

Prevalence and Determinants of Childhood Obesity in Dhaka City

Rahman SMM^{1*}, Kabir I², Bhuyan MAH³, Akter BMD⁴, Hossain SM⁵

¹Academic Wing, Institute of Public Health, Dhaka, Bangladesh; ²Former Senior Scientist, ICDDR,B, Dhaka, Bangladesh; ³Former Director and Professor, Institute of Nutrition and Food Science, University of Dhaka, Dhaka, Bangladesh; ⁴Maternal and Child Health and Training Institute, Dhaka, Bangladesh; ⁵Former Director General, Directorate General of Health Services, Dhaka, Bangladesh

Abstract

Background: Obesity amongst the children is increasing worldwide at an alarming rate in both developed and developing countries. Obese children are at higher risk of developing coronary heart disease, non-insulin dependent diabetes, respiratory disease etc.

Methods: A case-control study, preceded by a cross sectional survey was conducted, aimed to estimate the prevalence rate and to identify the factors associated with the development of childhood obesity in Dhaka city. A multi stage probability proportionate to size (PPS) cluster sampling method was used to obtain the sample. To identify the obese children, a pre-tested questionnaire was used to collect data on age, weight and height among the randomly selected 5000 children of 2-10 years age group from 12 government primary and 23 private elementary schools, 4 hospitals, 8 health centres and 12 immunisation centres (on National Immunisation Days) from all the 12 thanas (civil administrative sub-districts) of Dhaka city. Survey included a medical history and physical examination to assess the eligibility of the subjects for the study.

Results: Of 5000 children, 380 (7.6%) were identified as obese using the criterion of weight for height >120% as a cut-off point. Obesity was positively correlated with the increase of age in both sexes ($r = 0.76$). Of all obese children, 216 (56.8%) were boys and 164 (43.2%) were girls. Prevalence of obesity was significantly higher among the boys than girls ($p=0.007$). The study was conducted among the 220 cases of 380 obese children ($Wt/Ht > 120\%$) and 220 randomly selected controls, matching age and sex, using a semi-structured questionnaire for identifying the factors associated with the development of childhood obesity. Information also collected from parents of both cases and controls. Family income ($p<0.001$) and expenditure on food ($p<0.001$) were significantly higher among the cases. Data showed that parental obesity was significantly associated with the obesity in children. There was an association between obesity of the children and parents' educational status ($p<0.001$). There was no difference in the working hours of parents outside households between the cases and controls. Energy balance was significantly higher among the cases. Dose response of energy balance shows, the estimated relative risk of obesity increases with higher levels of energy balance up to a maximum of odds ratio 3.41 ($p<0.001$). A significant difference ($p<0.001$) was found in hours of television-video viewing between the cases and controls.

Conclusion: Findings of this study show that the obesity among the children is caused by a positive energy balance over a considerable period, is related to environmental factors including energy intake, energy expenditure and other behavioural aspects. Appropriate interventions like behavioural change regarding energy intake and physical activity are thus recommended to address the childhood obesity- an emerging public health problem in Dhaka city.

Keywords: Childhood obesity, Physical activity, Energy balance, Television viewing

Introduction

Obesity is a form of malnutrition characterised by excessive fatness in the body - a detriment to good health and well being.^{1,2} Obesity is not a recent phenomenon. Its historical roots can be

traced to the Paleolithic era more than 25000 years ago. Clinical evidence of obesity can be dated as far back as Graeco-Roman times, but little scientific progress was made towards understanding the condition until the 20th century. Throughout most of the human history, weight gain and fat storage have been viewed as signs of health and prosperity.^{1,3,4}

*Correspondence: Shah Md. Mahfuzur Rahman, Institute of Public Health, Mohakhali, Dhaka, Bangladesh; e-mail: smahfuzbd@gmail.com

Obesity is a critical health concern among children. Childhood obesity has reached epidemic proportions globally, both in developed and developing countries, even in those countries where deficiency diseases represent a severe public health problem.^{3,5,6} In many countries, it poses an urgent and serious challenge. The Sustainable Development Goals, set by the United Nations in 2015, identify prevention and control of non communicable diseases as core priorities. Among the non communicable disease risk factors, obesity is particularly concern and has the potential to negate many of the health benefits that have contributed to increased life expectancy.⁷

In 2014, an estimated 41 million children under 5 years of age were affected by overweight or obesity globally. Of them, 48% overweight lived in Asia and 25% in Africa. In Africa, the number of children who are overweight or obese has nearly doubled since 1990, increasing from 5.4 million to 10.3 million.⁸ In USA, it was estimated in a nationally representative study, the prevalence of obesity was 17% among the 2-19 years age group.⁹

In Bangladesh, a study was conducted in January, 1997 in a school and a MCH clinic in Dhaka city, among 316 the children of 2-10 years age group, the prevalence of obesity (Wt/Ht >120%) was to be found 13%. The study also revealed that obesity was positively correlated with increase of age and family income.¹⁰ A countrywide cross-sectional epidemiological study shows that among the 6-15 years old children both from urban and rural areas 3.5% were obese and 9.5% were overweight.¹¹

Obesity is a recognized disease in its own right and consequently causing a number of chronic, non-communicable and life threatening diseases including noninsulin dependent diabetes mellitus, cardiovascular diseases, gall bladder diseases and hormone sensitive and gastrointestinal cancers as well as high risk for some non fatal conditions like back pain, arthritis, infertility and poor psychosocial functions including social isolation, peer problems etc.^{3,5,12-20} The most public health important consequence of childhood obesity is that it commonly tracks into adulthood and persistence rises with age among obese children with an increased risk for poor health

outcomes.^{3,12,21,22} It is estimated that 70% of obese children become obese adult.¹² The economic cost of obesity is an important issue for health care providers and policy makers. In several developed countries, the cost of obesity has been assessed, and it was 2-7% of total health care cost.

A number of aetiological factors like genetic, life styles, emotional over eating, indulgence, parental neglect, medical factors, geographical location, seasonality, socio-economic status, demographic factors have been identified as in different epidemiological studies.^{3,5,12,23-26}

Genetic factors play an important role in childhood obesity.^{27,28} It is correlated with family histories of obesity.²⁹ Though fatness does run in families, but the genetic component does not follow simple Mendelian principles and the influence of the genotype on the aetiology of obesity may be attenuated or exacerbated by non-genetic factors.³⁰ The relative importance of inherited factors in the development and maintenance of obesity is still being actively debated. The discovery of Leptin (167- amino acid protein of 16 k Da) has led to a renewed interest in genetic and metabolic influences in the development of obesity -a fat derived hormone may be the long sought after 'lipostat' factor postulated to govern energy balance a negative feedback loop originating in adipose tissue and acting on centers in the brain.^{3,31} Prenatal malnutrition particularly in early gestation is associated with obesity in later life.^{32,33} Studies indicated that low birth weight is associated with obesity in later life.³⁴⁻³⁷ There are many debates on sex distribution of obesity. Study shows that females are more obese than males but other studies showed that there was no significant difference in childhood obesity regarding sex distribution.^{3,10,32} Higher prevalence of obesity in children occurred with increase of age.^{10,38-40} Studies suggest that there are critical periods during childhood that influence development of obesity including gestation and early infancy, the period of adiposity rebound that occurs between 5-7 years of age.^{33,41} It is revealed that the preadolescent period between 7 and 11 years attracts particular attention because juvenile obesity may begin during this interval of accelerated growth.⁴²

The prevalence of obesity declines with increasing family size. It is higher among single children.³⁵ Birth order has no consistent influence on prevalence of obesity, although some data suggest that greater prevalence among younger children of large families.⁴³ Family structure or family life is closely associated with the development and maintenance of childhood obesity.²⁶ Dietz and Gortmaker found that the prevalence of obesity was associated with higher income levels.²⁴

Simply, obesity is a result of positive energy balance over a considerable period. Body weight is primarily regulated by a series of physiological process but is also influenced by external societal and cognitive factors.³ Rolland and Bellisle reported that there was no relationship between body mass index (BMI) and skinfold thickness with energy intake in 7 to 12 y old children.⁴⁴ Sunnegardh et al also showed no relationship between skinfold thickness and energy intake in 8-y old boys and girls.⁴⁵ Studies that compared energy intake of lean and obese children have generally not shown that obese children have higher energy intakes than do lean children.⁴⁶⁻⁵⁰ The recent worldwide increase in the prevalence of childhood obesity may be due impart to a decrease in children's physical activity.⁵⁴ It is reported that obese children exhibit decreased performance during exercise compared with normal body weight peers.⁵² Other studies revealed that obese children expend more energy for resting metabolic rate and physical activity than their counterparts.^{46,47} Studies show that lean children are more physically active where as others do not show a significant difference.^{46,53,48,50,54}

This study was aimed to explore in depth evidences on the prevalence of childhood obesity- to provide a baseline data and to identify the factors contributing to the problem and the pertinent issues requiring further research and to develop recommendations to alert nutrition and health experts to consider it as a public health problem.

Materials and Methods

This case-control study was preceeded by a cross sectional survey, conducted among 2-10 years age group in Dhaka city during 1999-2001. This age group was considered, as the children's age less than 2 years and above 10 years may be heavier and fatter for development and physiological

reasons.⁵¹ A total of 12 Government primary schools, 23 Private elementary schools, 4 Hospitals, 8 Maternal and Child Health Centres and 12 Immunization Centers (on National Immunization Days) in all 12 thana (Civil administrative-subdistrict) of the Dhaka city were the study sites. Referring to the study and considering the prevalence rate 10% or less, using the Inverse sampling method for rare items, 5000 children of 2-10 years age group was surveyed to obtain the cases.^{10,52} Child suffering from any illness for last one month, handicapped children, child taking drug like steroid, cyproheptadine, phenothiazine etc causing weight gain, and parents unwilling for invasive investigations were excluded from the study. It included a medical history and physical examination to assess the eligibility of the subjects for the study. The study was ethically cleared by the Bangladesh Medical Research Council.

A three stage probability proportionate to size (PPS) cluster sampling method was used to obtain the representative sample of 5000 children of 2-10 years age using the current census data. The design provided a systematic sampling based on the current population census of 12 thanas of Dhaka city.^{53,54-58}

Of 5000 children, 380 (7.6%) were identified as obese using the criterion of weight for height >120% of NCHS-WHO reference as a cut-off point. An equal number of non obese children (<120% of NCHS-WHO reference as controls were selected randomly matching age, sex and stratum of sampling.³³

Due to shifting of accommodation including transfer of place of works of parents, unwillingness to participate in the study by the parents and illness of either parents and/or children (during case- control study period) a total of 160 obese children could not be included in the study. Even with this exclusion, the total number of cases and controls were within statistically sound sample size [ci =0.05 two sided, B = 0.20, Po 0.15 and R=2 and the estimated sample size was 207 using table for estimation sample size for case-control study.⁵⁹ Finally, the case control study part of the study was conducted among the 220 cases and 220 controls.

Data were collected at the school or laboratory or at prefixed places. For this, parents were communicated through school authority or by the investigator himself. A semi-structured

questionnaire was used for identifying the factors associated with the development of childhood obesity. Data on socio-economic, demographic, anthropometry, 24 hours dietary recall and 24 hours physical activity recall were collected. Information was also collected from the parents of both cases and controls.

Data collection: Socio-economic and demographic information: Age of the child as years and months (eg. 6 years 6 months) have been recorded where designated month is the last completed month.⁶⁰ Later on, the age of the both cases and controls were checked by the parents. Information on demographic characteristics like birth order, family structure and family size and other family information like monthly total family income, monthly family expenditure on foods were collected through interviewing the parents.

Measurement of height and weight: Height and weight were measured to the nearest 0.5 cm and 0.1 kg respectively following procedures described elsewhere.^{60,63,64}

Assessment of energy intake and expenditure: A 24-hours dietary recall was used to assess the dietary intake of the both cases and controls. The parents and their child were interviewed together to complete the recall all foods and beverages consumed by the child during the previous 24 hours- a typical day of a week, as described elsewhere.^{60,63,64}

Estimation of total energy expenditure: A simple, pictorial chart, depicting 24-hour physical activities commonly performed by children was used to assist the recall. The participants checked off the amount of daily time spent on various activities including, sleeping, brushing teeth, washing hands and mouth, breakfast, bathing/showering, dressing, walking, climbing/stepping down stairs, sitting in class room, reading, writing sports watching television etc. which were recorded using a semi-structured questionnaire. Total energy expenditure of 24 hours was calculated according to the published literatures.⁶⁴⁻⁶⁸

Estimation television video viewing hours: Television and video viewing was measured among the both cases and controls. They were asked the time spent on watching television, movies, videos each day of previous week-a typical week. They also asked to recall dividing a day into 4 quarter. To assist the recall, the

programmes of different TV channels published in local newspapers were shown. Parents were also asked to assist the recall. On the basis of daily TV video viewing weekly total hours spent on TV video was calculated.⁶⁹⁻⁷¹

Data analysis: All statistical analyses were done using SPSS PC. Chi-square tests were done to see the association between categorical variables. Comparisons between groups were performed by t tests and non-parametric, Mann-Whitney tests. Correlations between continuously distributed variables were assessed by using Spearman correlation analyses. Logistic regression was used to examine factors associated with obesity. Other appropriate statistical tests were done for analyses of the data. Two tailed *p* values <0.05 were considered significant.

Results

This study was conducted among 5000 children of 2-10 years age group in Dhaka city. Of them, 2510 were boys and 2490 girls. Of the total 5000 study population, 380 were identified as obese, using cut off level weight for height above 120% of NCHS WHO reference. But using weight for height Z scores equal or above +2SD, 280 were identified as obese (table I).

Table I: Childhood obesity by different Indicators

Indicator	Obesity		Percent of total population (5000)
	Cut off value	No. of obese children	
Wt/ht	>120	380	7.6%
WHZ	>2.0	280	5.6%

Of the total 380 obese children, only 14 were stunted, 5 were boys and 9 were girls. Chi square test shows, stunting were negatively associated (*p*<0.002) with obesity among the study population (table II).

Table II: Distribution of stunting and obesity

Sex	Stunting	Obese	Non obese	Total	Total
Boys	Stunted	5	149	154	2510
	Not Stunted	210	2145	2356	
Girls	Stunted	9	180	189	2490
	Not Stunted	155	2146	2301	
Total		380	4620	5000	5000

Out of 440 study children (220 cases and 220 controls), single age distribution in 4-10 years children were 6, 7, 13, 25, 44, 90 and 35 in each group. The mean age in each group was 8.3± 1.4

years. Out of total 440 study children, 220 were cases (standard weight for height >120%) and 220 were controls (standard weight for height <120%). Of them, 149 were boys and 71 were girls in each group. Out of 220 cases, 113 were 1st issue, 768 2nd issue, and 39 were 3rd and above issue, But among 220 controls 1st, 2nd and 3rd and above birth order were 106, 58, 56 respectively. No significant ($\chi^2 = 4.060, p = 0.131$) association was to be found between birth order and obesity of the children.

Among the study subjects, 74 cases and 59 controls were from the family having members up to 4, 86 cases and 106 controls from the family of 5-6 members, 35 of cases and 31 controls were from the family of 7-8 members and, of family size of above 8, 25 were cases and 24 were controls. There was no significant association ($\chi^2 = 4.038, p = 0.257$) between family size and childhood obesity.

Considering family structure, among the cases (n=220) 166 from nuclear and 54 joint family and, among the controls (n=220), 177 from nuclear and 50 from joint family. It is evident that obesity of children was not significantly ($\chi^2 = 0.201, p = 0.654$) associated with family structure.

Out of 220 parents of cases, 10, 57, 63 and 90 had have education, below Secondary School Certificate (SSC) level, Secondary and Higher Secondary School Certificate (HSC) level, Graduation level and Masters and above respectively. Among the 220 parents of controls, 57 were below SSC, 80 were SSC and HSC holders, 46 were graduates and 27 were Masters and above. It is evident from the table, with increase of fathers' level of education, obesity of the children also increased. So, it may be interpreted, there was a significant association ($\chi^2 = 61.60, p < 0.001$) between fathers' education level and childhood obesity.

It was to be found that with increase of level of mothers' education, obesity of children also increased. There was a significant ($\chi^2 = 56.246, p < 0.001$) association between mother's education level and childhood obesity. It is evident that, out of 51 children of professionals, 70.6% were obese; followed by 63.3% obese children among 128 children of businessman parents, out of 134 children of service holder parents 54.5% children were obese ($\chi^2 = 57.48, p < 0.001$).

Among the mothers of cases (220), 185 were housewives and 35 were nonhousewives (day labourer, sweepers, hawkers, service holders, professionals, businessman etc.) No statistical significant ($\chi^2 = 2.4, p < 0.12$) association was to be found between mothers' occupation and childhood obesity.

Of 114 fathers of cases worked more than 10 hours daily outside household against 122 fathers of controls. It is evident from the Chi-square test, no significant ($\chi^2 = 0.585, p > 0.444$) association between fathers working hours outside and obesity in children. Of 186 mothers of cases worked less than 3 hours in a day outside household against 196 mothers of controls. Out of mothers of 58 children, 34 mothers of cases worked more than 3 hours outside household daily against 24 mothers of controls. Chi-square test shows, no significant ($\chi^2 = 1.986, p = 0.159$) association between mothers working hours outside household and obesity in children (table III).

Table III: Difference of parents' working hours outside households among cases and controls

Parents' working hrs	Subject	Mean	"t" value	p value
Fathers' hrs outside Household	Cases (n=220)	10.93±2.34	1.665(Special "t" test as F Significant)	0.097 Not Significant
	Controls (n=220)	11.11±2.13		
Mothers' hrs outside Household	Cases (n=220)	1.18±2.91	0.83 (Special "t" test as F Not Significant)	0.407 Not Significant
	Controls (n=220)	0.76± 0.76		

The mean working hours in a day outside households, were 10.93 hrs and 11.11 hrs of fathers' of cases and controls respectively. Independent sample "t" test shows, no significant difference of working hours between the two groups of fathers ($p = 0.47$). Similarly, no significant difference of working hours outside household of mothers of cases and controls was to be found. So, it may be concluded here, parents' working hours outside household has no significant effect on obesity in children in the study population.

With the increase of monthly family income level, the number of obese children also increased significantly (χ^2 for trend = 36.48, $p < 0.001$). With the increase of monthly family expenditure on food, the number of obese children also increased. There is a significant (χ^2 for trend = 39.82 $p < 0.001$) association between monthly family expenditure on food and obesity among children (table IV).

Table IV: Difference of monthly family income and monthly family expenditure on food between cases and controls.

Variables	Subjects	Mean ± SD	t value	p value
^a Monthly family income (TK)	Case (n=220)	23720±17576	t= 4.752 Special 't' test as F is significant	p <0.001
	Control (n=220)	16652±13338		
^b Monthly family expenditure (Tk.)	Case (n=220)	8678±4316	t=5.489 (Simple 't' test as F not significant)	p <0.001
	Control (n=220)	6616±3531		

a) Non parametric test Mann — Whitney test (mean rank: case 260.65 and control 180. 35) *p*<0.001

b) Non parametric test: Mann-Whitney test (mean rank: case 260.33 and control 180. 67) *p*<0.001

The mean difference of monthly family income, monthly per capita income and monthly family expenditure on food between cases and controls are statistically significant (*p*<0.001).

Energy intake among the obese children was found to be significantly higher than the non obese counterpart. Perversely, the energy expenditure and physical activity level were found to be significantly lower among the obese children (table V).

Table V: Energy intake, energy expenditure, physical activity level among obese and on obese children

Variable	Obese (n=220)	Non obese (n=220)	p value
Energy intake (mean, kcal/day, for both sex)	2056±751	1558±529	<i>p</i> <0.001
Energy expenditure (mean, kcal/day, for both sex)	1868±313	1495±200	<i>p</i> <0.01
Physical activity level (mean, for both sex)	1.35±0.14	1.40±0.09	<i>p</i> <0.001

Furthermore, regarding the energy balance, taking as the reference group children who had no energy balance, the estimated relative risk of obesity increases with higher levels of energy balance upto a maximum of odds ratio 3.41 ($\chi^2 = 46.38, p < 0.001$).

The weekly total hours of television and video viewing of children showed that with increase hours of TV video viewing, number of obese children also increased. Chi-square test shows there was a statistically significant ($\chi^2 = 13.13, 86S, p = 0.004$) association between duration of TV video viewing and childhood obesity (table VI).

Table VI: Distribution of hours of TV-Video viewing and childhood obesity

Weekly hrs of TV-video viewing	Cases (n=220)	Controls (n=220)	Total (N=440)
Up to 7	20	33	53
8-14	76	93	169
15-21	85	76	161
22-above	39	18	57
Total	220	220	440

The mean hours of TV-Video viewing in a week was 16.31 ± 6.37 hrs and 14.01± 5.89 hrs among cases and controls children respectively. Cases enjoy TV-Video more hours than their controls, which was statistically significant (table VII; *p*<0.001).

Table VII: Difference of mean TV-Video viewing among cases and controls

	Cases/ Controls	Mean ±SD (Hrs)	't' value	p value
Weekly TV-Video Viewing	Cases (n=220)	16.31±6.37	3.929 (Sample "t" test as F Not Significant)	<i>p</i> < 0.001
	Controls (n=220)	14.01±5.89		

Out of 46 obese fathers (of both obese and non obese children), 34 having obese children, 12 non obese children. Furthermore, out of the rest 394 fathers which were non obese, 186 having obese children. Chi square test shows, that there was a significant ($\chi^2 = 10.70625, p < 0.001$) association between fathers obesity and child's obesity.

On the other hand, out of 146 obese mothers (of both obese and non obese children), having 96 obese children and 50 non-obese children. Furthermore, out of 294 non-obese mothers, 124 having obese children. It is evident from the Chi square test, there was a significant ($\chi^2 = 20.75762, p < 0.001$) association between mothers obesity and obesity in children (table VIII).

Table VIII: Distribution of fathers' obesity (using wt/ for ht %) and childhood obesity

Father's obesity	Child obesity		
	cases (n=220)	Controls (n=220)	Total (n=440)
Obesity (wt/ht>120%)	34	12	46
Non obesity (wt/ht<120%)	186	204	394
Total	220	220	440

Using BMI, it was to be found that obese mothers (*p*<0.001) have more obese children than the obese fathers (*p*<0.01). But both parents obese having non-significant number of obese children (table IX, *p*<0.08).

Table IX: Distribution of mothers' obesity (using wt/ht %) and childhood obesity

Mother's obesity	Child obesity		
	cases (n=220)	Controls (n=220)	Total (n=440)
Obesity (wt/ht>120%)	96	50	146
Non obesity (wt/ht<120%)	124	170	294
Total	220	220	440

Discussion

The prevalence of obesity reported in this sample of Dhaka city was 7.6%, using weight for height more than 120% of NCHSIWHO reference. There was a significant difference ($p<0.007$) between the prevalence of obesity between boys (56.8%) and girls (43.2%). This difference is similar to the data reported in Italian and Malaysian population.^{73,74}

In this study, highest prevalence (14.0%) of obesity was found in 9-10 years old children, but no statistically significant difference was to be found between boys and girls ($p=0.067$) in this subgroup, which is inconsistent with the finding of Kotani et al among the Japanese children.⁷⁵ Data of this study show, prevalence of obesity among the study population increased with age ($r=0.76$, chi-square for age trend 117.61 $p<0.001$). The trend was higher among the boys (χ^2 for trend 69.03) than girls (χ^2 for trend 44.25). The age trend of this study is consistent with the other studies.^{5,10,24,38,39} The mean age of the obese children was 8.3 years. The result of the present study is consistent with the study of Wilkinson et al.³⁵

Among the 5000 study children of 2-10 years age group, the mean weight and height of the 6-10 years subgroup were 24.63 ± 7.18 kg and 126.03 ± 9.74 cm respectively. Childhood nutritional stunting, an indicator of chronic undernutrition has been suggested as one factor contributing to high rates of obesity in developing countries because of the observed positive association between stunting and adolescent and adult obesity.⁷⁵⁻⁸³ In this study, the association between obesity and stunting in children was not statistically significantly. Amongst the 5000 study children, 2510 were boys. Of the total boys, 154 were stunted and only 5 (3.25%) boys of them were obese, against 9 (4.76%) girls were obese amongst 189 stunted girls. Sawaya et al found that stunted girls appeared to be at even greater risk of

obesity than boys, with a 35% prevalence of obesity compared with 11% in stunted adolescent boys. In the present study, though the association between stunting and obesity is not significant but it was to be found that girls were more obese than their stunted boys counterpart, which is similar with findings of Sawaya et al.⁷⁸ Hoffman reported that stunting increases the risk of obesity in developing countries, particularly in girls and women.⁸⁴

Both birth order and family size have been related to childhood obesity. But in this study no significant association was to be found between childhood obesity and birth order or family size. Finding of this study is inconsistent with other studies in which the prevalence of obesity is high among single children and declines with increasing family size.^{35,85} Birth order has no consistent effect on prevalence of obesity although some data suggest greater prevalence among younger children of large families.^{43,86,87} Stettler et al found that first-born status independently associated with the development of increased adiposity.⁸⁸ But contradictory results were found in earlier studies conducted in European and African populations.^{86,88,89} It was observed that the risk of obesity in 10 years old girls in USA decreased as the number of siblings increased.⁸⁸ Other studies show that, some one third of obese children were only children another one third were last born.^{90,91} Wilkinson found that being an only child was one of the common 'at risk' factors for obesity.³⁵ The characteristic of family structure or family life are closely linked to the development and maintenance of obesity in children.²⁶ In this study, obesity in children was not significantly associated with family structure, which is inconsistent with the findings of Wilkinson et al.³⁵ Intact parental relationships are also important for the growth and development of the children.

Despite the frequent use of education as a determinant of socio economic class, its effect on rates of obesity had rarely been examined as independent variable. No available studies establish the relation of parental education to prevalence of obesity among children.⁴³ In this study, there was a significant association between obesity in children and parents education level.

With increase of parents' education level, obesity also increased among children. Occupation and education levels are related to income level. Most of the parents with higher education are engaged in high earning occupation, which may influence on family expenditure on food.

In addition to education, income, occupation, place of resident etc. are the determinants of socio-economic status. In developed countries, socio-economic status is inversely correlated with the obesity. In USA, the overweight and obesity epidemic is disproportionately higher in children from low-income and education, and higher unemployment households. But in developing countries, it is positively correlated.^{3,92,22}

In this study, childhood obesity is significantly associated with the higher level of family income (χ^2 for trend 36.48 $p < 0.001$). Results also show, the monthly family expenditure on food (χ^2 for trend 39.82 $p < 0.001$) is directly associated with the obesity among the children.

The finding of this study regarding income level was consistent with the other studies but inconsistent with the findings revealed by other authors.^{3,93-95} It is evident that with increase of income level, the expenditure on food also increased, which eventually causes higher energy intake. It may be mentioned here, parents' occupation also having influence on childhood obesity in this study. Children of parents having occupation of higher income are more obese than their low earning occupation counterpart. Despite economic data from several other studies, no consistent class trends are apparent.^{37,96,97} Socio-economic status of the families of obese children was higher in contrast to their non-obese counterpart, which is similar to other developing countries where childhood obesity is emerging or increasing due to socio-economic transition. Populations experiencing rapid socioeconomic and/or nutritional transitions appear to be at particular risk.⁷

Body weight is dependent on the balance between energy intake, in the form of food and drink, and energy expenditure. Daily energy expenditure consists of resting energy expenditure, the energy required to metabolize food (thermic effect of food), and energy expended as a result of activity.

When energy intake and expenditure are in balance, weight remains stable. A net excess in energy, whether through greater intake of lesser expenditure leads to weight gain. In children, some of this extra energy may be used for linear skeletal growth; but in both children and adults net excess energy intake leads to increase in both lean body mass and adipose tissue.

Increase in positive energy balance being closely associated with the lifestyle adopted and the dietary intake preferences.⁹⁸ In this study, energy intake was significantly ($p < 0.001$) higher among the obese children (2056±751 Kcal/day.) than the non obese counterpart (1558±529 kcal/day). But there was no statistically significant difference between boys and girls regarding calorie intake for both cases and controls. Physical activity level is significantly higher among the non obese children against their obese counterpart (1.40±0.09 Vs 1.35±0.14). The obesity epidemic is related to lower levels of PA among children in comparison with the evidence-based recommendations.²² The authors described elsewhere regarding the energy intake and expenditure and physical activities.⁶⁴

Researchers have identified television viewing as one of the prominent potential modifying factors that can lead to obesity in childhood.²² Each hourly increment of television viewing by adolescents has been associated with a 2% increase in the prevalence of obesity.⁹⁹ In this study, the television and video viewing is significantly associated with the obesity among the children. Obese children viewed television significantly ($p < 0.001$) more hours than controls (16.31 Vs 14.01 hrs). The mean duration of television exposure was 16.31 hours per week among the obese children, which was almost equal to children of India as reported by Gupta et al. Similar agreement with a number of studies.^{99,69} Dietz and Gortmaker reported that the association between television viewing and obesity was statistically significant and persisted when other potential variables were controlled.⁹⁹ Multiple cross sectional surveys have suggested that as the number of hours of media usage increases, body fat percentage and the risk of overweight also increases in a dose-response

manner.¹⁰⁰⁻¹⁰³ Television viewing may be related to more dietary intake including increased opportunity for snacking, less physical activity, metabolic rate, or some combination of these.¹⁰⁴ Tanasescu et al have identified from their case-control study among Puerto Rican prepubertal children that hours of daily TV viewing was associated with obesity, which is consistent with the present study.¹⁰⁵

Obese parents are more likely to have obese children and are independent of the sex of the parent or child. This pattern exists even when children are reared apart from their biological parents. Parents provide both the genes and environmental context for their children's growth and development and familial patterns of adiposity are the results of gene-environment interaction.^{105,106} In this study, parental obesity was significantly associated with obesity in children. Maternal obesity was more significantly ($p < 0.001$) associated than the fathers' obesity ($p < 0.01$). Findings of this study are consistent with the findings of other studies.^{107,108} It is widely accepted that parental behaviors and practices shape many aspects of children's development.⁴⁶ The parents' food preferences, the quantities and variety of foods in the home, the parents' eating behaviour, and the parents' physical activities patterns work in concert to establish an emotional environment and weight outcomes. In this study, most of the mothers were housewives and primary caretakers of the children, typically provide with structure for meals, by offering foods, and others and by using child feeding practices that provide information to the child about how much and what to eat and mothers' behaviours, life styles etc. influence children. Due to this maternal influence, mothers' obesity is more significantly associated with children obesity. But children of both obese parents were not statistically significant ($p = 0.08$) which is inconsistent with the study of Lake et al. Lake et al reported that children with two obese parents are more likely to be obese compared with children with only one obese parent, who in turn are more likely to be obese than children without obese parent. The tracking of obesity into adulthood is stronger in children when both parents are obese.

Logistic regression was used to examine the factors associated with the obesity in the study population. Of the factors significantly associated with the obesity in children as indicated earlier, the regression analysis shows, the significant association of age of the children, energy intake and energy expenditure. Energy expenditure ranked the highest significant level.

The findings of this study support the hypothesis that socio-economic status, dietary habit and life style factors are associated with the development of obesity in children.

Conclusion

The findings of this study indicate that the childhood obesity is an emerging health problem in Dhaka city, where it coexisted with the undernutrition. Results show parental education level, occupation, family income and family expenditure on food are positively correlated with the obesity in the children. The positively associated factors are widely accepted components and collectively express as the composite index of socio-economic status.

There was a significantly higher energy balance existed among the obese children. Based on the findings of the study, that childhood obesity is prevailing in the families those are passing a rapid socio-economic transition which led to an environment that promotes a sedentary life styles and consumption of high fat, energy dense diets. So, optimum energy intake and expenditure should be considered for a healthy and economically productive life for a better tomorrow. Finally, it may be concluded that as no data are available regarding prevalence and determinants of childhood obesity in Dhaka city during the nineties of the last century, findings of this study will provide an baseline data for the researchers and the policy makers.

Acknowledgements

Authors gratefully acknowledge the financial supports of the Bangladesh Medical Research Council (BMRC) for this study. Authors are also thankful to the children and their parents and the authorities of educational institutions and health facilities.

Conflict of interest: Authors declared no conflict of interest.

References

1. Poleman CM, Packerpaugh NJ. Nutrition Essentials and Diet Therapy. 6th ed. Philadelphia: W B Saunders Company. 1991; 2:13-29.
2. Pi-Sunyer FX. Obesity~: In: Shills M E, Young V R eds. Modern Nutrition in Health and Disease. 7 ed. Philadelphia: Lea and Febiger. 1988.795-8 16.
3. World Health Organization. Obesity: Preventing and managing the global epidemic. WHO Technical Report Series 894. Geneva: WHO, 2000.
4. Shetty PS. Obesity in children in developing societies Indicator of Economic Progress or a Prelude to a Health Disaster? [Editorial]. Indian Pediatrics. 1999; 36:11-15.
5. Steering Committee. The Asia Pacific perspective: Redefining obesity and its treatment. Health communications Australia Pty. Limited. 2000:1-56.
6. Florencio TM, Ferreira HS, de Franca AP, Cavalcante JC, Sawaya AL. Br J Nutr. 2001; 86:277-84.
7. [WHO. Report of the commission on ending childhood obesity. World Health Organization 2016. Geneva, Switzerland] [UNICEF, WHO, World Bank. Levels and trends in child malnutrition: UNICEF-WHO-World Bank joint child malnutrition estimates. UNICEF, New York; WHO, Geneva; World Bank, Washington DC:2015.]
8. Ogden CL, Carroll MD, Lawman HG, Fryar CD, Kruszon-Moran D, et al. (2016) Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. JAMA. 315: 2292-2299.
9. Rahman SMM, Akter BMD, Siddiqui MZA, Rashid M. Prevalence of childhood obesity in Dhaka city. Mymensingh Medical Journal. 1998; 7:3-6.
10. Bulbul T, Hoque M. Prevalence of childhood obesity and overweight in Bangladesh: findings from a countrywide epidemiological study. BMC Pediatr. 2014; 14:2-8.
11. Mellm L. To: President Clinton Re: Combating childhood obesity. J Am Diet Assoc. 1993; 93: 265-66.
12. Power C, Lake JK, Cole TJ. Body mass index and height from childhood to adulthood in the 1958 British birth cohort. A. J Clin Nutr. 1997; 66:1094-101.
13. Bronner YL. Nutritional status outcomes for children: Ethnic, cultural, and environmental factors. J Am Diet Assoc. 1996; 96: 89 1-900,903.
14. Das~upta 5, Hazra SC. The utility of waist circumference in assessment of obesity. Indian Journal of Public Health 1999; 4:132-135.
15. Solomon CG, Manson JE. Obesity and mortality: a review of the epidemiologic data. Am J Clin Nutr. 1997; 66 (suppl):1044S-50S.
16. Poskitt EME Management of childhood obesity. Indian J Pediatr. 1988; 55: 470-78
17. Zack PM, Harlan WR, Leaverton PE, Comom-Huntley J. A longitudinal study of body fatness in childhood and adolescence. J Pediatr. 1979; 95: 126 - 130.
18. de Onis M, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. Am J Clin Nutr. 2000; 72:1032-9.
19. Katz ES, D'Ambrosio CM (2010) Pediatric obstructive sleep apnea syndrome. Clin Chest Med. 31: 221-234
20. Maffei C, Tatro L. Long term effects of childhood obesity on morbidity and mortality. Horm Res. 2001; 55 (suppl 1):42-5.
21. Williams SE, Greene JL. Childhood Overweight and Obesity: Affecting Factors, Education and Intervention. Journal of Childhood Obesity. 2018; 3: 1-7.
22. Goran MI, Kaskoui M, Johnson R, Martinez C, Kelly B, Hood V. Energy expenditure and body fat distribution in Mohawk children. Pediatrics. 1995; 95: 89-95.
23. Diez WH, Oortmaker SL. Factors within the physical environment associated with childhood obesity. Am J Clin Nutr. 1984; 39: 619 -24.
24. Waxman M, Stunkard AJ. Calorie intake and expenditure of obese boys. J Pediatr. 1980; 96:187- 193.
25. Lissau I, Sorensen TIA. Parental neglect during childhood and increased risk of obesity in young adulthood. Lancet. 1994; 343: 324 - 327.
26. Boyuchard C. Current understanding of the etiology of obesity: genetic and nongenetic factors. Am J Clin Nutr. 1991; 53 (suppl):1561S-65S.
27. Gain SM, Bailey SM, Solomon BA. Effect of remaining family members on fatness prediction. Am J Clin Nutr. 1981; 34:148.
28. Anavian J, Brenner DJ, Fort P, Speiser PW. Profiles of obese children presenting for metabolic evaluation. J Pediatr Endocrinol Metab. 2001; 14:1 145-50.
29. Jebb S A. Aetiology of obesity. In: Finer N. ed. Obesity. Br Med Bull. 1997; 53: 264-85.
30. Wilding J, Widdowson P, William G. Neurobiology: In: Finer N. ed. Obesity. Br Med Bull. 1997; 53: 286-306.
31. Ravelli GP, Stein Z, Susser M. Obesity in young men after famine exposure in utero and early infancy. N Engl J Med. 1976; 295: 349-53.
32. Dietz WH. Critical periods in childhood for the development of obesity. Am J Clin Nutr. 1994; 59:955-9.

33. Somerville SM, Rona RI, Chinn S. Obesity and respiratory symptoms in primary School. *Arch Dis Child.* 1984; 59:94044.
34. Wilkinson PW, Parkin JM, Pearlson J, Philips PR. Obesity in childhood: A community study in Newcastle Upon Tyne. *Lancet.* 1997; Feb 12:350-52.
35. Barker M, Robinson S, Osmond C, Barker DJ. Birth weight and body fat distribution in adolescent girls. *Arch Dis Child.* 1997; 77: 38 1-83.
36. Kuh D, Hardy R, Chaturvedi N, Wadsworth ME. Birth weight, childhood growth and abdominal obesity in adult life. *J obes Relat Metab Disord.* 2002; 26:40-7.
37. Tiwary C Ivi, Holguin AH. Prevalence of obesity among children of military dependents at two major medical centers. *Am J Public Health.* 1992; 32:354-357.
38. Gortmaker SL, Dietz WH, Sobol AM and Welder CA. Increasing pediatric obesity in the United States. *Am J Dis Child.* 1987; 141: 535-540.
39. Gutierrez-Fisac JL, Regidor E, Rodriguez C. Economic and social factors associated with body mass index and obesity in the Spanish population aged 20-64 years. *Eur J Pub Health.* 1995; 5:193-198.
40. Power C, Parsons T. Nutritional and other influences in childhood as predictors of adult obesity. *Proc Nutr Soc.* 2000; 59:267-72.
41. Mullins AG: The prognosis in juvenile obesity, *Arch Dis Child.* 1958; 33:307.
42. Dietz WH Jr, Gordon JE, Obesity in infants, children and adolescents in the United States. II. Causality. *Nutrition Research.* 1981; 1:193-208.
43. Rolland Cachera MF, Bellisle F. No correlation between adiposity and food intake: why are working class children fatter? *Am J Clin Nutr.* 1986; 44:779-87.
44. Sunnegradh J Bratteby LE, Hagman U, Samuelson G, Sjolín S. Physical activity in relation to energy intake and body fat in 8 and 13 year of children in Sweden. *Acta Paediatr Scand.* 1986; 75:955-63.
45. Johnson ML Burke BS, Mayer J. Relative importance of inactivity and overeating in the energy balance of obese high school girls. *Am J Clin Nutr.* 1956; 4:37-44.
46. Stefanik PA, Heald FP, Mayer J. Caloric intake in relation to energy output of obese and non-obese adolescent boys. *Am J Clin Nutr.* 1959; 7:55-61.
47. Bradfield RB, Paulos J, Grossman L. Energy expenditure and heart rate of obese high school girls *Am J Clin Nutr.* 1971;24:1482-8.
48. Durin JVGA, Lonergan ME, Good J, Ewan A. A cross sectional nutritional and anthropometric study with an interval of 7 years on 611 young adolescent schoolchildren *Br J Nutr.* 1974; 32: 169-79.
49. Wilkinson PW, Parkin JM, Perlson G, Strong H, Sykes P. Energy intake and physical activity in obese children *Br Med J.* 1977; 1: 756.
50. Mahan LK, Arlm M. Krause's Food, Nutrition and Diet Therapy. 8th ed. Philadelphia: W.B. Saunders Company. 1992; 318.
51. Cochran WG, Sampling technique. A Wiley publication in applied statistics 3rd Ct. New York: John Wiley and sons. 1977; 76-79.
52. Zimmermann MB, Hess SY, Hurrell RF. A national study of the prevalence of overweight and obesity in 6-12 y-old Swiss children: body mass index, body-weight perceptions and goals. *Eur J Clin Nutr.* 2000; 54:568-72.
53. Bangladesh Population Census 1991, Vol.2. Union Statistics. Bangladesh Bureau of Statistics. Statistics Division, Ministry of Planning Govt. of the Pimple's Republic of Bangladesh. Dhaka, 1993.
54. Bangladesh Statistical Pocket Book 1999. Bangladesh Bureau of Statistics. Statistics Division, Ministry of Planning Govt. of the Pimple's Republic of Bangladesh. Dhaka, 1999.
55. Jasim Uddin M, Sirajuddin A K M Mazumder M A et al. ESP Services in Dhaka City. An Inventory of GOB and NGO Health Facilities. ICDDR, B. Dhaka, 1999.
56. Child Survey: Admission, Attendance and Dropout. District Primary Education Office. Dhaka, 1999.
57. Small Area Atlas of Bangladesh. Mouzas and Mahallahs of Dhaka district. Bangladesh Bureau of Statistics. Statistics Division, Ministry of Planning Govt. of the Pimple's Republic of Bangladesh. Dhaka, 1985.
58. Schlesselman JJ. Case Control Studies. New York: Oxford University Press, 1982. Aurelius G, Khanls NC, Truc DB, Ha 11 Lmdgren G. Height, Weight and Body Mass Index (BMI) of Vietnamese (Hanoi) School Children Aged 7-1 years Related to Parents Occupation and Education. *J Trop Pediatr.* 1996; 42:21-42.
59. United Nations. How to weigh and measure children. New York: UN, 1986.
60. Anonymous. Obesity and Cardiovascular Disease Risk Factors in Black and white girls: The NHLBI Growth and Health Study. *Am J Public Health.* 1002; 82: 1613-1620.
61. Johnson S. Brich LL. Parents' and children's adiposity and eating style. *Pediatrics.* 1994; 95:653-661.
62. Rahman SMM, Kabir, Akter BMD, Begum H, Khaled MA, Rashid HA, Bhuyan MAH, Malek MA, Khan MR. Energy intake and expenditure of obese and non obese urban Bangladeshi children. *Bangladesh Med R Councl Bull.* 2002; 28:54-60.

63. World Health Organization. Energy and protein requirements. WHO. Technical Report Series 724. Geneva: WHO, 1985.
64. James WPT, Schofield EC. Human Energy requirements. New York: Oxford University Press, 1990.
65. Torun B, Davies PSW, Livingstone MBE, Paolisso M, Sackett R, Spurr GB. Energy requirements and dietary energy commendations for children and adolescents 1 to 18 years old. In: Scrimshaw NS, Waterlow JC, Schurch B. eds. Energy and protein requirements. Eur J Clin Nutr. 1996; 50 (Suppl 1): S27- S81.
66. Briggs M, Calloway DH. Bogert's Nutrition and physical Fitness. 10th Edn. Philadelphia: Saunders College Publishing. Robinson TN, Hammer LD, Killen JD, Kraemer HC, Wilson DM, Hayward C Taylor CB. Does television viewing increase obesity and reduce physical activity? Cross-sectional and longitudinal analyzed among adolescent girls Pediatrics. 1983; 91:273-280.
67. Howard LT et al. Children's television viewing habit and the family environment. Am J Dis Child. 1990; 144:357-59.
68. Taras HL, Sallis JF, Patterson TL, Nader PR, Nelson JA. Televisions influence on children's diet and physical activity. J Dev Behav Pediatr. 1989; 10:176-80.
69. Steinberger J et al. Relationship between insulin resistance and abnormal lipid profile in obese adolescents. J Pediatr. 1995; 126: 690-95.
70. Maffei C, Schutz Y, Piccoli R, Gonfinatini E, Pirelli L. Prevalence of obesity in children in north east Italy. Obes. 1993; 17:287-94.
71. Kotani E et al. Two decades of annual medical examinations in Japanese obese children: do obese children grow into obese adults? Int J Obes Relat Metab Disord. 1997; 21:912-21.
72. Waterlow JC. Causes and mechanisms of linear growth retardation (stunting). Eur J Clin Nutr. 1994; 48:S1-4
73. Must A. Childhood energy intake and cancer mortality in adulthood. Nutr Rev. 1999; 57:214
74. Waterlow JC. Protein energy malnutrition. London: Edward Arnold, 1992.
75. Sawaya AL, Dallal G, Solymos G et al. Obesity and malnutrition in a shantytown population in the city of Sao Paulo, Brazil. Obes Res. 1995; 3:107S-15S.
76. Popkin BM, Richards MK, Montiero CA. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. J Nutr. 1996: 26:3009-16.
77. Sawaya AL, Dallal G, Solymos et al. Obesity and malnutrition in a shantytown population in the city of Sao Paolo, Brazil. Obes Res 1995; 3 (suppl): 107S-15-S.
78. Doak C, Monteiro C, Popkin B. The coexistence of obesity and undernutrition in the same households is an emerging phenomena in lower income countries. FASEB J. 1999; 26:3009-16
79. Krahenbuhl JO, Schutz Y, Jequier E. High fat versus high carbohydrate nutritional supplementation: a one year trial in stunted rural Gambian children. Eur J Clin Nutr. 1998; 52:213-22.
80. Sawaya AL, Grillo LP, Vereschi I, da Silva A, Roberts SB. Mild stunting is associated with higher susceptibility to the effects of high fat diets: studies in a shantytown population in Sao Paulo, Brazil. J Nutr. 1997; 128 (suppl): 4 15S-20S.
81. Hoffman DI, Sawaya AL, Coward WA et al. Energy expenditure of stunted and non stunted boys and girls living in the Shantytowns of Sao Paulo, Brazil. Am J Clin Nutr. 2000; 72:1025-31.
82. Hillman RW: Infant feeding patterns and oral habits of overweight and underweight children. Am J Clin Nutr. 1963; 13:326-30.
83. Ravelli GP, Belmont L. Obesity in nineteen year old men: family size and birth order associations. Am J Epidemiol. 1979; 109:66-70.
84. Cutting TM, Fisher JO, Grimm-Thomas K, Birch LL. Like mother, like daughter: familial Patterns of overweight are mediated by mothers' dietary disinhibition. Am J Clin Nutr. 1999; 69:608-13.
85. Stettler N, Tershakovec AM, Zemel BS et al. Early risk factors for increased adiposity: a cohort study of African American subjects followed from birth to young adulthood. Am J Clin Nutr. 2000; 72:378-83.
86. Darwish OA, Khalil Iviii, Sarhan AA, Mi HE. Aetiological factors of obesity in children. Hum Nutr Clin Nutr. 1985; 39:131-6
87. Bruch H, Touraine G: Obesity in childhood. V: the family frame of obese children. Psychosom Med. 1940; 2:141-206.
88. Bruch H: Obesity in childhood. Am J Dis Child. 1940; 59:739-781.
89. Popkin BM et al. A review of dietary and environmental correlates of obesity with emphasis on developing countries. Obes Res. 1995; 3 (Suppl 2): 145S-153S.
90. Sherry B, Springer DA, Comick FA, Garrett SM. Short, Thin or obese? Comparing growth indexes of children from high and low poverty areas. J Am Diet Assoc. 1992; 92:1092-95.
91. James PT, Leach R, Kalamara E, Shayeghi M. The worldwide obesity epidemic. Obes Res. 2001; 9 (suppl 4):228S-233S.
92. National Center for Health Statistics. Health promotion and disease prevention. United States 1985. Vital Health Star (10) No. 163. DHHS publication no (P115) 88; 1591. Washington D.C

- U.S Department of Health and Human Services. 1987;20.
93. Hanley MG, Hams SB, Gittelsohn et al. Overweight among children and adolescent in a Native Canadian Community: prevalence and associated factors. *Am J Clin Nutr.* 2000;71 :693-700.
 94. Wang Y. Cross National Caparison of childhood obesity: The epidemic and the relationship between obesity and socio-economic study. *Int J Epidemiol.* 2001; 30:1129-36.
 95. Krushnapriya Sahoo, Bishnupriya Sahoo, Ashok Kumar Choudhury, Nighat Yasin Sofi, Raman Kumar, and Ajeet Singh Bhadoria Childhood obesity: causes and consequences. *J Family Med Prim Care.* 2015; 4: 187–192.
 96. Dietz WH, Gortmaker SL. Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. *Pediatrics.* 1985; 75:807-12.
 97. Andersen RE, Crespo CJ, Bartlett SJ, Cheskin LJ, Pratt M (1998) Relationship of physical activity and television watching with body weight and level of fatness among children: Results from the third national health and nutrition examination survey. *JAMA.* 279: 938-942.
 98. Hernández B, Gortmaker SL, Colditz GA, Peterson KE, Laird NM, et al. (1999) Association of obesity with physical activity, television programs and other forms of video viewing among children in Mexico City. *Int J Obes Relat Metab Disord.* 23: 845-854.
 99. Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, et al. (1996) Television viewing as a cause of increasing obesity among children in the United States, 1986-1990. *Arch Pediatr Adolesc Med.* 150: 356-362.
 100. Hancox RJ, Poulton R (2006) Watching television is associated with childhood obesity: But is it clinically important? *Int J Obes (Lond).* 30:171-175.
 101. Klein JD, Sesselberg Potential implications for childhood obesity. *Pediatrics.* 1993; 91:281-86.
 102. Tanasescu M, Ferris AM, Hinunelgreen DA, Rodriguez N, Perez-Excamilla R. Biobehavioral factors are associated with obesity in Puerto Rican children, *J Nutr.* 2000; 130:173442.
 103. O'Loughlin J, Gray-Donald K, Paradis G, Meshefedjian G. One and two year predictors of excess weight gain among elementary school children in multiethnic, low-income, inner city neighborhoods. *Am J Epidemiol.* 2000; 152:73946.
 104. Gain SM, Clark CC. Trends in fatness and the origins of obesity. *Pediatric.* 1976; 57: 443-56.
 105. Esposito-Del Puente A, Scalfi L, De Filippo E et al. Familial and environmental influences on body composition and body fat distribution in childhood in southern Italy. *Int J Obes Relat Metab Disord.* 1994; 18:596-601.
 106. Johnson SL, Birch LL. Parents' and children's adiposity and eating styles. *Pediatrics.* 1994; 94:653-61.
 107. Fisher JO, Birch LL. Fat preferences and fat consumption of 3 to 5 year old children are related to parental adiposity. *J Am Diet Assoc.* 1995; 95:759-64.
 108. Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Arch Dis Child.* 1997; 77:376-81