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Parental history of type 2 diabetes mellitus, metabolic syndrome, and cardiometabolic risk factors in Asian Indian adolescents

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Abstract

The objective was to study the influence of parental history of type 2 diabetes mellitus on prevalence of the metabolic syndrome (MS) and other cardiometabolic risk factors in Asian Indian adolescents. Adolescents aged 12 to 19 years (N = 321) were recruited from the Chennai Urban Rural Epidemiology Study. Based on parental diabetic status, 3 groups were studied: group 1, offspring of parents with normal glucose tolerance (n = 105); group 2, offspring of 1 diabetic parent (n = 114); and group 3, offspring of 2 diabetic parents (n = 102). Subjects underwent blood pressure and anthropometric measurements as well as an oral glucose tolerance test and a fasting lipid profile. Metabolic syndrome was diagnosed using the International Diabetes Federation definition. Body mass index ($P < .001$) and waist and hip circumference ($P < .05$ for group 2 and $P < .001$ for group 3) were significantly higher in groups 2 and 3 compared with group 1. High-density lipoprotein cholesterol was significantly lower in groups 2 and 3 compared with group 1 ($P < .05$). Serum triglycerides were significantly higher in group 3 ($P < .05$) compared with the other 2 groups. Adolescents in group 3 ($P < .001$) and group 2 ($P < .05$) were significantly more overweight and had more abdominal obesity compared with those in group 1. Impaired fasting glucose and impaired glucose tolerance were also significantly higher in group 3 compared with the other 2 groups. High blood pressure showed an increasing trend from group 1 to group 3 (P for trend < .05). Two metabolic abnormalities were present in 7.6%, 14.9%, and 22.5% of adolescents in groups 1, 2, and 3, respectively (trend χ^2: 9.04, $P = .003$). Prevalence of MS was higher in groups 2 and 3 compared with group 1 but did not reach statistical significance because of small numbers. The cardiometabolic profile of the parents was similar to that of the adolescents. Parental history of type 2 diabetes mellitus increases risk of not only glucose intolerance but also other cardiometabolic risk factors like overweight, low high-density lipoprotein cholesterol, and high blood pressure in Asian Indian adolescents.

1. Introduction

The term *metabolic syndrome* (MS) refers to a clustering of cardiometabolic risk factors including abdominal obesity, glucose intolerance, dyslipidemia, and hypertension [1]. Metabolic syndrome has been recognized as a risk factor for cardiovascular disease (CVD) and mortality [2]. Asian Indians are known to have very high rates of type 2 diabetes mellitus [3] and CVD [4]. Obesity and a family history of type 2 diabetes mellitus are associated with increased risk of type 2 diabetes mellitus in youth, irrespective of ethnic background [5,6]. Findings from the Framingham Offspring Study show that individuals with 1 diabetic parent have an odds ratio of 3.5 for developing diabetes that further increases to 6.1 if both parents are affected [7]. The heritability of MS has earlier been shown in adults [8-10]. Recent studies also suggest that a parental history of diabetes may be associated with cardiometabolic abnormalities in their adult offspring [11-13]. However, there are very few studies on this aspect in children and adolescents worldwide [14-16]. In Asian Indian adolescents, although there are a few studies on prevalence and risk factors of MS [17-19], there are no studies to date on parental history of diabetes and MS. In the present study, we looked at the influence of parental history of type 2 diabetes mellitus on the

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prevalence of MS and cardiometabolic risk factors in Asian Indian adolescents.

2. Study design

Subjects for this study were recruited from April 2004 to April 2006 from the Chennai Urban Rural Epidemiology Study (CURES), a cross-sectional epidemiologic study in Chennai city in south India that sampled 26 001 individuals aged at least 20 years based on a systematic random sampling technique. The detailed study design of CURES is described elsewhere [20], and the sampling frame is shown in our Web site (http://www.drmohansdiabetes.com/mdrf/CURES.pdf).

For the current study, participants from CURES (parents) were asked if they had an adolescent aged 12 to 19 years in their household. If they answered yes, they were briefed about the study and requested to bring the adolescent to Dr. Mohan's Diabetes Specialities Centre, a tertiary center for diabetes, for enrollment in the study. A total of 403 families had adolescents in the age group of 12 to 19 years, of which 385 families could be contacted (95.5% response rate). Fifty-five families with parents having impaired glucose tolerance (IGT) were excluded from the study. Of the remaining 330 families, 321 families participated in the study. As we studied only 1 adolescent per family (proband), we had 321 adolescents.

Institutional ethical committee approval was obtained, and informed consent was obtained from all study subjects at least 18 years of age or from the parent if the subjects were younger than 18 years. The adolescents as well their parents were administered a detailed questionnaire to collect information regarding their demographic, socioeconomic, diabetic, and general health status.

The adolescents (N = 321, 50.8% male) were then assigned to 1 of 3 groups based on their parental diabetic status: group 1, offspring of parents both of whom had normal glucose tolerance (NGT) (n = 105); group 2, offspring of 1 diabetic parent (n = 114) and the other with NGT; and group 3, offspring of 2 diabetic parents (n = 102). Diagnosis of type 2 diabetes mellitus was accepted in parents if they had been diagnosed and were on treatment for the same by a physician. In all remaining parents, an oral glucose tolerance test was performed; and the World Health Organization (WHO) criteria were used for diagnosis of diabetes. Parents with type 1 diabetes mellitus and gestational diabetes were not included in the study. All the adolescents enrolled in the study also underwent an oral glucose tolerance test using 1.75 g of glucose per kilogram body weight, but not exceeding 75 g as per the WHO criteria. A fasting venous sample was obtained for both parents and adolescents, after ensuring 8 hours of overnight fast, for estimation of plasma glucose and serum lipids (using Hitachi 912 Autoanalyzer [Roche Diagnostics, Mannheim, Germany] with kits supplied by Roche Diagnostics).

Blood pressure (BP) and anthropometric measurements for both parents and adolescents were obtained using standardized techniques by 2 trained interviewers [20]. Inter- and intraobserver coefficients of variation were less than 5%.

Height was measured with a tape to the nearest centimeter. Subjects were requested to stand upright without shoes with their back against the wall, heels together, and eyes directed forward.

Weight was measured with a spring balance that was kept on a firm horizontal surface. Subjects wore light clothing and stood upright without shoes, and weight was recorded to the nearest 0.5 kg. The scale was calibrated every day with "standard" weights.

Body mass index (BMI) was calculated using the formula weight (in kilograms)/height (in meters)^2.

Waist circumference was measured using a nonstretchable measuring tape. The subjects were asked to stand erect in a relaxed position with both feet together on a flat surface. Waist girth was measured as the smallest horizontal girth between the costal margins and the iliac crests at minimal respiration. Measurements were made to the nearest 0.1 cm.

Hip circumference was taken as the greatest circumference at the level of greater trochanters (the widest portion of the hip) on both sides. Measurements were made to the nearest 0.1 cm.

For both waist and hip circumference, 2 measurements were made; and the mean of the 2 readings was taken as the final value.

Blood pressure (BP) was recorded in the sitting position in the right arm to the nearest 1 mm Hg using the electronic OMRON machine (Omron, Tokyo, Japan). Two readings were taken 5 minutes apart, and the mean of the 2 readings was taken. If the difference between the first and the second reading was greater than 10 mm Hg for systolic pressure and/or greater than 6 mm Hg for diastolic pressure, then a third measurement was made; and the mean of all 3 measurements was taken as the BP.

3. Definitions

Normal glucose tolerance (NGT): Subjects were confirmed to have NGT if the fasting plasma glucose was less than 100 mg/dL (<5.6 mmol/L) and 2-hour postload plasma glucose (2-h PG) was less than 140 mg/dL (<7.8 mmol/L) [21].

Impaired glucose tolerance (IGT): IGT was diagnosed if the 2-h PG was at least 140 mg/dL (≥7.8 mmol/L) and less than 200 mg/dL (<11.1 mmol/L) [21].

Impaired fasting glucose (IFG): IFG was diagnosed if fasting plasma glucose was at least 100 mg/dL (≥5.6 mmol/L) and less than 126 mg/dL (<7.0 mmol/L) based on the American Diabetes Association definition [22].

Diabetes: Diagnosis of diabetes was based on the WHO Consulting group criteria [21], that is, fasting plasma glucose of at least 126 mg/dL (≥7.0 mmol/L) and/or 2-h PG of at

least 200 mg/dL (≥11.1 mmol/L) or self-reported diabetes on treatment with a physician(for parents).

High BP: High BP was diagnosed in parents and adolescents if the BP was at least 130/85 mm Hg (for adolescents) and/or based on drug treatment of hypertension (for parents) [23].

Generalized obesity: Generalized obesity was defined using the WHO Asia-Pacific definition [24] as BMI of at least 25 kg/m^2 (for parents). For adolescents, *overweight* was defined based on standard definitions for child overweight given by Cole et al [25].

Abdominal obesity: Abdominal obesity was defined based on the recent International Diabetes Federation (IDF) definition for children and adolescents, that is, waist circumference of at least 89 cm for males and at least 80 cm for females of ages 12 to 16 years and at least 90 cm for males and at least 80 cm for females older than 16 years [26]. For parents, modified WHO Asia-Pacific guidelines for abdominal obesity, that is, at least 90 cm for men and at least 80 cm for women, were used [24].

Hypercholesterolemia: Hypercholesterolemia was diagnosed in parents and adolescents if serum cholesterol levels were at least 200 mg/dL (≥5.2 mmol/L) or if they were under drug treatment of hypercholesterolemia (for parents) [23].

Hypertriglyceridemia: Hypertriglyceridemia was diagnosed in parents and adolescents if serum triglyceride levels were at least 150 mg/dL (≥1.7 mmol/L) or if they were under drug treatment of hypertriglyceridemia (for parents) [23].

Low high-density lipoprotein (HDL) cholesterol: Low HDL cholesterol was diagnosed if levels were less than 40 mg/dL (<1.04 mmol/L) for adolescents aged 12 to 16 years. For adolescents older than 16 years and for parents, levels of less than 40 mg/dL (<1.04 mmol/L) for males and less than 50 mg/dL (<1.3 mmol/L) for females were used [26,27].

Metabolic syndrome: Metabolic syndrome was defined based on the IDF definition for children and adolescents in those subjects aged 12 to 16 years [26] and the IDF adult definition in those older than 16 years [27]. A subject was classified as having the MS by the presence of abdominal obesity plus the presence of 2 or more of the following clinical features: fasting blood glucose (≥100 mg/dL), high BP (≥130/85 mm Hg), elevated triglycerides (≥150 mg/dL), and reduced HDL cholesterol (HDL cholesterol <40 mg/dL for 12-16 years, and <40 mg/dL for males and <50 mg/dL for females >16 years).

4. Statistics

Statistical analyses were performed using SPSS for Windows version 10.0 software (SPSS, Chicago, IL). Preliminary descriptive analyses were conducted to check for the distribution of the variables of interest. Age adjustment was done using linear regression method. Values were expressed as mean ± SD except for income where mean ± SEM was used. Student *t* test or 1-way analysis of variance, as appropriate, was used to compare continuous variables; and the χ^2 test was used to compare proportions among groups. *P* value of less than .05 was considered significant.

5. Results

The clinical and biochemical characteristics of the adolescents (after age adjustment) are shown in Table 1. There was no significant difference in the mean age or sex distribution among the 3 study groups. Adolescents in groups 2 and 3 had significantly higher BMI ($P < .001$), waist circumference ($P < .05$ for group 2 and $P < .001$ for

Table 1
General characteristics of adolescents in the 3 categories

Variables	Adolescents		
	Group 1 (both parents nondiabetic) (n = 105)	Group 2 (1 parent diabetic) (n = 114)	Group 3 (both parents diabetic) (n = 102)
Male n (%)	62 (59.0)	53 (46.5)	48 (47.1)
Age (y)	15 ± 2	16 ± 2	16 ± 2
BMI (kg/m^2)	18.1 ± 3.7	20.5 ± 4.1†	22.3 ± 5.1†,‡
Waist circumference (cm)	67.6 ± 9.7	72.7 ± 10.7*	74.2 ± 11.8†
Hip circumference (cm)	81.6 ± 12.5	87.4 ± 12.2*	90.3 ± 14.1†
Waist-to-hip ratio	0.83 ± 0.10	0.83 ± 0.09	0.82 ± 0.10
Systolic BP (mm Hg)	105 ± 12	107 ± 13	107 ± 13
Diastolic BP (mm Hg)	65 ± 8	66 ± 9	67 ± 10
Fasting plasma glucose (mg/dL)	86 ± 7	88 ± 17	90 ± 16
Total cholesterol (mg/dL)	135 ± 23	136 ± 23	141 ± 28
Serum triglycerides (mg/dL)	78 ± 36	79 ± 33	90 ± 37*,‡
HDL cholesterol (mg/dL)	44 ± 10	41 ± 8*	41 ± 9*
Non-HDL cholesterol (mg/dL)	85 ± 23	88 ± 22	93 ± 27*
LDL cholesterol (mg/dL)	75 ± 21	79 ± 20	81 ± 23

All variables adjusted for age. LDL indicates low-density lipoprotein.

* $P < .05$ and

† $P < .001$ compared with adolescents with both parents nondiabetic.

‡ $P < .05$ compared with adolescents with 1 parent diabetic.

Table 2
Age-adjusted prevalence of cardiometabolic risk factors among the adolescents

Variables	Adolescents		
	Group 1 (both parents normal) (n = 105)	Group 2 (1 parent diabetic) (n = 114)	Group 3 (both parents diabetic) (n = 102)
IFG (FBG ≥100 mg/dL) n (%)	5 (4.8)	2 (1.8)	8 (7.8)‡
IGT (2-h PG ≥140 mg/dL and <200 mg/dL) n (%)	1 (1.0)	2 (1.8)	8 (7.8)*,‡,§
Diabetes (FBG ≥126 and/or 2-h PG ≥200 mg/dL) n (%)	0	1 (0.9)	1 (1.0)
High BP (BP ≥130/≥85 mm Hg) n (%)	2 (1.9)	5 (4.4)	8 (7.8)§
Overweight (BMI)[a] n (%)	12 (11.4)	26 (22.8)*	42 (41.2)†,‡,‖
Abdominal obesity (WC: male ≥89 cm, female ≥80 cm for 12-16 y; and male ≥90 cm, female ≥80 cm for those >16 y) n (%)	2 (1.9)	16 (14.0)*	20 (19.6)†,‖
Hypercholesterolemia (≥200 mg/dL) n (%)	0	0	2 (2.0)
Hypertriglyceridemia (≥150 mg/dL) n (%)	6 (5.7)	6 (5.3)	6 (5.9)
Low HDL cholesterol (<40 mg/dL for those between 12-16 y; and male <40 mg/dL, female <50 mg/dL for those >16 y) n (%)	40 (38.1)	62 (54.4)*	54 (52.9)*,§

WC indicates waist circumference; FBG, fasting blood glucose.
[a] Based on Cole et al [25].
* $P < .05$ and
† $P < .001$ compared with adolescents with both parents normal.
‡ $P < .05$ compared with adolescents with 1 parent diabetic.
§ P for trend < .05.
‖ P for trend < .001.

group 3), and hip circumference ($P < .05$ for group 2 and $P < .001$ for group 3) compared with those in group 1, with a linear increase from group 1 to group 3. Although fasting plasma glucose, total cholesterol, and low-density lipoprotein cholesterol showed a similar linear trend, the differences did not reach statistical significance. Group 3 had significantly higher serum triglycerides ($P < .05$) compared with groups 1 and 2, whereas HDL cholesterol was significantly lower in groups 2 and 3 compared with group 1 ($P < .05$). Adolescents in group 3 had a significantly higher non-HDL cholesterol compared with those in group 1 ($P < .05$).There was no significant difference in the mean systolic and diastolic BP and waist-to-hip ratio between the study groups. There was no association of any of the cardiometabolic factors with socioeconomic status. The mean incomes were (mean ± SEM) as follows: group 1, Rs 12 937 ± 2878; group 2, Rs 9640 ± 1064; and group 3, Rs 14 015 ± 1407.

Table 2 shows the age-adjusted prevalence of various cardiometabolic risk factors among the adolescents. Prevalence of IFG was significantly higher in group 3 compared with group 2 ($P < .05$). Prevalence of IGT was also significantly higher in group 3 compared with the other 2 groups ($P < .05$, P for trend < .05). One individual each in groups 2 and 3 was diagnosed to have type 2 diabetes mellitus. Prevalence of high BP showed an increasing trend from group 1 to group 3 (P for trend < .05). Adolescents in group 3 were significantly more overweight (41.2%) compared with those in group 2 (22.8%) ($P < .05$), who in turn were more overweight than those in group 1 (11.4%) ($P < .05$, P for trend < .001). Abdominal obesity was also significantly higher in groups 2 and 3 compared with group 1 ($P < .05$ for group 2 and $P < .001$ for group 3, P for trend < .001). Prevalence of low HDL levels was significantly higher in groups 3 and 2 compared with group 1 ($P < .05$, P for trend < .05). There was no significant difference in

Table 3
Age-adjusted prevalence of various metabolic abnormalities in the adolescents

Metabolic abnormalities	Adolescents			Trend χ^2, P value
	Group 1 (both parents normal) (n = 105)	Group 2 (1 parent diabetic) (n = 114)	Group 3 (both parents diabetic) (n = 102)	
Any 1 abnormality n (%)	45 (42.9)	69 (60.5)	70 (68.6)	14.06, $P < .0001$
Any 2 abnormalities n (%)	8 (7.6)	17 (14.9)	23 (22.5)	9.04, $P = .003$
3 Abnormalities n (%) (abdominal obesity + any 2 metabolic abnormalities–MS [IDF criteria])	0	3 (2.6)	3 (2.9)	2.45, $P = .117$

Table 4
Prevalence of cardiometabolic risk factors among the parents

Variables	Parents		
	Group 1 (both normal) (n = 210)	Group 2 (1 diabetic) (n = 228)	Group 3 (both diabetic) (n = 204)
High BP (BP ≥130/≥ 85 mm Hg) n (%)	47 (22.4)	55 (24.1)	50 (24.5)
Generalized obesity (BMI ≥25 kg/m^2) n (%)	105 (50.0)	137 (60.1)*	135 (66.2)*,§
Abdominal obesity (WC: male ≥ 90 cm, female ≥ 80 cm) n (%)	132 (62.9)	162 (71.1)	154 (75.1)*,§
Hypercholesterolemia (≥200 mg/dL) n (%)	43 (20.5)	44 (19.3)	52 (25.5)
Hypertriglyceridemia (≥150 mg/dL) n (%)	48 (22.9)	65 (28.5)	99 (48.5)†,‡,‖
Low HDL cholesterol (male <40 mg/dL, female <50 mg/dL) n (%)	138 (65.7)	159 (69.7)	149 (73.0)

* $P < .05$ and
† $P < .001$ compared with both parents normal.
‡ $P < .001$ compared with 1 parent diabetic.
§ P for trend < .05.
‖ P for trend < .001.

the prevalence of hypercholesterolemia or hypertriglyceridemia between the study groups. There was no sex difference in any of the metabolic abnormalities studied in the 3 groups. Duration of diabetes in parents was 6.4 ± 4.6 years for adolescents in group 2 and 8.8 ± 4.2 years for those in group 3.

Table 3 shows that 7.6%, 14.9%, and 22.5% of adolescents in groups 1, 2, and 3, respectively, had at least 2 metabolic abnormalities (trend χ^2: 9.04, $P = .003$) and that 2.6% and 2.9% of adolescents in groups 2 and 3, respectively, had the MS as diagnosed by the IDF criteria for adolescents (trend χ^2:2.45, $P = .117$, not significant).

Prevalence of cardiometabolic risk factors among the parents is presented in Table 4. Similar to the profile of the adolescents, generalized obesity and abdominal obesity were significantly higher in the parents of group 3 compared with those of the other 2 groups ($P < .05$, P for trend < .05). Hypertriglyceridemia was also significantly higher in parents of group 3 compared with those of the other 2 groups ($P < .001$, P for trend < .001). Although high BP and low HDL cholesterol were higher in parents of groups 2 and 3 compared with those in parents of group 1, they did not reach statistical significance. Hypercholesterolemia was not significantly different between the 3 groups.

6. Discussion

To our knowledge, this study is the first, in a south Asian population, to have looked at parental history of type 2 diabetes mellitus in relation to prevalence of cardiometabolic risk factors and MS in adolescent offspring. The study shows that a parental history of type 2 diabetes mellitus increases the risk of not only glucose intolerance but also other cardiometabolic risk factors like obesity, low HDL cholesterol, and hypertension in Asian Indian adolescents. Earlier studies have shown that the risk for developing type 2 diabetes mellitus increases when 1 or both parents are affected [28,29]. Although this association has been widely explored in adults, there is paucity of such data in adolescents. In this study, we found that 2 individuals (1 each from groups 2 and 3) had developed type 2 diabetes mellitus, in addition to a significant number with prediabetes (IGT and IFG). The emergence of type 2 diabetes mellitus in adolescence has important implications for both the health of the individual and the nation's health service resources. Studies have also shown that psychologic health is often poor in adolescence [30]; and with the added stress of the physical changes of puberty, treatment compliance is often very difficult [31]. A recent study among urban south Indian schoolchildren reported that 67.7% of children had 1 or more cardiometabolic abnormalities [18]. Other studies have documented cardiometabolic risk factor clustering in adolescence [32-34]. However, none of these studies specifically looked at parental history of diabetes in relation to cardiometabolic risk factors in the offspring.

The Japanese American Family Study is an investigation of risk factors for coronary heart disease, type 2 diabetes mellitus, and MS in 68 Japanese-American families [10]. This study showed that all the individual components of the MS had a significant genetic component, with the highest heritability being for triglycerides and HDL cholesterol. Similar results of heritability have been shown in a study done by Edwards et al [35], which showed that body mass, fat distribution, insulin, glucose, and lipids were all heritable risk factors. Another study, which looked at the heritability of MS, is the Northern Manhattan family study [9], which found that the MS had a heritability of 24%. Similar studies have been done in Omani [36], Dutch [37], German [38], British [39], and Mexican [40] populations. However, all these studies have been done in adults. Literature in children and adolescents is varied and few.

The Chin-Shan community family study [41] showed familial aggregation of the MS in Chinese adolescents. In another study, Brazilian adolescents with a family history of type 2 diabetes mellitus were stratified into 3 groups: G_0,

normal; G_1, overweight; and G_2, obese. It was found that none of the subjects in G_0 and G_1 had the MS, whereas in G_2, the prevalence was 26.1% [42]. Similar results were found in a study done in Turkey by Altinli et al [14]. Here the children were divided into 3 groups based on diabetic parentage: group 1, offspring of parents with type 1 diabetes mellitus; group 2, offspring of parents with type 2 diabetes mellitus; and group 3, control group. It was found that children in group 1 were no different from the control group. However, the children in group 2 (offspring of parents with type 2 diabetes mellitus) had various cardiometabolic abnormalities. Another group in Malaysia [15] also showed that the MS was evident in nonobese children of diabetic Malays.

Earlier studies from India have shown that, even in nondiabetic offspring of diabetic parents, abnormalities of insulin secretion as well as insulin resistance could be present [43,44]. An earlier study from our group showed that, if both parents were diabetic, there was an increased risk of developing type 2 diabetes mellitus in the offspring [45]. However, that study was done in adult offspring. The present study shows that, among offspring of diabetic parents, various components of the MS appear to manifest in adolescence itself. This suggests that a parental history of type 2 diabetes mellitus may increase risk of not only diabetes but also of other cardiometabolic risk factors (with specific reference to overweight, low HDL cholesterol, and high BP) in the offspring. This clustering of cardiometabolic risk factors in adolescence tracks into adulthood, suggesting that the early diagnosis of MS might identify adolescents at increased risk of premature CVD [46-50]. This underscores the need for risk factor identification and their management even in adolescence.

This study has certain limitations. Firstly, the number of individuals studied were small; and hence, these results have to be viewed with caution. Secondly, the cross-sectional nature of the design does not allow for cause-effect relationships to be made. Finally, this study does not show true heritability because only phenotypic associations have been studied, which may be a combination of heritability and shared environment. Indeed, the increased prevalence of cardiometabolic abnormalities among parents who were diabetic suggests that environmental factors may well play a role in the association noted in the adolescents. However, the increased prevalence of metabolic abnormalities in the adolescents in groups 2 and 3 could also be explained by the presence of diabetes in the parents because, by definition, 1 or both parents had diabetes in these groups. It is well known that type 2 diabetes mellitus is associated with cardiometabolic risk factors. However, in the adolescent offspring, the increased prevalence of cardiometabolic risk factors cannot be explained by diabetes per se because only 2 adolescent subjects had diabetes. Only genetic studies will prove whether it is the diabetes per se or the cardiometabolic risk factors in the parents that tracked down to the adolescents.

In conclusion, there is an increase in the prevalence of cardiometabolic risk factors in the offspring of Asian Indian diabetic parents, even during adolescence. Offspring of diabetic parents should therefore be screened not only for type 2 diabetes mellitus but also for other cardiometabolic risk factors. Changes in dietary habits and increase in physical activity could help prevent or at least delay the development of these risk factors and thereby help reduce morbidity and mortality due to CVD later in life.

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