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PREDICTING ADULT METABOLIC SYNDROME FROM CHILDHOOD BODY MASS INDEX:

Follow-up of the New Delhi birth cohort

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Abstract

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Competing interests
All authors declare that the answer to the questions on the BMJPG's competing interest form are all NO and therefore have nothing to declare.

What is already known on this topic

- Developing countries making rapid economic progress are experiencing 'epidemic' rates of type 2 diabetes and metabolic syndrome in young adults.
- Obesity in childhood is a risk factor for these adult disorders, but is an unsatisfactory marker for identifying at-risk children in developing countries, where childhood obesity is still rare.

What this study adds

- In a developing country setting, we have shown that children gaining body mass index faster than their peers, even though they are well below international definitions of obesity, are at increased risk of developing diabetes or metabolic syndrome in adult life.
- We have devised a screening test and suitable body mass index charts that could be used by clinicians to identify at-risk children, based on serial changes in their body mass index, who could be offered advice about healthy diet and lifestyle as one approach to disease prevention.

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Objectives—To assess whether serial measurements of childhood body mass index (BMI) give clinically useful predictions of the risk of developing adult metabolic syndrome and impaired glucose tolerance or type 2 diabetes.

Design/setting—Follow-up of a community-based birth cohort in Delhi, India.

Participants—1,492 men and women aged 26-32 years whose BMI was recorded 6-monthly throughout childhood.

Main outcome measures—The predictive value of childhood BMI for adult metabolic syndrome (MS) defined using waist circumference, blood pressure and fasting glucose, triglyceride and HDL-cholesterol concentrations, and impaired glucose tolerance (IGT) and diabetes (DM) diagnosed by oral glucose tolerance tests.

Results—Twenty-five percent of subjects had MS and 15% had IGT/DM. Both outcomes were associated with greater childhood BMI gain (MS: OR 1.63 [95% CI 1.44 to 1.85]; IGT/DM: 1.39 [1.20 to 1.60] per unit increase in within-cohort BMI SD-score between 5-14 years). Best predictions of adult disease were obtained using a combined test comprising i) any increase in BMI SD-score between 5-14 years and ii) a BMI SD-score >0 at 14 years (MS: sensitivity 45%, specificity 78%; IGT/DM: 37%, 73%). Likelihood ratios were low (MS: 1.4-2.0; IGT/DM: 1.2-1.4). A single high BMI measurement at 14 years (overweight or obese, International Obesity Task Force criteria) was highly specific but insensitive (MS: sensitivity 7%, specificity 97%; IGT/DM: 8%, 97%). Charts for plotting BMI SD-scores through childhood were produced.

Conclusions—Serial measurements of childhood BMI give useful predictions of adult risk and could guide advice to children and parents on preventing later disease.

Keywords

Childhood body mass index; type 2 diabetes; metabolic syndrome; predictions

INTRODUCTION

The incidence of type 2 diabetes and metabolic syndrome is increasing worldwide, most rapidly in developing countries like India[1,2]. This has been attributed to greater availability of food, urbanisation and industrialisation, and reduced physical activity, resulting in increased adiposity. It is predicted that by 2030, 75% of the world's adult diabetic patients will be in developing countries, and that India will have 80 million[1]. Action to prevent disease is urgently needed.

Childhood obesity is a risk factor for adult diabetes, hypertension and dysplidemia[3-5] and treating it may prevent later disease. However, identifying and treating only those children who are already obese is inappropriate for developing countries. Among adults born in Delhi, India, whose body mass index (BMI) was measured serially throughout childhood, those who developed impaired glucose tolerance (IGT)/diabetes or metabolic syndrome were well below internationally-recognised criteria for obesity in childhood, but had accelerated childhood BMI gain relative to the rest of the cohort[6-8]. Such children would not easily be identified using standard 'distance' BMI charts.

We have now used the Delhi data to assess how well childhood BMI predicts adult IGT/diabetes and metabolic syndrome and whether serial BMI measurements give better predictions than single measurements, and to devise charts that could be used by clinicians to monitor BMI changes and thus estimate adult risk in individual children.

METHODS

The cohort was established in 1969-19726. Married women living in a 12 km^2 area of Delhi (N=20,755) were followed up. There were 9,169 pregnancies, resulting in 8,181 live births. Trained personnel recorded the weight and length of the babies within 72 hours of birth and 6 monthly (±15 days) until 14-21 years. Gaps in funding interrupted measurements for one year in 1972-1973, and 2.5 years in 1980-1982. At recruitment, 60% of families had an income >50 rupees per month (national average 28 rupees) and 15% of parents were illiterate (national average 66%). Nevertheless, 43% of families lived in one room. Hindus were the majority religious group (84%), followed by Sikhs (12%), Christians (2%), Muslims (1%) and Jains (1%).

Current Study

In 1998-2002 we retraced 2,584 (32%) of the cohort. The study was approved by the All India Institute of Medical Sciences research ethics committee, and informed verbal consent was obtained from each subject. Weight, height and waist circumference were measured using standardized techniques. Blood pressure was recorded using an automated device (Omron 711) with the subjects seated, after five minutes rest (mean of two readings). Plasma triglyceride (fasting) and glucose (fasting and 120-minutes after a 75g glucose load) concentrations were analyzed by standard enzymatic methods using Randox kits on a Beckman autoanalyser. Fasting HDL-cholesterol was estimated using the same method after phosphotungstate precipitation. IGT and diabetes (DM) were defined using WHO criteria9. Metabolic syndrome (MS) was defined using International Diabetes Federation criteria10 (waist circumference ≥90 cm (men) or ≥80 cm (women) plus two or more of the following: a) fasting serum triglyceride ≥1.7 mmol/l; b) HDL-cholesterol <1.0 mmol/l (men) or <1.3 mmol/l (women); c) hypertension defined as on treatment for hypertension or systolic blood pressure ≥139 mmHg or diastolic pressure ≥85 mmHg and d) known diabetes or fasting plasma glucose ≥5.6 mmol/l.

Statistical Analyses—We used all recorded data (not just for subjects recruited for this study) to derive sex-specific within-cohort SD-scores for each subject at birth, age six months and at birthdays from 1-21 years6,11. Interpolated values were used if a measurement was made within 6 months (up to 1 year), 1 year (age of 2 years), 1.5 years (age of 3 years), and 2 years (all older ages). We also derived SD-scores for cohort children relative to an international reference (United States Center for Disease Control, CDC12). Because earlier analyses showed that BMI gain only after age 2 years predicted adult IGT/diabetes6, and because the number of measurements dropped after age 14 years, we limited our analysis to 2-14 years. Associations of childhood BMI SD-scores, and changes in BMI SD-scores, with adult outcomes were analysed using multiple logistic regression. Sex differences were tested using interaction terms. Risks for adult MS and IGT/diabetes were modelled as a function of BMI SD-scores at pairs of ages in childhood, including the linear and quadratic terms for both BMI SD-scores, and their interaction (the product of both SD-scores), regardless of statistical significance, to fit the full quadratic surface. We display the results using contour lines derived from this model (Figure 1). We assessed the ability of combinations of childhood BMI SD-scores and changes in childhood BMI SD-scores to predict adult outcomes, expressed as their sensitivity, specificity, attributable fraction, positive and negative predictive values, and likelihood ratio13. Data analysis was carried out using SPSS version 12.0; graphs were plotted using FigSys (Biosoft, Cambridge, UK).

RESULTS

Of 2,584 subjects traced, 1,492 had fasting blood samples and 1,442 had complete glucose tolerance tests. Compared with the original cohort, the mean childhood BMI of participants was approximately 0.1 SD lower. At all ages from 2-14 years they had a low mean BMI by international criteria (Table 1). The men and women studied had a mean age of 29 years; 25% had MS, 4% had DM and 11% had IGT (Table 1).

The prevalence of MS was higher in men and women whose childhood BMI was higher, and this association strengthened with increasing age of the childhood BMI measurement (Table 2a, Figure 1). The prevalence of IGT/DM was higher in subjects who had a lower BMI up to the age of 8 years, but from 11 years onwards, the pattern was similar to that for MS. The prevalence of both outcomes was higher in men and women whose BMI SD-score increased with time, especially at older ages (Table 2b, Figure 1). All associations were similar in both sexes. When adult BMI was included in the regression models almost all the associations became non-significant. Odds ratios at all ages were similar if CDC rather than Delhi SD-scores were used (Table 2).

Predictive tests

We investigated combinations of within-cohort BMI SD-scores as predictors of adult risk. The most practical test, and the one that gave optimal predictions, defined ‘test-positive’ children as those with a combination of any increase in BMI SD-score between two ages *and* a BMI SD-score ≥0 at the later age. For example (Table 3), for MS, and BMI SD-scores from 5-14 years, this test identified 28% of children as positive, and had a sensitivity (test-positive given that disease is positive) of 45%, specificity (test-negative given that disease is negative) of 78%, attributable fraction of 24% (the percentage of cases of MS that could potentially be avoided if all children were test-negative, assuming causality), positive predictive value (disease positive given that the test is positive) of 41%, negative predictive value (disease negative given that the test is negative) of 81% and likelihood ratio (the multiplicative factor by which test-positivity increases the odds of disease) of 2.0. Predictions were better at older ages, and with longer time intervals between BMI measurements. For IGT/DM the corresponding figures were: ‘test-positive’ 29%, sensitivity 37%, specificity 73%, attributable fraction 12%, positive predictive value 20%, negative predictive value 87% and likelihood ratio 1.4, similar at all ages and time intervals of BMI measurements (Table 3). There were no significant differences between the sexes.

When using the CDC reference, we defined ‘test-positive’ as an increase in BMI SD-score between two ages, plus a BMI SD-score at the later age of ≥-1.0. Percentages of ‘test-positive’ children and predictive parameters varied widely with age (Table 3).

Predictions based on single BMI measurements

We defined children who were overweight or obese at 5 (n=19), 8 (n=10), 11 (n=19) and 14 (n=53) years according to the International Obesity Task Force BMI criteria14. Forty-seven percent of the overweight/obese children at 14 years developed MS and 33% developed IGT/DM. Being overweight/obese was highly specific for identifying children at risk of MS or IGT/DM (Table 3), but because few of the children who developed adult pathology were overweight/obese, the sensitivity and attributable fraction were very low. If a lower BMI cut-off was selected (>75th within-cohort percentile), obtaining similar percentages of ‘test-positive’ children to the combined test, the sensitivity improved but remained inferior to that of the combined test (Table 3). Likelihood ratios were similar (regardless of the cut-off) to those obtained using the combined test.

Charts

Figure 2 shows charts based on the Delhi data and designed to estimate BMI SD-scores at all ages. The continuous wavy lines are used to plot actual BMI, and the equivalent SD-score is indicated on the left-hand scale. The upper unshaded zone of the graph indicates SD-scores >0. A child whose BMI is in this zone at the end of any age interval and who moves horizontally upwards on the chart is 'test-positive'. A similar chart based on CDC reference data is available on request.

DISCUSSION

We studied a large sample of young Indian adults who had serial measurements of BMI in childhood. As children, they were thin by international standards, but as adults had a high prevalence of overweight/obesity, diabetes and metabolic syndrome, an increasingly common scenario in urban developing country populations. Accelerated BMI gain during childhood was associated with an increased risk of adult IGT/DM and MS, probably mediated by increased adult adiposity7. Our main objective was to determine whether screening tests could be derived from serial childhood BMI measurements to detect high-risk children. Data from the US Bogalusa Heart Study was used to predict risk of MS from single childhood BMI values15 but as far as we know, this is the first such analysis using longitudinal BMI data.

Strengths of the study were that it was population-based, and anthropometric data were collected by trained personnel at unusually frequent intervals. Only 19% of the original cohort participated, and they are likely to be unrepresentative. However, the differences in their mean childhood BMI from the rest of the cohort, though statistically significant, were small.

Predictions for MS were superior to those for IGT/DM. Sensitivity was 30-45% for MS and 29-37% for IGT/DM. Positive predictive values ranged from 32-41% for MS and 18-20% for IGT/DM. Thus a substantial proportion, though a minority, of 'test-positive' children developed disease, and a positive test could thus cause unwarranted anxiety for families. Specificity and negative predictive values were high (76-81% for MS and 72-87% for IGT/DM) indicating that a negative test is reassuring. The attributable fraction (9-24% for MS, 6-12% for IGT/DM) and likelihood ratio (1.4-2.0 for MS and 1.1-1.6 for IGT/DM) were low.

The longitudinal test gave superior predictions to those based on single overweight/obese BMI values, currently often used to identify children needing intervention. The latter, though highly specific (>97%), picked up few of the children at risk and thus had a low sensitivity (≤7%). This differs from findings in the Bogalusa study, in which a single overweight/obese BMI value recorded between the ages of 4 and 15 years had a sensitivity of 38% and specificity of 87% for predicting adult MS15. If a lower BMI cut-off was used in Delhi, predictions were closer to those obtained using the combined test, but sensitivity remained lower.

Opinions will vary on the usefulness of these predictions. Likelihood ratios of >5.0 or <0.2 are generally considered important clinically16 and those for our combined test ranged from 1.1 to 2.0. No single predictive test can be taken in isolation, and likelihood ratios were similar for overweight/obesity, which most clinicians would consider worthy of intervention. Likelihood ratios do not change according to disease prevalence, and a 10 to 100% increase in the odds of developing disease is important if that disease is common and serious. Considering that these are predictions across an intervening period of several

decades, we think they are impressive, although better predictive indices, possibly metabolic profiles in adolescence, should be sought.

Given the very high prevalence of disease in young adulthood in this population, it could be argued that screening tests are redundant; there should clearly be *population-wide* attempts to reduce adiposity. Such interventions, however, have not been adopted even in rich countries, and are unlikely to happen quickly in developing countries. Parents in India who can afford to, do take their children to paediatricians regularly, and individual-level risk-assessment could be useful. Since observational studies cannot prove causation, intervening to limit BMI gain will not necessarily prevent adult disease. Furthermore, no consistently effective interventions have been identified17-20. However, the seriousness of the diabetes epidemic in India demands the formulation of policy and clinical guidelines based on best evidence. Although randomised trials extending from childhood into adulthood have not been performed, lifestyle intervention trials have documented short-term improvements in adiposity, metabolic parameters and endothelial function17-22. A commonsense approach would be to suggest that parents of test-positive children should be advised to consider lifestyle changes, to continue monitoring the child's BMI, and to obtain relevant investigations (blood pressure, lipid profile and glucose concentrations).

Our analysis raises several practical issues. It would be possible to identify 'test-positive' children using a conventional BMI chart (crossing centiles upwards and above the 50th percentile at the later measurement). These changes could, however, appear subtle on a standard chart, and furthermore, clinicians may not be concerned about a child of average BMI, even if climbing centiles. Our new charts show upward and downward movement more clearly. They are more complex than standard charts, however, and clinicians would need additional explanation/guidance to use them. Since detailed growth data is not available for all populations, an external reference would be advantageous and also facilitate global comparisons. We adapted our test to overcome the large BMI differences between the Delhi and CDC populations. The CDC reference, however, produced widely varying predictions because these differences varied with age. Our results suggest that 'local' reference data, based on children as similar as possible to those in the population under consideration, are best for identifying high-risk children. We cannot, therefore, recommend the clinical use of our chart in non-Indian populations, or in children that differ greatly in BMI from the Delhi cohort. Comparable analyses need to be carried out, and charts derived, in different populations. Because our measurements were obtained as part of a research study, they are likely to have been more precise, resulting in better predictions, than could be achieved in clinical practice. However, in a simulation exercise, introducing random BMI measurement error, and misclassification of cases and controls, predictive parameters were little changed (details available on request).

Some of the predictive indices (positive and negative predictive values and attributable fraction) are influenced by the prevalence of the outcome. Although the prevalence of metabolic syndrome and IGT/DM was already high in the Delhi cohort, it will increase further with time. We are currently re-studying the cohort after an interval of 5 years, and will re-assess the predictions.

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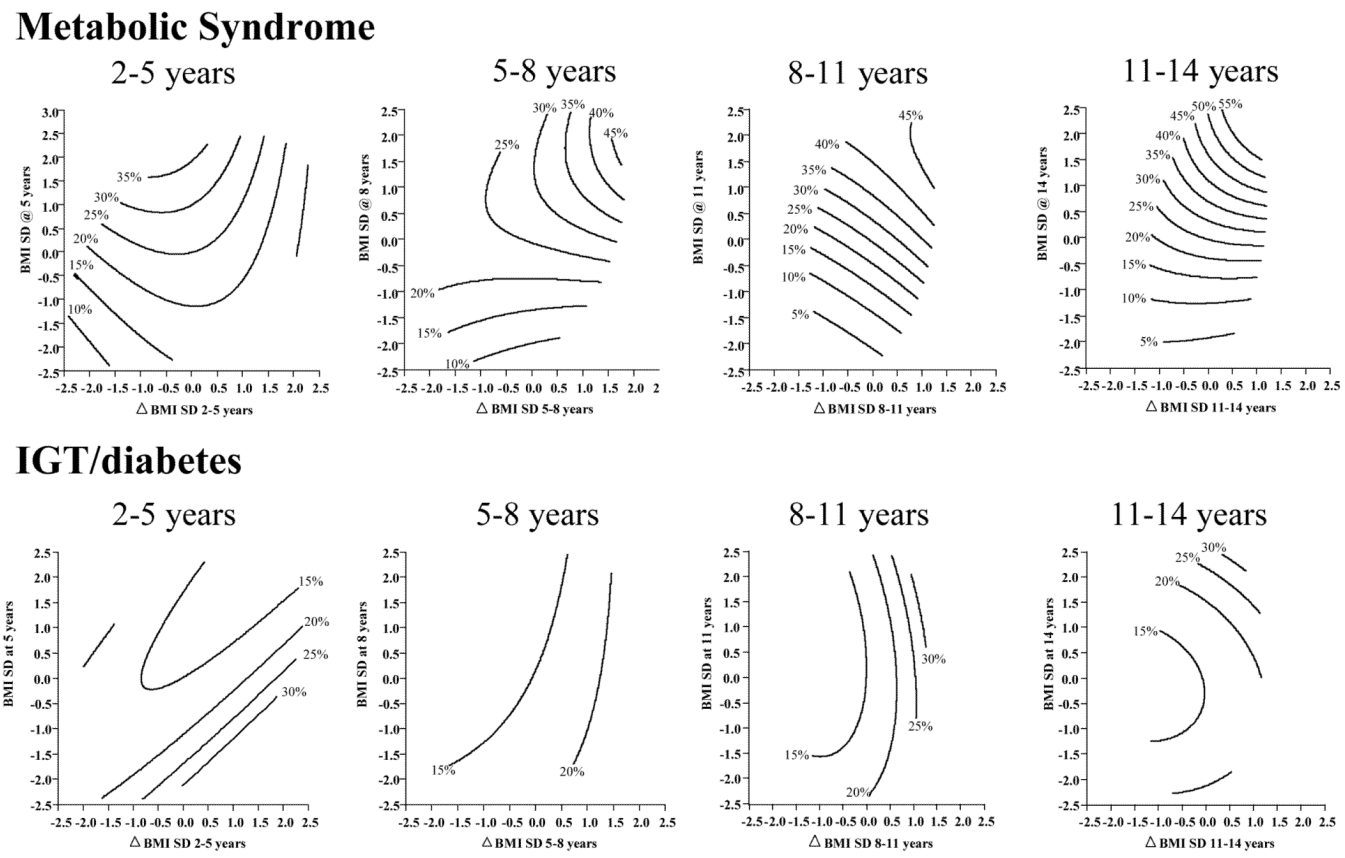

Figure 1. Prevalence (%) of (a) metabolic syndrome and (b) IGT/diabetes according to absolute BMI SD-score and changes in BMI SD-score during childhood

The contour lines show the prevalence (%) of metabolic syndrome (upper panel) or IGT/diabetes (lower panel) for different combinations of change in BMI SD-score between two ages (x-axis) and BMI SD-score at the later age (y axis). They are drawn so that they cover the most 'central' 95% of the observed data points, in order to exclude areas where there were few observations. Perfectly vertical contour lines would indicate that the prevalence varies mainly with *change* in BMI SD-score, rather than with absolute attained BMI SD-score; horizontal contours indicate the opposite, and diagonal contours indicate that prevalence varies with both these parameters.

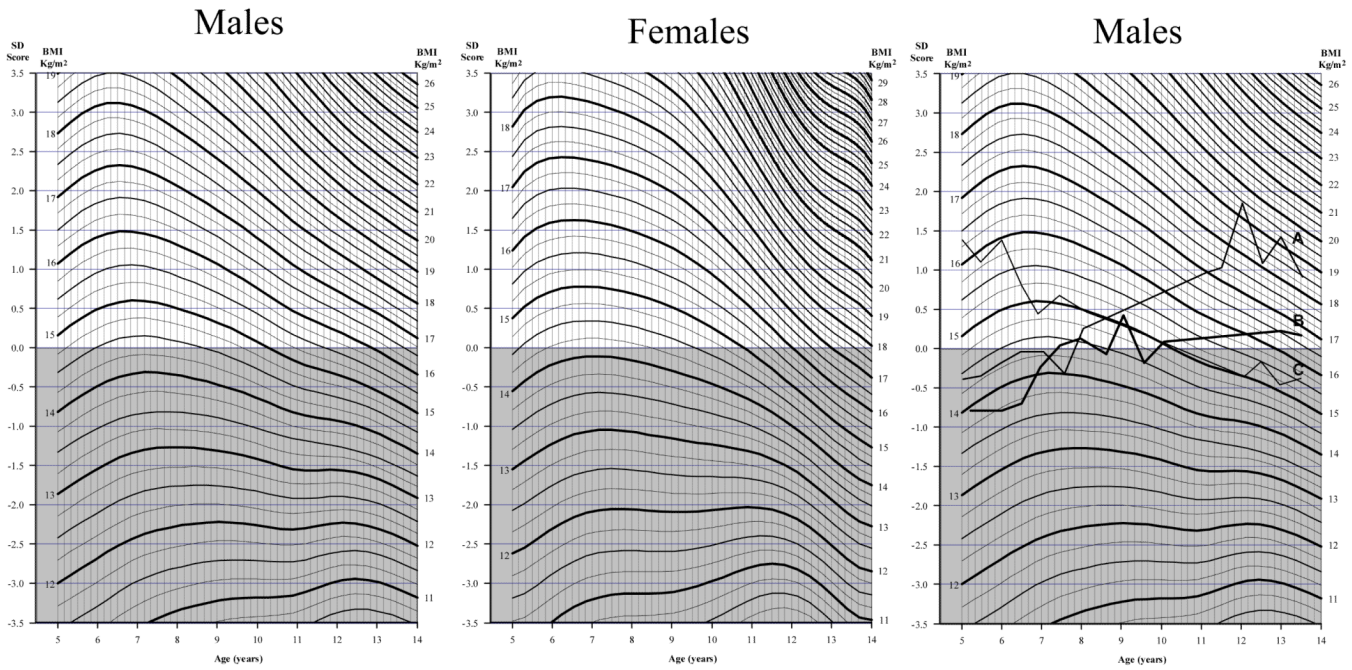

Figure 2. Chart for plotting BMI at different ages during childhood and converting BMI values into SD-scores (based on within-cohort data)

Measured BMI is plotted on the appropriate wavy line, at the child's age. The SD-score can be read off the y-axis at the same horizontal level. This procedure is repeated at later ages. 'Test-positive' is defined as any increase in SD-score between measurements and an SD-score above the median at the later measurement. The right-hand chart shows the BMI trajectory of 3 cohort children; children A is 'test positive' at ages 5-8 and 8-11; child B is 'test positive' at ages 5-8 and 11-14 and child C is 'test-negative' at all ages.

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Table 1

Childhood body mass index (BMI), adult anthropometry and risk factors, and prevalence of components of the metabolic syndrome and impaired glucose tolerance and diabetes

		MALES			FEMALES	
Childhood BMI (kg/m^2)	**N**	**Mean (SD) BMI**	**CDC†SD-score**	**N**	**Mean (SD) BMI**	**CDC SD-score**
2 years	831	**15.8** (1.2)	**-0.8**	600	**15.4** (1.2)	**-0.9**
5 years	861	**14.9** (1.0)	**-0.6**	622	**14.7** (1.1)	**-0.5**
8 years	857	**14.5** (1.3)	**-1.2**	619	**14.2** (1.2)	**-1.2**
11 years	828	**15.3** (1.7)	**-1.2**	600	**15.2** (1.8)	**-1.3**
14 years	874	**16.7** (2.4)	**-1.5**	628	**18.2** (2.7)	**-0.6**
Adult measurements	**N**	**Mean (SD)**		**N**	**Mean (SD)**	
Age (years)	884	**29.2**	(1.3)	634	**29.2**	(1.4)
Height (cm)	884	**169.7**	(6.4)	632	**154.9**	(5.7)
Weight (kg)	884	**71.8**	(14.0)	634	**59.3**	(13.4)
BMI (kg/m^2)	884	**24.9**	(4.3)	632	**24.7**	(5.1)
Waist circumference (cm)	884	**90.2**	(12.1)	634	**79.6**	(12.4)
Triglyceride concentration (mmol/l) *	868	**1.57**	(1.7)	623	**1.05**	(1.5)
HDL cholesterol concentration (mmol/l) *	869	**1.12**	(1.3)	621	**1.24**	(1.3)
Systolic blood pressure (mm Hg)	878	**118**	(11)	625	**107**	(11)
Diastolic blood pressure (mm Hg)	878	**78**	(10)	625	**73**	(9)
Fasting glucose concentration (mmol/l) *	869	**5.4**	(1.2)	623	**5.3**	(1.2)
Prevalence of adult metabolic syndrome components§ and IGT/diabetes	**N**	**No. Positive**	**%**	**N**	**No. Positive**	**%**
High waist circumference	884	454	**51**	634	289	**46**
Hypertriglyceridaemia	868	358	**41**	623	66	**11**
Low HDL cholesterol	869	297	**34**	621	345	**56**
High blood pressure or on treatment for hypertension	878	242	**28**	626	76	**12**
Glucose intolerance, fasting hyperglycaemia or known diabetes	869	359	**41**	623	228	**37**
Metabolic syndrome	869	265	**30**	623	114	**18**

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		MALES		FEMALES		
Diabetes	849	41	**5**	593	22	**4**
IGT	849	95	**11**	593	61	**10**
IGT or diabetes	849	136	**16**	593	83	**14**

[*] Geometric means and standard deviations

[†] SD-scores for childhood BMI according to CDC reference

[§] According to International Diabetes federation criteria10 (see Methods).

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Table 2

Percentage of subjects with metabolic syndrome and IGT or diabetes in young adult life according to (a) BMI SD-score and (b) *change* in BMI SD-score at different ages during childhood

(a) BMI SD-score												
Age (years)	**Within-cohort (Delhi) SD-Score for body mass index**								**Odds ratio per unit SD-score* (95% CI)**		**p value***	**CDC, OR per unit SD-score***
	Below -1		**-1 to 0**		**0 to +1**		**Above +1**					
Metabolic syndrome												
2	**23.8**	(181)	**21.7**	(530)	**27.5**	(516)	**30.4**	(181)	**1.20**	(1.04 to 1.37)	0.01	1.18
5	**19.1**	(188)	**24.3**	(506)	**26.0**	(534)	**31.4**	(229)	**1.19**	(1.05 to 1.35)	0.006	1.20
8	**17.7**	(209)	**22.4**	(558)	**27.6**	(486)	**37.6**	(197)	**1.36**	(1.21 to 1.53)	<0.001	1.41
11	**11.1**	(190)	**21.4**	(585)	**31.2**	(448)	**39.7**	(179)	**1.63**	(1.43 to 1.85)	<0.001	1.69
14	**10.8**	(231)	**19.4**	(588)	**33.5**	(463)	**42.3**	(194)	**1.87**	(1.65 to 2.12)	<0.001	1.78
IGT/Diabetes												
2	**21.6**	(176)	**16.1**	(510)	**12.1**	(496)	**13.6**	(177)	**0.84**	(0.71 to 1.00)	0.04	0.85
5	**17.3**	(185)	**16.8**	(487)	**13.9**	(518)	**12.4**	(218)	**0.89**	(0.76 to 1.04)	0.14	0.90
8	**17.4**	(207)	**15.4**	(538)	**12.6**	(468)	**18.1**	(188)	**1.00**	(0.86 to 1.16)	1.00	0.97
11	**16.8**	(184)	**14.2**	(564)	**12.9**	(433)	**22.0**	(173)	**1.13**	(0.97 to 1.31)	0.11	1.09
14	**12.9**	(225)	**14.0**	(563)	**14.2**	(450)	**23.2**	(190)	**1.22**	(1.06 to 1.41)	0.005	1.14

(b) Change in BMI SD-score												
Age (years)	**Change in within-cohort (Delhi) SD-score for body mass index**								**Odds ratio per unit change in SD-score* (95% CI)**		**p value***	**CDC, OR per unit change in SD-score***
	Drop >1		**Drop 0 to 1**		**Rise 0 to 1**		**Rise >1**					
Metabolic syndrome												
2-5	**20.0**	(170)	**26.0**	(497)	**27.4**	(507)	**21.4**	(206)	**1.03**	(0.90 to 1.18)	0.065	1.01
5-8	**21.2**	(113)	**23.5**	(681)	**26.1**	(537)	**40.0**	(100)	**1.35**	(1.15 to 1.59)	<0.001	1.31
8-11	**13.8**	(29)	**23.1**	(714)	**28.1**	(587)	**42.2**	(45)	**1.69**	(1.33 to 2.15)	<0.001	1.84
11-14	**25.9**	(27)	**21.5**	(656)	**28.8**	(681)	**36.4**	(33)	**2.04**	(1.57 to 2.66)	<0.001	1.97
IGT/Diabetes												
2-5	**11.0**	(164)	**16.7**	(484)	**13.7**	(489)	**17.4**	(195)	**1.02**	(0.86 to 1.20)	0.83	1.04
5-8	**13.6**	(110)	**14.2**	(657)	**15.3**	(518)	**23.7**	(97)	**1.25**	(1.02 to 1.52)	0.03	1.21
8-11	**10.3**	(29)	**12.8**	(680)	**16.6**	(574)	**31.8**	(44)	**1.74**	(1.30 to 2.32)	<0.001	1.60

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(b) Change in BMI SD-score												
Age (years)	**Change in within-cohort (Delhi) SD-score for body mass index**								**Odds ratio per unit change in SD-score* (95% CI)**		**p value***	**CDC, OR per unit change in SD-score***
	Drop >1		**Drop 0 to 1**		**Rise 0 to 1**		**Rise >1**					
11-14	**30.4**	(23)	**12.9**	(635)	**16.8**	(659)	**15.2**	(33)	**1.33**	(0.97 to 1.82)	0.08	1.21

*adjusted for sex and adult age by logistic regression; figures in parentheses are the number of subjects (not cases) in each cell.

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Table 3

Prediction of adult metabolic syndrome and IGT/diabetes

Starting age (years)	Finishing Age (years)	Metabolic Syndrome							IGT/Diabetes						
		'test positive' (%)	Sensitivity (%)	Specificity (%)	Attributable fraction (%)	Positive predictive value (%)	Negative predictive value (%)	Likelihood ratio	'test positive' (%)	Sensitivity (%)	Specificity (%)	Attributable fraction (%)	Positive predictive value (%)	Negative predictive value (%)	Likelihood ratio
Delhi reference															
5	8	26	32	76	9	32	77	1.4	26	30	75	6	18	86	1.2
	11	25	38	79	18	39	79	1.9	25	32	76	9	19	86	1.3
	14	28	45	78	24	41	81	2.0	29	37	73	12	20	87	1.4
8	11	23	30	80	9	34	77	1.5	23	29	78	7	19	86	1.3
	14	28	41	77	18	38	79	1.8	28	37	74	12	20	87	1.4
11	14	28	44	77	21	39	80	1.9	29	37	72	11	19	87	1.3
CDC reference															
5	8	11	17	91	7	40	76	2.0	11	14	90	3	19	85	1.3
	11	14	22	89	9	41	77	2.0	14	21	87	8	22	86	1.6
	14	25	39	79	18	39	79	1.9	25	34	76	11	20	87	1.4
8	11	21	30	82	11	36	77	1.6	22	27	79	7	19	86	1.3
	14	39	51	65	19	33	79	1.5	39	44	62	9	17	86	1.2
11	14	41	53	63	20	33	80	1.4	41	44	60	5	16	86	1.1
Overweight or obese (IOTF)															
	5	1	1	98	-1	11	74	0.3	1	1	99	0	13	85	0.8
	8	1	1	99	0	30	75	1.3	1	1	99	0	22	85	1.6
	11	1	2	99	1	37	75	1.7	1	2	99	1	22	85	1.6
	14	4	7	97	3	47	75	2.6	4	8	97	4	33	86	2.8
>0.67 SD above the within-cohort mean (>75th percentile)															
	5	26	31	76	7	31	76	1.3	25	23	74	-4	13	84	0.9
	8	22	32	81	12	36	78	1.6	22	25	78	3	17	85	1.1
	11	19	29	85	13	40	78	2.0	19	23	82	5	19	86	1.3
	14	21	35	84	18	43	79	2.2	21	26	80	7	19	86	1.3

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Definition of 'test-positive': An increase in BMI SD-scores between the starting and finishing ages AND a BMI SD-score at the finishing age of above 0 (Delhi reference) or above minus 1 (CDC reference). 'Overweight or obese' defined according to International Obesity Task Force (IOTF) criteria14.