity is associated with a much higher likelihood of continued overweight. This risk is much higher when the child has obese parents. Studies have shown that parental obesity is the most important risk factor for

obesity in children, owing both to the genetic influence and the shared environment.^{5,6}

Several critical periods in childhood have been identified in the development and persistence of obesity.⁷ In the gestational period, there is a direct association between high birthweight and subsequent adiposity.^{8,9} Likewise, low birthweight with excessive catch-up growth will predispose to adulthood obesity. (**Fig 3**)¹⁰ Thereafter, in the first year of life, children undergo changes in nutritional behaviour which may influence adiposity in later life. Another crucial

period is ages 5 – 8 years, where adiposity increases after its nadir in childhood ('adiposity rebound'). An early adiposity rebound may serve as an index of further obesity.¹¹ The final risk period for the development of persistent obesity is adolescence.¹²

Classification and consequences

Several studies have compared the US-NHANES criteria for defining overweight as obesity using age- and gender specific 85th and 95th centile cut-offs with those of the more recent US CDC using similar percentile cut-offs, and the IOTF alternative set of cut-offs based on centiles passing through the BMI 25 and BMI 30 at age 18. Flegal et al.¹³ showed that the different methods give approximately similar results, but with some significant discrepancies especially among the younger children. In general, the IOTF method appears to give a more conservative view of the extent of overweight and obesity among pediatric populations, compared with methods based on 85th and 95th centiles of US-based reference populations. (**Table I**)

Among the most common sequelae of childhood obesity is hypertension and dyslipidaemia, with an increase in the risk for coronary heart disease (**Fig. 4**)¹⁴, type 2 diabetes, respiratory problems, pseudotumour cerebri and orthopaedic and psychosocial problems. (**Table II**) Since 85% of obese youngsters will become obese adults, the comorbidity profiling exerts an important toll on most societies.

Treatment of childhood obesity

Reviews of the treatment of paediatric obesity have shown that weight reduction can achieve improvement in several co-factors.¹⁵ Compared with young children, adolescents are less likely to accept a highly controlled home or school regime and may provide greater difficulty by refusing treatment and failing to attend appointments.¹⁶ For convenience an addendum with

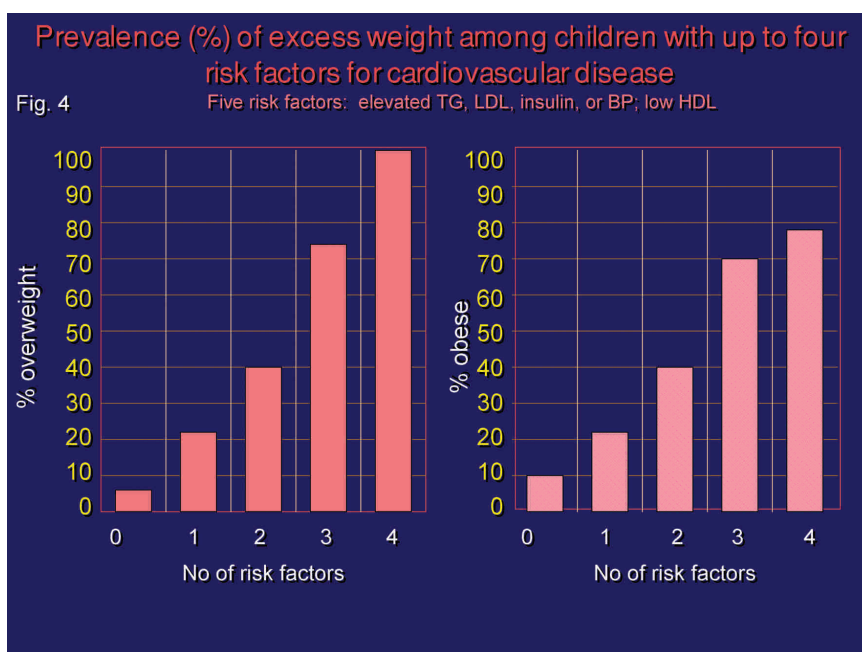
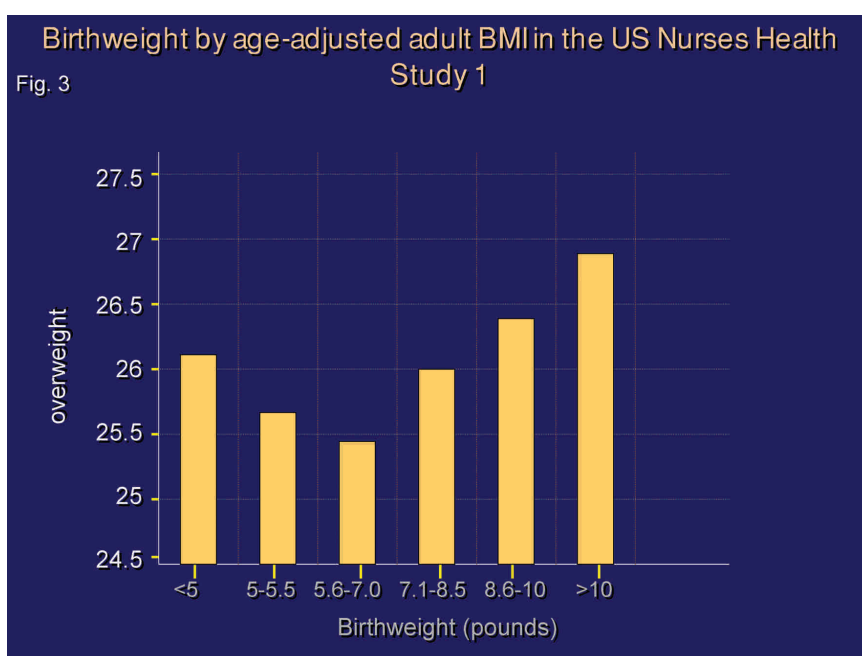


Table I: Comparisons of prevalence rates of overweight and obesity using different criteria

	Age 6 - 8		Age 12 - 13	
	Boys	Girls	Boys	Girls
"overweight"				
NHANES / WHO > 85 TH	25%	31%	30%	30%
CDC > 85 TH	23%	23%	29%	31%
IOTF > BMI 25 equivalent	18%	23%	29%	31%
"obese"				
NHANES / WHO > 95 TH	13%	17%	11%	12%
CDC > 95 TH	11%	11%	12%	12%
IOTF > BMI 30 equivalent	8%	8%	9%	10%

Table II: Physical complaints associated with childhood and adolescent obesity

Pulmonary:	Sleep apnoea Asthma Pickwickian syndrome
Orthopaedic:	Accelerated linear growth Slipped capital epiphyses Blount's disease (tibia vara) Tibial torsion Flat feet Ankle sprains
Neurological:	Idiopathic intracranial hypertension (e.g. pseudotumour cerebri)
Gastrointestinal:	Cholelithiasis Liver steatosis / non-alcoholic steatohepatitis Gastro-esophageal reflux
Endocrine:	Insulin resistance / impaired glucose tolerance Type 2 diabetes Menstrual abnormalities / early puberty / premature adrenarche Polycystic ovary syndrome Hypercorticism
Cardiovascular:	Hypertension Dyslipidaemia

treatment guidelines and resources has been provided.

A recent study on sibutramine in combination with behavioural therapy in adolescents showed some success.¹⁷ A few reports on the use of orlistat in children or adolescents have been presented as abstracts,¹⁸ and β -agonists and other hormonal drugs are currently under development. Concerning the more rare causes of childhood obesity, the results of the use of growth hormone in Prader-Willi syndrome are equivocal.¹⁹

Summary of childhood eating behaviour research

What do we know about how children eat?

Biological Factors:

Children have a preference for sweets and salt and fear new and unfamiliar foods. They are predisposed to learn to prefer energy-dense foods, but can effectively self-regulate when provided with healthful food choices.²⁰

Parenting Factors:

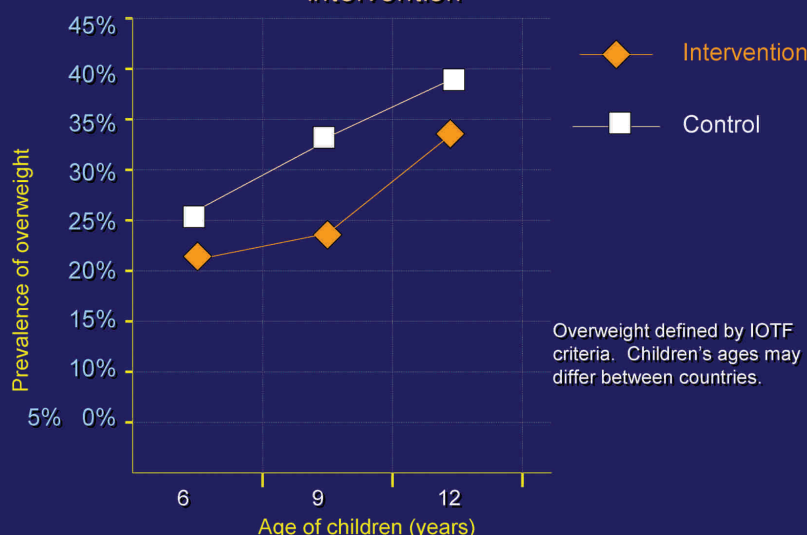
Children need approximately 10 exposures to new foods to accept them. They can learn to prefer new foods as they become increasingly familiar. Children are more likely to eat foods that teachers, parents and peers are eating, and will overeat when they are rewarded for eating. Children will also desire palatable foods that they can see but are forbidden to have.²¹

What do we need to learn?

- Are some children biologically vulnerable to with self-regulating food intake?
- Which comes first – parental restriction or childhood disinhibition?
- Can children self-regulate intake when surrounded by palatable high-calorie foods?
- How does advertising affect children's food preferences and intake?
- How can parents encourage healthful eating without being perceived as withholding?

Fig. 5

Crete: rising rates of overweight are reduced by a school-based intervention



Summary of childhood obesity stigma research

WHAT DO WE KNOW ABOUT THE STIGMA OF CHILDHOOD OBESITY? No-one is immune to negative attitudes towards obese children. Even children as young as 3 years old can experience negativity, that will become stronger with age.²² Teachers and college admission officers exhibit negative attitudes towards obese students, whilst parents exhibit negative views of obese children in both subtle and visible ways. The impact of this stigmatization can be profound. In addition, childhood obesity is often linked to lower self-esteem and body esteem.²³ Childhood obesity is associated with a higher incidence of depression, and obese children often blame themselves for their weight and other's negative reactions to them.²⁴ Parents may also experience the stigma attached to their apparent inability to address their child's weight.

WHAT DO WE NEED TO LEARN?

- How can children learn to accept peers of all sizes and shapes?
- How can we disentangle concern over medical issues from character stereotypes?
- How can parents accept their obese children, including their bodies?
- How can parents support their obese children in coping with social stigma?

Summary of proposed societal level interventions

Change the food environment

- Remove non-nutritive snack foods and sugared drinks from schools.
- Discourage adults from feeding "poor" foods to children.
- Pressurise restaurants to provide 'kids meals' that are economical and nutritious.
- Change the role of the food industry.
- Pressurise the food industry to stop developing and advertising nutritionally poor 'children's foods'.

- Encourage the food industry to create healthful and affordable food products for children. Change the view of obesity. Add weight to the list of personal characteristics that are unacceptable reasons for discrimination. Include weight tolerance when we teach tolerance of other individual differences. Educate the public on how to accept obese people, while fighting the diseases linked to obesity.

Conclusions

About 40% of commercials shown during children's programmes are for food products, most of them high in fat or sugar. Exploitative advertising targeting the young is now witnessed at virtually every level of the fast food industry, as well as for commercial soft drink companies and industries involved in producing high fat or high refined carbohydrate snacks.²⁵ Strategies for encouraging health and preventing obesity at national level should be discussed, and the role of health ministries in developing public health, food and physical activity strategies should be underlined. In principle, schools provide an excellent setting for preventing obesity (Fig. 5)²⁶, and will also be the target of WHO's 'Health Promoting Schools Programme'.✶

References

1. Micic D. Obesity in children and adolescents – a new epidemic? Consequences in adult life. *J pediatr Endocrinol* 2001; 14:1345-1352.
2. Troiano F, Flegal K. Overweight children and adolescents: Description, epidemiology and demographics. *Pediatrics* 1998; 101 (Suppl):497-504.
3. Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China and Russia. *Am J Clin Nutr* 2002; 75(6):971-7.
4. Perusse L, Chagnon YC, Dionne FI, Bouchard C. The human obesity gene map: the 1996 update. *Obesity Res* 1997; 5:49-61.
5. Whitacker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Eng J Med* 1997; 337:869-873.
6. Maffei C, Talamini G, Tak L. Influence of diet, physical activity and parents' obesity on children's adiposity: A four-year longitudinal study. *Int J Obes Relat Metab Disord* 1998; 2: 758-764.
7. Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr* 1994; 59:955-959.
8. Seidman DS, Loo A, Gale R, Stevenson DK, Donon YL. A longitudinal study of birth weight and being overweight in late adolescence. *Am J Dis Child* 1991; 145:782-785.
9. Whitacker RC, Pepe MS, Wright JA, Seidel KD, Dietz WH. Early adiposity rebound and the risk of adult obesity. *Pediatrics* 1998; 101:E5.
10. Martorell et al. Birth weight and adult hypertension and obesity in women. *Circulation* 1996; 94:1310-1315.
11. Williams S, Davie G, Lam F. Predicting BMI in young

- adults from childhood data using two approaches to modeling adiposity rebound. *Int J Obes Relat Metab Disorders* 1999; 23: 348-354.
12. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult obesity. *Pediatrics* 1998; 101:518-525.
13. Obesity in Europe. International Obesity Task Force with the European Childhood Obesity Group. Copenhagen: IOTF 11 Sept. 2002. [<http://www.iotf.org/media/euroobesity.pdf>].
14. Freedman DS, Srinivasan SR, Berenson GS. Risk of cardiovascular complications. In: Burniat W, Cole T, Lissau I, Poskitt EME eds. *Child and adolescent obesity. Causes and consequences; prevention and management*. Cambridge, UK: Cambridge University Press; 2002:221-239.
15. Knip M, Naitinen O. Long-term effects of weight reduction on serum lipids and plasma insulin in obese children. *Am J Clin Nutr* 1993; 57(4): 490-3.
16. Epstein LH. Methodological issues and ten-year outcomes for obese children. *Ann NY Acad* 1993; 692:237-249.
17. Berkowitz RI, Wadden TA, Tereshakovec AM, Cronquist JL. Behaviour therapy and sibutramine for the treatment of adolescent obesity. *JAMA* 2003; 289:1805-1812.
18. WHO report: Obesity in young people. The coming crisis in public health. Edited by Lobstein KT, Baur L and Uany R. Published by IASO. 231 North Gower Street, London
19. Molnar D, Molecka-Tandera E. Drug therapy. In: Burniat W, Cole T, Lissau I, Poskitt EME Eds. *Child and adolescent obesity. Causes and consequences: Prevention and management*. Cambridge, UK: Cambridge University Press; 2002: pp 345-54.
20. Birch LL, Marlin DW. I don't like it; I never tried it: Effects of exposure on two-year-old children's food preferences. *Appetite* 1982;3:353-360.
21. Korvisto U, Fellerinjo J, Sjödén P. Relations between parental mealtime practices and children's food intake. *Appetite* 1994;22:245-257.
22. Kimm SY, Sweeney CG, Janosky JE, MacMillan JP. Self-concept measures and childhood obesity. A descriptive analysis. *J Dev Behav Pediatr* 1991;12:19-24.
23. Mendelsohn BK, White DK. Relation between body-esteem and self esteem of obese and normal children. *Percept Mot Skills* 1982;54:899-905.
24. Wallace WJ, Sheslow D, Hassink S. Obesity in children: A risk for depression. In: Williams CL. Eds. *Annals of the NI/ Academy of Sciences*. New York, 1993, pp 301-2.
25. Du Toit G, van der Merwe M-T. The epidemic of childhood obesity. *SAJM* 2003; 93:49-50.
26. Moschandrea J. Health and nutrition education in primary schools in Crete: changes in chronic disease, risk factors following a 6 year intervention programme. *Br J Nutr* 2002; 88(3):315-324.

Addendum

GUIDELINES AND RESOURCES

1. **USA:** The Maternal and Child Health Bureau, Health Resources and Services administration, Department of Health and Human Services '**Obesity Evaluation and Treatment: Expert Committee Recommendations**' Barlow SE, Dietz WH. *Pediatrics* 1998; 102(3).
2. **France:** Expert advisory group '**Childhood Obesity. Screening and prevention. Synthesis and recommendations**' [www.inserm.fr/servcom/servcom.nsf/7f476b2583842f98c12569b400384eee/6477a23a05c9289180256b7d004cda49/\\$FILE/Obesity%20Synthesis.pdf](http://www.inserm.fr/servcom/servcom.nsf/7f476b2583842f98c12569b400384eee/6477a23a05c9289180256b7d004cda49/$FILE/Obesity%20Synthesis.pdf).
3. **UK:** NHS Centre for reviews and dissemination '**The Prevention and Treatment of Childhood Obesity**' *Effective Health Care* 7 (6) 2002.
4. **UK:** Royal College of Paediatrics and Child Health, and National Obesity Forum. '**An approach to weight management in children and adolescents in primary care**' 2002 (available on http://www.rcpch.ac.uk/publications/recent_publications/Approach_2PAGES_TOGETHER.pdf). See also www.nationalobesityforum.org.uk
5. **SCOTLAND:** Scottish Intercollegiate Guidelines Network '**Obesity in children and young people. A national clinical guideline**' 2003 (available at <http://www.sign.ac.uk/pdf/sign69.pdf>). See also www.hebs.scot.nhs.uk/learningcentre/obesity/childhood
6. **ITALY:** Italian Society of Pediatric Nutrition '**Diet therapy for pediatric obesity**'. Consensus document.
7. **GERMANY:** Guidelines for assessment, treatment and prevention, of the German Society for Paediatrics and Adolescent Medicine, Adipositas (see www.a-g-a.de).