

Research article

Impact of obesity on glucose and lipid profiles in adolescents at different age groups in relation to adulthood

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Abstract

Background: As obesity is rapidly becoming a major medical and public health problem, the aim of our study was to determine: 1) if obesity in Caucasian adolescents at 5 different Tanner stages are associated with obesity in adulthood and its obesity-associated abnormal glucose and lipid profiles, 2) the type of fat distribution is associated with glucose and lipid profile abnormalities, and 3) the risk level and the age of appearance of these abnormalities.

Methods: For the first study, data analyses were from a case-control study of adolescents classified according to their BMI; a BMI $\geq 85^{\text{th}}$ percentile for age and sex as overweight, and those with a BMI $\geq 95^{\text{th}}$ percentile as obese. Subjects with a BMI $< 85^{\text{th}}$ percentile were classified as controls. WC:AC ratio of waist circumference to arm circumference was used as an indicator of a central pattern of adiposity. Two other indices of central adiposity were calculated from skinfolds: Central-peripheral (CPR) as $(\text{subscapular skinfold} + \text{suprailliac skinfold}) / (\text{triceps skinfold} + \text{thigh skinfold})$ and ratio of subscapular to triceps skinfold (STR). The sum of the four skinfolds (SUM) was calculated from triceps, subscapular, suprailliac and thigh skinfolds. SUM provides a single measure of subcutaneous adiposity. Representative adult subjects were used for comparison. Glucose and lipid profiles were also determined in these subjects. Abnormal glucose and lipid profiles were determined as being those with fasting glucose ≥ 6.1 mmol/l and lipid values $\geq 85^{\text{th}}$ percentile adjusted for age and sex, respectively. Prevalence and odds ratio analysis were used to determine the impact of obesity on glucose and lipid profiles at each Tanner stages for both sexes. Correlation coefficient analyses were used to determine the association between glucose and lipid profiles and anthropometric measurements for both sexes. The second study evaluated in a retrospective-prospective longitudinal way if: 1) obesity in adolescence is associated with obesity in adulthood and 2) the nature of obesity-associated risk factors. Incidence and odds ratio analysis were used to determine the impact of obesity on glucose and lipid profiles at 7 different age groups from 9 to 38 years old in both sexes between 1974 to 2000.

Results: Overall, glucose and lipid profiles were significantly ($P < 0.01$) associated with all anthropometric measurements either in male and female adolescents. WC:AC, CPR, STR and SUM are stronger predictors of both glucose and lipid profiles than BMI. Obese and overweight adolescents of Tanner stages III and higher are at increased risk of having an impaired glucose and lipid profiles than normal subjects with odds ratios of 5.9 and higher. Obesity in adolescents of 13–15 years old group is significantly ($P < 0.01$) associated with obesity in adulthood (with odds ratios of at least 12 for both men and women) and abnormal glucose (odds ratio of ≥ 8.6) and lipid profiles (odds ratio of ≥ 11.4).

Conclusions: This study confirmed that adolescents aged between 13 and 15 years old of both sexes with a BMI \geq 85th percentile are at increased risk of becoming overweight or obese adults and presenting abnormal glucose and lipid profiles as adults. This emphasizes the importance of early detection and intervention directed at treatment of obesity to avert the long-term consequences of obesity on the development of cardiovascular diseases.

Background

Obesity is rapidly becoming one of the most important medical and public health problems of our time. Obesity is associated with a high rate of morbidity and early mortality if left untreated [1–4]. Studies indicate that the presence of obesity increases the risk for developing cardiovascular diseases and diabetes [5–8]. Its role as a health hazard in adults has been well recognized for some time [1–13] but little attention has been paid to childhood and adolescent obesity [14–17].

The prevalence of obesity among US children is approximately of 22–30% and has increased at an accelerating rate in the past several years [18–20]; the prevalence of obesity is $>$ 30% in some pediatric populations [20,21]. Obesity in childhood is associated with obesity in adulthood [22–25], and is a predictor of all causes of obesity-associated mortality during adulthood, particularly mortality from cardiovascular diseases, which are independent of adult weight [24,25]. Epidemiologic evidence supports the theory that the relation between obesity and disease risk begins early in life. For instance, adolescents who died in accidents had fatty streaks in the coronary arteries and aorta that were found at autopsy, and were associated with blood lipid profile, blood pressure, and obesity status obtained at one or more points antemortem [26,27]. Moreover, a recent longitudinal study indicated that the occurrence of overweight, hypertension, and dyslipidemia in young adults (aged 19–32 y) was associated with these same risk factors in childhood. Data from the Children's Hospital Medical Center in Cincinnati have shown that the incidence of type 2 diabetes among adolescents increased 10-fold from 1982 to 1994, and virtually all diagnosed cases of type 2 diabetes occurred in obese individuals [28]. These observations are likely to reflect national trends and certainly deserve both confirmation, and a sharper focus.

To test the hypothesis that being an overweight or obese adolescent is associated with early appearance of glucose and lipid profile abnormalities, we performed a case-control study quantifying the odds ratio of overweight and obesity on these abnormalities through different Tanner's stages. The association between glucose and lipid profiles and the distribution of adipose tissue was also tested in this study. The objective of the second study was to determine, from a retrospective-prospective longitudinal study

of overweight and obese male and females having abnormal glucose and/or lipid profiles at which age obesity in adolescence can be predictive of adult obesity and its obesity-associated cardiovascular risk factors.

Methods

Study I

The first investigation was designed as a case-control study and was performed in hospitals associated with Montreal and McGill Universities. Many of the subjects were also directly recruited from nursery schools. This study was conducted to determine the relationship between various indicators of adiposity and risk factors for cardiovascular diseases in Caucasian normal, overweight, and obese male and female adolescents. The prevalence and odds ratio of being overweight or obese on glucose and lipid profiles was also determined. Each Tanner stage is associated with important changes in genital development and sexual hormone profiles, which could have an impact on adipose tissue distribution and glucose and lipid profiles. Informed written consent was obtained from the parents and/or of all participating subjects. The participants were recruited from local and national advertising. To be included in the study, adolescents and adults had to meet the following criteria: 1) be of Caucasian ethnicity; 2) be free from diagnosed endocrine conditions that could lead to obesity, and 3) not taking any medication affecting body weight. They were selected for the study on the basis of their body mass index. Standing height was measured with a wall-mounted tape, and weight was measured with the same standard balance scale.

Adolescents were classified according to BMI; \geq 85th percentile as overweight, and \geq 95th percentile for age and sex as obese [29–32]. Recent workshops concluded that the BMI has been validated by studies on total body fat [32]. The standards used to identify overweight and obesity in children and adolescents should agree with the standards used to identify grade 1 and grade 2 overweight (BMI of 25 and 30, respectively) in adults [33]. Adolescents with a BMI $<$ 85th percentile were classified as controls (Group 1), Group 2 were overweight (BMI $>$ 85th percentile and $<$ 95th percentile) and group 3 were obese (BMI \geq 95th percentile). Adults with a BMI $<$ 25 were classified as controls, between 25 and 30 as overweight and \geq 30 as obese. Sexes were grouped separately.

Table 1: Anthropometric measurements with categories of BMI and Tanner stages. A group of adults was also used for comparison purpose. Adults with a BMI < 25 were classified as controls, between 25 and 30 as overweight and ≥ 30 as obese. A BMI of 25 and 30 correspond to 85th and 95th percentile respectively.

Tanner's Stage	Age (years)	BMI(%)	WC:AC (ratio)	CPR (ratio)	STR (Index)	SUM
Male						
Tanner I						
Normal (n = 38)	10.7 ± 0.4	76.7 ± 1.8	2.3 ± 0.05	1.3 ± 0.04	0.7 ± 0.02	51 ± 6
Overweight (n = 36)	10.8 ± 0.5	87.2 ± 1.0	3.0 ± 0.07	1.7 ± 0.04	0.9 ± 0.02	70 ± 9
Obese (n = 34)	10.6 ± 0.6	96.7 ± 1.1	3.4 ± 0.07	2.2 ± 0.05	1.3 ± 0.04	86 ± 11
Tanner II						
Normal (n = 35)	11.9 ± 0.6	76.4 ± 2.0	2.4 ± 0.03	1.2 ± 0.03	0.6 ± 0.02	47 ± 8
Overweight (n = 32)	11.8 ± 0.6	88.3 ± 1.4	3.1 ± 0.06	1.7 ± 0.05	0.9 ± 0.02	76 ± 8
Obese (n = 35)	11.8 ± 0.5	97.1 ± 1.0	3.6 ± 0.05	2.3 ± 0.03	1.4 ± 0.03	90 ± 7
Tanner III						
Normal (n = 39)	13.3 ± 0.7	77.4 ± 2.3	2.4 ± 0.02	1.4 ± 0.04	0.8 ± 0.03	49 ± 8
Overweight (n = 36)	13.2 ± 0.6	88.9 ± 1.1	3.3 ± 0.07	2.0 ± 0.04	1.3 ± 0.02	85 ± 10
Obese (n = 36)	13.3 ± 0.5	97.4 ± 0.9	4.2 ± 0.09	2.5 ± 0.05	1.8 ± 0.03	110 ± 9
Tanner IV						
Normal (n = 33)	13.9 ± 0.5	78.1 ± 3.2	2.5 ± 0.05	1.5 ± 0.03	0.8 ± 0.02	53 ± 8
Overweight (n = 31)	14.0 ± 0.5	90.0 ± 1.1	3.4 ± 0.08	2.1 ± 0.03	1.4 ± 0.03	88 ± 12
Obese (n = 33)	13.8 ± 0.6	96.8 ± 0.4	4.1 ± 0.09	2.6 ± 0.05	1.9 ± 0.02	108 ± 11
Tanner V						
Normal (n = 38)	14.6 ± 0.3	79.5 ± 2.4	2.5 ± 0.04	1.4 ± 0.04	0.7 ± 0.03	54 ± 7
Overweight (n = 35)	14.6 ± 0.5	87.4 ± 0.8	3.4 ± 0.05	2.1 ± 0.05	1.3 ± 0.02	86 ± 12
Obese (n = 36)	14.7 ± 0.4	96.6 ± 0.4	3.9 ± 0.08	2.6 ± 0.06	2.0 ± 0.02	115 ± 14
ADULTS						
Normal (n = 44)	31.2 ± 0.3	22.2 ± 0.5	2.4 ± 0.03	1.4 ± 0.04	0.7 ± 0.02	54 ± 8
Overweight (n = 43)	30.6 ± 0.6	28.4 ± 0.8	4.1 ± 0.05	2.4 ± 0.06	1.5 ± 0.02	106 ± 12
Obese (n = 45)	31.5 ± 0.4	33.6 ± 0.4	4.9 ± 0.07	2.9 ± 0.05	2.4 ± 0.02	145 ± 14
Female						
Tanner I						
Normal (n = 35)	10.3 ± 0.5	76.3 ± 2.8	2.3 ± 0.05	1.2 ± 0.04	0.8 ± 0.01	48 ± 5
Overweight (n = 30)	10.3 ± 0.6	88.2 ± 1.0	2.9 ± 0.07	1.6 ± 0.04	0.9 ± 0.02	72 ± 8
Obese (n = 33)	10.2 ± 0.5	96.0 ± 0.3	3.4 ± 0.09	1.9 ± 0.03	1.0 ± 0.03	88 ± 7
Tanner II						
Normal (n = 31)	11.4 ± 0.6	77.3 ± 2.0	2.4 ± 0.04	1.3 ± 0.04	0.8 ± 0.02	54 ± 9
Overweight (n = 32)	11.3 ± 0.5	89.1 ± 1.9	3.2 ± 0.06	1.9 ± 0.05	1.2 ± 0.02	78 ± 8
Obese (n = 29)	11.4 ± 0.7	97.4 ± 0.6	3.7 ± 0.08	2.2 ± 0.04	1.5 ± 0.03	90 ± 9
Tanner III						
Normal (n = 37)	12.9 ± 1.0	77.6 ± 3.0	2.4 ± 0.04	1.3 ± 0.03	0.8 ± 0.03	55 ± 9
Overweight (n = 33)	12.8 ± 0.9	91.6 ± 2.0	3.5 ± 0.07	2.2 ± 0.05	1.1 ± 0.04	82 ± 7
Obese (n = 32)	12.9 ± 0.7	97.5 ± 0.5	4.1 ± 0.07	2.5 ± 0.04	1.5 ± 0.03	105 ± 10
Tanner IV						
Normal (n = 32)	13.6 ± 0.7	79.1 ± 1.8	2.3 ± 0.06	1.4 ± 0.05	0.9 ± 0.02	49 ± 6
Overweight (n = 29)	13.7 ± 0.8	90.4 ± 0.7	3.6 ± 0.07	2.3 ± 0.04	1.2 ± 0.04	90 ± 11
Obese (n = 28)	13.8 ± 0.7	96.8 ± 0.9	4.0 ± 0.06	2.5 ± 0.05	1.5 ± 0.03	108 ± 9
Tanner V						
Normal (n = 33)	14.7 ± 0.7	79.3 ± 2.5	2.4 ± 0.07	1.3 ± 0.03	1.0 ± 0.03	49 ± 6
Overweight (n = 34)	14.6 ± 0.8	89.9 ± 2.0	3.6 ± 0.06	2.2 ± 0.04	1.3 ± 0.03	92 ± 9
Obese (n = 34)	14.7 ± 0.7	97.0 ± 0.4	3.9 ± 0.06	2.5 ± 0.04	1.6 ± 0.03	107 ± 8
ADULTS						
Normal (n = 40)	33.4 ± 0.3	22.2 ± 0.4	2.4 ± 0.02	1.3 ± 0.03	0.7 ± 0.01	54 ± 5
Overweight (n = 42)	31.2 ± 0.5	27.8 ± 0.6	4.3 ± 0.05	2.7 ± 0.05	1.4 ± 0.02	116 ± 16
Obese (n = 40)	33.5 ± 0.5	32.6 ± 0.5	4.7 ± 0.07	3.0 ± 0.06	2.6 ± 0.03	150 ± 17

The results are expressed as means ± SD. P < 0.01 Normal vs overweight and P < 0.001 Normal vs Obese for either male and female. There is no differences between males and females for the variables measured WC:AC: waist to arm circumference. CPR: subscapular skinfold + suprailliac skinfold) / (triceps skinfold+ thigh skinfold), STR: ratio of subscapular to triceps skinfold, SUM:triceps, subscapular, suprailliac and thigh skinfolds in the total sample.

Table 2: Prevalence of impaired fasting glucose and lipid profile by categories of BMI in adolescents at different Tanner's stages and sex. A group of adults was used for comparison purpose.

Tanner's Stages	N	Impaired glucose profile		Impaired lipid profile	
		No Cases	Prevalence (%)	No Cases	Prevalence (%)
Male					
Tanner I					
Normal	38	0	0	0	0
Overweight	36	4	11.1	0	0
Obese	34	15	44.1	4	11.7
Tanner II					
Normal	35	0	0	0	0
Overweight	32	5	15.6	0	0
Obese	35	13	37.1	4	11.4
Tanner III					
Normal	39	4	10.3	3	7.7
Overweight	36	13	36.1	13	36.1
Obese	36	28	77.7	18	50.0
Tanner IV					
Normal	33	3	9.1	5	15.2
Overweight	31	14	45.1	14	45.1
Obese	33	31	94.0	32	88.8
Tanner V					
Normal	38	5	13.2	6	15.8
Overweight	35	13	37.1	14	40.0
Obese	36	36	100	32	88.8
ADULTS					
Normal	44	6	13.6	6	13.6
Overweight	43	18	41.8	17	39.5
Obese	45	42	100	42	93.3
Female					
Tanner I					
Normal	35	0	0	0	0
Overweight	30	3	10	0	0
Obese	33	12	36.4	4	12.1
Tanner II					
Normal	31	0	0	0	0
Overweight	32	4	12.5	0	0
Obese	29	13	44.8	4	13.8
Tanner III					
Normal	37	5	13.5	5	13.5
Overweight	33	12	36.4	13	39.4
Obese	32	26	81.3	18	56.3
Tanner IV					
Normal	32	4	12.5	4	12.5
Overweight	29	17	58.6	14	48.2
Obese	28	24	85.7	25	86.2
Tanner V					
Normal	33	4	12.1	5	15.2
Overweight	34	13	38.2	14	41.1
Obese	34	29	85.3	31	91.1
ADULTS					
Normal	40	6	15.0	6	15.0
Overweight	42	17	40.5	17	40.5
Obese	40	40	100	37	92.5

WC:AC was calculated from the ratio of waist circumference to arm circumference and used as a measure of fat distribution analogous to waist to hip ratio, on the assumption that a higher ratio of waist to arm circumference is an indicator of a more central pattern of adiposity. We used this measure in preference to waist to hip ratio, because a study which determined peripheral and abdominal adipose tissue content in children using magnetic resonance imaging found that WC:AC correlated positively with intra-abdominal adipose tissue ($r \pm 0.63$, $P = 0.001$) while waist to hip ratio did not ($r = 0.11$) [34]. Two other indices of central adiposity were calculated from

skinfolds: Central-peripheral (CPR) as subscapular skinfold + suprailliac skinfold) / (triceps skinfold+ thigh skinfold) and ratio of subscapular to triceps skinfold (STR). STR is an index of relative distribution of central subcutaneous adiposity. The sum of the four skinfolds (SUM) was calculated from triceps, subscapular, suprailliac and thigh skinfolds in the total sample. The SUM provides a single measure of subcutaneous adiposity. Anthropometric measurements were done by the same experienced evaluator to reduce variability which was measured at less than 0.5%.

Table 3: Sensitivity, specificity, and odds ratios for impaired glucose profile (IGP) vs normal glucose profile (NGP) levels for both sex at Tanner's stages 3 to 5 with a selected cutoff point for BMI values at the 85th percentile.

Tanner's stages	Cutoff point for BMI values		Sensitivity	Specificity	Odds ratio
	> 85 th	< 85 th			
Tanner III					
Males					
IGP(n = 47)	43	4		91%	
NGP (n = 54)	19	35	64.8%		
Total (n = 101)	62	39			19.6
Females					
IGP(n = 43)	38	5		88.4%	
NGP (n = 44)	12	32	72.7 %		
Total (n = 87)	50	37			20.3
Tanner IV					
Males					
IGP(n = 48)	45	3		93.5%	
NGP (n = 48)	18	30	71.4%		
Total (n = 96)	63	33			23.7
Females					
IGP(n = 45)	41	4		93.2%	
NGP (n = 45)	17	29	64.4%		
Total (n = 90)	58	32			17.5
Tanner V					
Males					
IGP(n = 54)	49	5		90.7%	
NGP (n = 55)	22	33	60%		
Total (n = 109)	71	38			14.7
Females					
IGP(n = 46)	42	4		87.9%	
NGP (n = 55)	26	29	61.8%		
Total (n = 101)	68	33			11.7
Adults					
Males					
IGP(n = 69)	63	6		86.4%	
NGP (n = 63)	25	38	64.3%		
Total (n = 132)	98	44			16.0
Females					
IGP(n = 63)	57	6		85%	
NGP (n = 59)	25	34	69.5%		
Total (n = 122)	82	40			12.9

An odds ratio of 19.6 means that the risk of IFG is 19.6 times higher in Tanner's stage 3 adolescents who are having a BMI > 85th than in those with a BMI < than 85th percentile.

Genital development was evaluated with the criteria of Tanner [35], and was performed with a questionnaire and physical exam by an experienced physician, in which stage 1 is prepubertal, stage 2 is initial development of the genitalia, stages 3 and 4 are intermediate, and stage 5 is mature. Stage of sexual maturation in the girls was determined on the basis of a combination of menarcheal status and breast and pubic hair development [36]. The stages of sexual maturation were performed with a questionnaire and physical exam done by an experienced physician as follow: premenarcheal with no secondary sexual development (stage 1). Premenarcheal with some secondary sexual development (stage 2); premenarcheal at the time of study but attained menarche within 6 months (stage 3); postmenarcheal with secondary sexual development not yet mature (stage 4); and postmenarcheal with secondary sexual development (stage 5).

The subjects were instructed to arrive to the clinic at 8:00 AM after 12-hour overnight fast. BMI was measured and a venous blood sample was taken followed by a breakfast. Fasting blood samples were stored at 4°C. Serum glucose and insulin levels and lipid profile were determined. They completed a questionnaire on risk factors for atherosclerosis including family history of coronopathy, smoking, high blood pressure, personal history of coronopathy or myocardial infarct, history of cerebrovascular disease or occlusive peripheral disease. Most overweight and obese subjects had at least two risk factors for atherosclerosis.

The diagnostic criteria for glucose profile were those approved by the American Diabetes Association [37]. Diabetes is defined as a fasting serum glucose equal or higher than 7 mmol/L (126 mg/dl) and impaired fasting glucose with values between 6.0 and 7.0 mmol/L. Subjects with high fasting glucose levels were referred to their own physicians for further clinical evaluation.

Lipid profile was also performed on the fasting blood sample. The blood lipid variables investigated were triglyceride (TRG), total cholesterol (TC), high-density lipoprotein (HDL) cholesterol concentrations, low-density lipoproteins (LDL) cholesterol concentrations and the ratio of total:HDL cholesterol (T:HDL) [38]. All biochemical measurements were performed in an accredited hospital laboratory.

Study 2

The second investigation was designed as a retrospective-prospective longitudinal study from 1974 to 2000 and was performed from hospitals associated with Montreal and McGill Universities. This study was conducted to determine the age of apparition of abnormal glucose and lipid profiles in a group of overweight and obese young Caucasians adults presenting abnormal glucose and/or li-

pid profiles. The study was performed from chart review of participants. 2000 charts were pre-selected by the archivists. To be included in the study, young adults had to meet the following criteria: 1) be of Caucasian ethnicity; 2) be between the ages of 32–38 y; 3) be free from diagnosed endocrine conditions that could have led to obesity, 4) not having taken any medication affecting body weight, 5) presenting a BMI ≥ 25 at adult age; 6) presenting abnormal glucose and/or lipid profiles; 7) presenting at least 2 risk factors for cardiovascular diseases as explained earlier, and having been seen by their physicians between 1974 to 2000. Among the 2000 charts, only 128 males and 112 females were having pertinent data at the different 7 age-groups, permitting longitudinal study of all the subjects used in this study. These charts were reviewed by 2 reviewers. A sample of 20 charts were reviewed by the two reviewers to determine variability between reviewers. The inter-chart reviewer differences was less than 0.2%. Data from the subjects were presented by specific age group, which we think may approximate the criteria of Tanner [35] for genital development. WC:AC ratio was removed from the anthropometric measurements because the concept of waist circumference, waist to hip ratio or waist circumference to arm circumference was unknown or not used to quantify central adiposity.

Statistical procedures

SAS PROC MIXED version 6.11 (SAS Institute, Cary, North Carolina) was used to analyze longitudinal and correlated data. The analyses were interpreted using $P < 0.01$ as the level of significance. An unpaired T-test was used to analyze the difference between male and female for each variables measured at each Tanner stages. Pearson correlations were computed to evaluate the relationship between specific continuous variables. Odds ratios were used to quantify risk in our population.

Results

Study 1

Table 1 shows the main anthropometric measurements of our study population. Pubertal status was appropriate for age in all subjects; all girls over 14 y had reached menarche and all boys over 14 y had Tanner Stage grades of 3 or more. The distribution of obese and overweight adolescents in our study did not differ from that expected in an average North American population. As defined in methods WC:AC ratio which is an indicator of central adiposity was significantly higher in both ($P < 0.01$) overweight and ($P < 0.001$) obese adolescents in comparison with controls, either in adolescents of different Tanner's stages or adults of both sexes. The CPR value which is representative of central-peripheral obesity was also significantly higher ($P < 0.01$) in both overweight and ($P < 0.001$) obese adolescents. The same is true for the STR and

Table 4: Sensitivity, specificity, and odds ratios for impaired lipid profile (ILP) vs normal lipid profile (NLP) for both sex at Tanner's stages 3 to 5 with a selected cutoff point for BMI values at the 85th percentile.

Tanner's stages	Cutoff point for BMI values		Sensitivity	Specificity	Odds ratio
	> 85 th	< 85 th			
Tanner III					
Males					
ILP (n = 34)	31	3	46.7%	91.2%	9.1
NLP (n = 77)	41	36			
Total (n = 111)	72	39			
Females					
ILP (n = 36)	31	5	48.5%	86.1%	5.9
NLP (n = 66)	34	32			
Total (n = 102)	65	37			
Tanner IV					
Males					
ILP (n = 48)	43	5	47.5%	89.6%	7.8
NLP (n = 59)	31	28			
Total (n = 107)	64	33			
Females					
ILP (n = 43)	39	4	83%	87.5%	15.2
NLP (n = 46)	18	28			
Total (n = 89)	47	32			
Tanner V					
Males					
ILP (n = 54)	48	6	58.2 %	88.8%	11.1
NLP (n = 55)	23	32			
Total (n = 109)	71	38			
Females					
ILP (n = 50)	45	5	66.2%	84.8%	11.5
NLP (n = 51)	22	28			
Total (n = 101)	68	33			
Adults					
Males					
ILP (n = 65)	59	6	67.0%	86.4%	12.9
NLP (n = 67)	29	38			
Total (n = 132)	88	44			
Females					
ILP (n = 60)	54	6	65.8%	85%	10.9
NLP (n = 62)	28	34			
Total (n = 122)	82	40			

Normal lipid profile values were those < 75th percentile for age and sex, moderate risk > 75th percentile and < 90th percentile, and high risk \geq 90th percentile. For comparison between adolescent and adults percentiles were used. ILP was determined as being \geq 85th percentile adjusted for age and sex. Normal blood cholesterol level in adults = TC < 200 mg/dl (5.2 mmol/L), moderate risk as 5.2 to 6.2 mmol/L and high risk if > 6.2 mmol/L (240 mg/dl). For LDL cholesterol; normal if < 3.4 mmol/L (130 mg/dl), moderate risk as 3.4 to 4.1 mmol/L and high risk if > 4.1 mmol/L (159 mg/dl). HDL cholesterol was defined at high risk if < 0.9 mmol/L (35 mg/dl). TRG levels was normal if < 2.8 mmol/l (250 mg/dl), moderate risk as 250–500 mg/dl and high risk if > 5.6 mmol/L (500 mg/dl). [38]

SUM values. For all the anthropometric measurements there were no significant differences between sexes.

Table 2 indicates that the prevalence of impaired glucose and lipid profiles were highest among those with a BMI above the 95th. The prevalence of impaired fasting glucose is already significant at Tanner stage 1 with percentage prevalence of 11.1% and 44.1% for overweight and obese

subjects respectively. The prevalence was significantly higher in both ($P < 0.01$) overweight and ($P < 0.001$) obese adolescents in comparison with controls, either in adolescents of different Tanner's stages or adults of both sexes. The prevalence of impaired fasting glucose did not differ significantly between adolescents and adults, either in male or female. Adolescents with BMI exceeding the 85th percentile are at increased risk of being classified as

Table 5: Correlation between anthropometric variables and fasting glucose levels, insulin levels and lipid profile in male adolescents including all Tanner's stages.

Variables	BMI	WC:AC	CPR	STR	SUM
Glucose	0.80	0.95	0.98	0.94	0.95
Insulin	0.71	0.94	0.97	0.96	0.96
TRG	0.82	0.93	0.95	0.92	0.95
TC	0.87	0.96	0.98	0.91	0.96
HDL	0.77	0.93	0.90	0.91	0.93
LDL	0.82	0.90	0.89	0.85	0.91
T:HDLC	0.73	0.88	0.86	0.87	0.90

All correlation coefficients were statistically significant at least $P < 0.01$. See text for abbreviations.

Table 6: Correlation between anthropometric variables and fasting glucose levels, insulin levels and lipid profile in female adolescents including all Tanner's stages.

Variables	BMI	WC:AC	CPR	STR	SUM
Glucose	0.80	0.87	0.82	0.75	0.92
Insulin	0.70	0.85	0.84	0.85	0.92
TG	0.78	0.94	0.95	0.76	0.93
TC	0.82	0.92	0.85	0.75	0.91
HDL	0.74	0.91	0.92	0.84	0.91
LDL	0.82	0.91	0.86	0.79	0.91
T:HDLC	0.75	0.92	0.92	0.86	0.93

All correlation coefficients were statistically significant at least $P < 0.01$. See text for abbreviations.

having diabetes or impaired fasting glucose. Also the prevalence of lipid profile abnormalities was highest among those above the 95th percentile for BMI. Adolescents with BMI exceeding the 85th percentile are at increased risk of having abnormal lipid profiles. Again, the difference between the three groups was already present at Tanner stage 1. The presence of an abnormal lipid profile was observed from Tanner stages III and above.

Sensitivity refers to the percentage of obese or overweight participants who were correctly predicted to be at risk for either glucose or lipid profiles abnormalities. Specificity refers to the percentage of participants who were correctly predicted to be in the remaining groups. BMI > 85th percentile were chosen as cutoff point for identifying overweight adolescents and BMI < 85th percentile were considered as normal subjects. Table 3 shows that the sensitivity and specificity to predict impaired fasting glucose at Tanner stages III and above were at least 60% and 80%, respectively. With use of this cutoff point, the odds ratio

of being at risk for impaired fasting glucose varied from 11.7 to 23.7 for males and females.

Table 4 shows that the sensitivity and specificity to predict an impaired lipid profiles at Tanner stages III and above were from 47.5% to 83% (males) and from 84.8% to 91.2% (females), respectively. Using a BMI at the 85th percentile as a cutoff point, the odds ratio for impaired lipid profiles varied from 9.1 to 15.2 for males and females.

The associations between anthropometric measurements, fasting glucose and insulin levels, and lipid profiles, at each Tanner stage are presented in Tables 5 and 6 respectively, with ($P < 0.01$ and less) for each group. Regression analyses were not required to establish significance.

Study 2

Table 7 shows the main anthropometric measurements of our study population at 7 different interval age groups from 9 to 38 years old. The BMI adjusted for age and sex was appropriate at each interval age group. CPR STR and

Table 7: Anthropometric measurements by age group and sex from childhood to adulthood.

Age group	Age (years)	BMI(%)	CPR (ratio)	STR (index)	SUM
Male					
9–12 years (N = 128)	10.6 ± 0.6	86.4 ± 1.1	2.3 ± 0.07	1.5 ± 0.05	89 ± 8
13–15 years (N = 128)	14.3 ± 0.4	87.7 ± 1.3	2.4 ± 0.05	1.6 ± 0.04	110 ± 6
16–19 years (N = 128)	18.1 ± 0.5	88.6 ± 1.7	2.5 ± 0.07	1.5 ± 0.04	123 ± 7
20–23 years (N = 128)	22.9 ± 0.7	86.3 ± 2.2	2.7 ± 0.06	1.8 ± 0.05	139 ± 10
24–27 years (N = 128)	26.3 ± 0.6	87.0 ± 2.4	2.8 ± 0.06	1.9 ± 0.07	155 ± 9
28–31 years (N = 128)	28.5 ± 1.4	87.5 ± 2.6	3.2 ± 0.07	2.3 ± 0.07	173 ± 11
32–38 years (N = 128)	35.0 ± 2.6	87.0 ± 2.6	3.2 ± 0.09*	2.4 ± 0.08*	190 ± 12*
Female					
9–12 years (N = 112)	11.3 ± 0.7	85.6 ± 1.4	2.4 ± 0.06	1.3 ± 0.05	91 ± 5
13–15 years (N = 112)	13.6 ± 0.4	85.8 ± 2.3	2.5 ± 0.07	1.5 ± 0.06	112 ± 7
16–19 years (N = 112)	17.8 ± 0.4	87.9 ± 1.8	2.5 ± 0.04	1.6 ± 0.04	125 ± 9
20–23 years (N = 112)	21.2 ± 0.6	87.5 ± 2.2	2.7 ± 0.06	1.9 ± 0.07	144 ± 9
24–27 years (N = 112)	25.3 ± 0.4	87.9 ± 2.1	2.8 ± 0.07	1.8 ± 0.06	160 ± 11
28–31 years (N = 112)	29.3 ± 0.4	87.2 ± 1.7	2.9 ± 0.08	2.1 ± 0.06	174 ± 10
32–38 years (N = 112)	35.1 ± 0.5	87.5 ± 2.1	3.1 ± 0.07*	2.4 ± 0.07*	192 ± 13*

CPR: subscapular skinfold + suprailliac skinfold) / (triceps skinfold+ thigh skinfold), STR: ratio of subscapular to triceps skinfold, SUM:triceps, subscapular, suprailliac and thigh skinfolds in the total sample. *: P < 0.01 significant difference between adulthood obesity and adolescent obesity.

Table 8: Incidence of impaired glucose and lipid profile by age group and sex from childhood to adulthood.

	N	Impaired glucose profile		Impaired lipid profile	
		No Cases	Incidence (%)	No Cases	Incidence (%)
Male					
9–12 years	128	42	32.8 %	38	29.7 %
13–15 years	128	56*	43.7 %	52*	40.6 %
16–19 years	128	60	46.9 %	58	45.3 %
20–23 years	128	64	50.0 %	62	48.3 %
24–27 years	128	76*	59.4 %	76*	59.4 %
28–31 years	128	96*	75.0 %	94*	73.4 %
32–38 years	128	108*	84.4 %	110*	85.9 %
Female					
9–12 years	112	36	32.1 %	36	32.1 %
13–15 years	112	48*	42.8 %	44*	39.3 %
16–19 years	112	52*	46.4 %	52*	46.4 %
20–23 years	112	56	50.0 %	54	48.2 %
24–27 years	112	66*	58.9 %	66*	58.9 %
28–31 years	112	84*	75.0 %	82*	73.2 %
32–38 years	112	94*	83.9 %	96*	85.7 %

* : P < 0.01 significant difference between age groups.

SUM were significantly increased (P < 0.01) with age corresponding with a higher fat mass with growth. For all the anthropometric measurements there were no significant gender differences.

Table 8 presents the incidence of impaired glucose and lipid profile between age groups. The incidences of im-

paired glucose and lipid profiles were already high at the first age group with incidences of approximately 30 % for both. The incidence increases progressively with age.

Fasting plasma glucose level (results not shown), HbA1c and fasting plasma insulin levels were significantly increased (P < 0.01) through puberty and growth. There

Table 9: Sensitivity, specificity, and odds ratios of the selected cutoff point for BMI values at the 85th percentile at 13–15 y old for identifying overweight at age 35 y

BMI at age 35 y	BMI percentile at 13–15 y		Sensitivity	Specificity	Odds ratio
	> 85 th	< 85 th			
Males					
> 25 (n = 110)	88	24	77.7 %	80.0 %	12.8
< 25 (n = 18)	4	14			
Total (n = 128)	92	38			
Females					
> 25 (n = 100)	80	20	83.3 %	80.0 %	20.0
< 25 (n = 12)	2	10			
Total (n = 112)	82	30			

Specificity = 88 of 110 subjects, Sensitivity: 14 of 18 subjects. An odds ratio of 12.8 means that the risk of 13–15 years old adolescents with a BMI ≥ 85th percentile of becoming an overweight or obese adults at 35 y old is 12.8 times higher than in those with a BMI < than 85th percentile

Table 10: Sensitivity, specificity, and odds ratios of the selected cutoff point for impaired fasting glucose (IFG) values at the 85th percentile at 13–15 y old for identifying IFG at age 35 y

BMI at age 35 y	BMI percentile at 13–15 y		Sensitivity	Specificity	Odds ratio
	> 85 th	< 85 th			
Males					
> 25 (n = 110)	84	26	77.8 %	76.4 %	11.3
< 25 (n = 18)	4	14			
Total (n = 128)	88	40			
Females					
> 25 (n = 98)	76	22	71.4 %	77.6 %	8.6
< 25 (n = 14)	4	10			
Total (n = 112)	80	32			

were no significant differences between sexes. TRG, TC, LDL and T:HDLC were also significantly increased (P < 0.01) with growth. The reverse was observed for HDL.

BMI values at the 85th percentile for 13–15 year old group were chosen as cutoff points for identifying overweight at age 35 y. The sensitivities were 77.7% and 83.3%, and the specificities 80.0% and 80.0%, for males and females respectively. With use of the cutoff point of the 85th percentile at this age group, the odds ratios for overweight at age 35 y were 12.8 for males and 20.0 for females (Table 9).

Table 10 presents the sensitivity, specificity and odds ratios for the selected cutoff point for predicting impaired fasting glucose (IFG) values at the 85th percentile at 13–15 year old group, at age 35. The sensitivities were 77.8% and 71.4%, and the specificities 76.4% and 77.6% for males

and females respectively. The odds ratios for abnormal IFG at age 35 y were 11.3 for males and 8.6 for females.

Table 11 presents the sensitivity, specificity and odds ratios for predicting impaired lipid profiles (ILP) at the 85th percentile at 13–15 year old group, for age 35. The sensitivities were 80.0% and 75%, and the specificities 77.7% and 79.2%, for males and females respectively. With use of the cutoff point of the 85th percentile at 13–15 year old group, the odds ratios of ILP at age 35 y were 14.0 for males and 11.4 for females.

Discussion

This study presents for the first time that: 1) obese and overweight adolescents at Tanner stages III and higher are at an increased risk of having impaired glucose and lipid profiles at maturity than normal subjects. Odds ratios of 9.0 and higher were measured in both genders. 2) all the

Table 11: Sensitivity, specificity, and odds ratios of the selected cutoff point for Impaired lipid profile (ILP) values at the 85th percentile at 13–15 y for identifying (ILP) at age 35 y

BMI at age 35 y	BMI percentile at 13–15 y		Sensitivity	Specificity	Odds ratio
	> 85 th	< 85 th			
Males					
> 25 (n = 108)	84	24	80.0 %	77.7 %	14.0
< 25 (n = 20)	4	16			
Total (n = 128)	88	40			
Females					
> 25 (n = 96)	76	20	75.0 %	79.2 %	11.4
< 25 (n = 16)	4	12			
Total (n = 112)	80	32			

anthropometric parameters tested were strongly ($P < 0.01$) associated with glucose and lipid profiles; 3), with a retrospective-prospective longitudinal study of the same subjects, the odds ratios of adolescents aged between 13 and 15 years old with a BMI \geq 85th percentile to becoming overweight or obese at 35 y old were 12.8 and 20.0 times higher than in those with a BMI < than 85th percentile for both male and female respectively, 4) with use of the same cutoff, the odds ratios of the same adolescents with BMI \geq 85th percentile for impaired fasting glucose at 35 y old were 11.3 times higher in males and 8.6 times higher in females and 5) the odds ratio of the same adolescents of having impaired lipid profiles, expressed by lipid profile values \geq 85th percentile at 35 y old is 14.0 in males and 11.4 in females. These observations suggest that between 13 to 15 years old is an appropriate age to initiate interventions in order to prevent adult obesity and its associated risk factors.

Study 1

The rationale underlying this part of the study is that the stage of sexual maturation is an important variable in applying anthropometric measurements as predictors of risk for cardiovascular diseases. On average, BMI seems to increase with stage of genital maturation in boys relative to fatness, and then, in contrast, decreases in the later stages of maturation reflecting the rapid growth of fat free mass with puberty. BMI in girls also increases with stage of sexual maturation, but relative fatness is variable, especially in girls who just attaining menarche (stage III). To obviate this bias, we preselected our subjects according to their BMI. For instance a BMI \geq 85th percentile (for age and sex) was classified as being overweight, and those with a BMI \geq 95th percentile as obese [29–32]. Recent workshops concluded that the BMI is an index of choice for evaluating obesity in adolescents, because it has been validated by studies of total body fat [32]. The criteria used to identify

overweight and obesity in children and adolescents agree with those used for grade 1 and grade 2 overweight (BMI of 25+ and 30+, respectively) in adults [33]. Using a representative sample from the general North American Caucasian population, we demonstrate that the BMI is also a good indicator for obesity in adolescents, however the readily-measured WC:AC, CPR, and STR, indices of central obesity, and SUM, a single measure of subcutaneous adiposity, are stronger predictor of both fasting glucose and insulin levels and lipid profiles, than BMI. We also demonstrated that each anthropometric variable taken separately can significantly predict fasting glucose and insulin levels and lipid profile in adolescents of both sexes with a probability of $P < 0.01$. Our results are in accordance with others of children aged 5 and 16, in whom, indicators of fatness such as weight-for-height, BMI and skinfold thicknesses have been associated with higher total and LDL cholesterol, lower HDL cholesterol, and higher triglyceride concentrations [40–43].

Our overweight and obese adolescents had higher fasting glucose, and insulin levels for BMI than subjects with BMI in the healthy range. Furthermore, the prevalence of obesity was comparable between boys and girls. In both sexes the prevalence of diabetes and impaired fasting glucose increased relative to chronological age in both overweight ($P < 0.01$) and obese subjects ($P < 0.001$). Previous study in Aboriginal children recognized to be at high risk for type II diabetes has shown that obesity is associated with high fasting glucose and fasting insulin levels. They also demonstrated that children with BMI exceeding the 85th percentile are at increased risk of diabetes or impaired fasting glucose, odds ratio of 5.1 with 95% coefficient interval 1.5, 17.0 [39]. With use of the cutoff point of the 85th percentile we demonstrated that adolescents of Tanner stage III and higher, of both sexes, are at increased

risk of having impaired fasting glucose, with odds ratios of 11.7 and higher.

Our overweight and obese adolescents had higher TC, LDL, TRG and lower HDL values which correlated with BMI, and other measures of fatness. In both sexes the prevalence of impaired lipid profile increased relative to chronological age in both overweight ($P < 0.01$) and obese subjects ($P < 0.001$). Adolescents of both sexes, with $BMI \geq 85^{\text{th}}$ are at increased risk of impaired fasting lipid profiles, with odds ratios of 9.1 and higher.

Study 2

The goal of the second study was to determine the childhood age of abnormal glucose and lipid profiles from overweight adults. By using a retrospective-prospective longitudinal study design we were able to track BMI, glucose and lipid profiles from late childhood and adolescent ages to adulthood in a white Caucasian populations. Then we were able to correlate their subsequent risk of obesity and its associated impaired glucose and lipid profiles. There is general agreement on the use of BMI with selected cutoff points as guidelines for desirable weight [44–48]. This study provided a basis for prediction of overweight and obesity in adults from their 13–15 year old group, suggesting that this simple measurement could be applied to the prediction of obesity in public health programs. The results presented here were comparable to those observed in a previous study showing that with use of the cutoff point of the 60th percentile at age 18 y, the odds ratios of overweight at age 35 y were of 15 for males and 27 for females, with corresponding SEs of 1.9 and 3.5 respectively. Previous data on 8–18 y olds in Project Heart-Beat demonstrated that the odds ratio of overweight in adulthood for those with childhood BMI values at the 95th percentile were 2–6 and 1.5 to 5 times as great as for those with a BMI values at the 75th percentile for males and females respectively [49].

The incidence of impaired glucose profile was comparable between boys and girls. In both sexes the incidence of diabetes and impaired fasting glucose increased significantly ($P < 0.001$) relative to chronological age in comparison to adults. The sensitivity of the selected cutoff point for BMI values at the 85th percentile at 13–15 years old for identifying impaired glucose profile at age 35 y was 76.4% and 77.6% for both male and female, respectively. Therefore, at least 75% of male and female adolescents aged between 13 and 15 years old who were having a $BMI \geq 85^{\text{th}}$ were correctly predicted to have impaired glucose profile at 35 y. On the other hand, 77.8% of male and 71.4% of female adolescents of this age group who were having a $BMI \leq 85^{\text{th}}$ were correctly predicted to have normal glucose profile at 35 y. As stated in the introduction obesity in childhood is associated with obesity in adulthood [22–

25], and is a predictor of all cause of obesity-associated mortality during adulthood, particularly mortality from cardiovascular diseases, which is independent of adult weight [24,25]. The results of the present study agreed with a previous study [50] indicating that the metabolic alterations caused by excess body fat are expressed early in the natural history of obesity. Such alterations seem clinically important even in children, as evidenced by an increase in glucose profile abnormalities in obese adolescents. These observations underscore the need for research efforts directed at the development of effective interventions for obese adolescents.

The incidence of impaired lipid profile was comparable between boys and girls. In both sexes the incidence of impaired lipid profile increased significantly ($P < 0.001$) relative to chronological age in comparison to adults. The sensitivity of the selected cutoff point for BMI values at the 85th percentile at 13–15 years old for identifying impaired lipid profile at age 35 y was 77.7% and 79.2% for both male and female, respectively. The specificity was 80.0% and 75.0%, for both male and female, respectively. If we consider that most of the subjects having a $BMI \geq 85^{\text{th}}$ were also presenting more than 2 risk factors for cardiovascular disease, this means that they are at increased risk for developing cardiovascular diseases. Few studies have examined the long-term effects of adolescent obesity on adult disease. Nonetheless, obesity present in childhood or adolescence seems to increase the likelihood of adult morbidity and mortality [51]. In the present study we demonstrated that in both men and women obese during adolescence, risk of cardiovascular diseases and diabetes were increased. These effects seem related both to the persistence of obesity and to the effects of childhood or adolescent obesity on the quantity and location of body fat distribution. The risk factors for adult disease that are associated with obesity in children and adolescents persist into adulthood or increase in prevalence if weight gain occurs. Although both total body fat and regional fat deposition could account for the association of childhood or adolescent obesity with adult disease, no studies to date have examined cardiovascular risk factors and related them to visceral fat.

Conclusion

The strengths of the study include the use of large samples of both genders, a wide age range, subjects representative of the population being studied, precise measurements of body composition and the use of well-accepted BMI classifications of overweight and obesity. By using a retrospective-prospective design in the second part of the study we were able to obtain the longitudinal data required to answer the questions addressed. In conclusion our study confirms the hypothesis that overweight and obese adolescents have higher fasting plasma glucose and insulin

levels, and an abnormal lipid profile relative to their lean peers. This study highlights the critical importance of early identification and intervention of adolescent obesity to avert the long-term consequences of it and to protect adolescent against the risk of cardiovascular diseases. There is an urgent need to follow cohorts at-risks children and adolescents to investigate cofactors acting in concert with obesity that are responsible for the early onset of risk factors for cardiovascular diseases, a phenomenon increasingly being observed in many populations.

Competing interests

None declared.

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