Cardiovascular disease risk factors in 5-year-old urban South African children — the Birth to Ten study

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Background. A birth cohort study, the Birth to Ten (BTT) study, commenced in the greater Johannesburg/Soweto metropole in South Africa in 1990. The overall BTT project collected antenatal, birth and early development information on these children as well as information that could help identify factors related to the emergence of risk of cardiovascular diseases (CVDs) in children.

Objective. To determine CVD risk profiles and their determinants in 5-year-old children living in an urban environment in South Africa.

Methods. Demographic and birth characteristics were collected on a sample of 964 5-year-olds whose parents agreed for blood samples to be taken from their children. The children's height and weight were measured using standardised procedures; blood pressure (BP) was measured with a Dinamap Vital Signs Monitor, and a non-fasting blood sample was drawn for lipid determinations. Information on exposure to tobacco smoke and additional health-related data were obtained by interview.

Results. No differences were found between the birth weight and gestational age of the 5-year-old CVD participants and the remainder of the children studied at birth. The systolic BP was significantly different between ethnic groups, with the BP of the black children significantly higher than that of the Indian and white children, while the diastolic BP of

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black children was also the highest. White children had the highest mean total cholesterol (TC) and low-density lipoprotein cholesterol (LDLC) levels, significantly higher than those in the black community. The coloured children's TC level was also significantly lower than that of the whites, while the LDLC level of the Indian children was significantly higher than that of the blacks. Overall, 64% of the children were exposed to environmental tobacco smoke (ETS), with the white group having the lowest rate (45% exposed to ETS). The coloured children were most frequently exposed to ETS, with 40.6% having primary caregivers who smoked; of these children 42% lived in homes with two or more smokers.

Conclusions. Tobacco control legislation will protect South Africans against tobacco sales promotions. This will be the first step towards increasing the priority of chronic disease prevention, health promotion and appropriate care for chronic diseases and their risk factors on the South African health policy agenda. The groups of children that were studied carried differing but significant levels of CVD risk. This suggests that the promotion of a healthy lifestyle should start in childhood, and should target the risk factors found in each group.

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It has been demonstrated that risk factors for cardiovascular diseases (CVDs) emerge at a young age in populations with high CVD rates.[1-4] However, an incubation period of 30 - 40 years is required for CVD to manifest in such communities. Furthermore, the World Health Organisation (WHO)/World Bank Global Burden of Disease (GBD)[5] study has predicted that CVD will be one of the major causes of death in developing countries by the year 2020. Should this prediction be correct, it suggests that the children who will present with high CVD rates by 2020 should currently have high risk factor levels for CVD. It would, therefore, be important to identify such children to ensure that appropriate interventions are implemented to prevent the GBD prediction. Currently there is a dearth of information on the risk profile of children in developing countries.

The epidemiological transition (from traditional living patterns to typical westernised lifestyles) that takes place in countries undergoing development is associated with a change from a low CVD level to very high risk factor levels preceding the eventual emergence of high CVD rates as predicted by the GBD study.[6] In South Africa a unique pattern of CVD risk profiles has been recorded in previous studies among adults, adolescents and 11-year-old children.[7-14] As expected, the adverse CVD risk profiles were found in those groups who had high CVD mortality rates, while the opposite was true for those groups with low levels of CVD risk.[12,13]

A birth cohort study, the Birth to Ten (BTT) study, conducted in the greater Johannesburg/Soweto metropole in South Africa from 1990, presented an ideal opportunity to study CVD risk profiles and determinants in preschool South African children.[15-17] The overall BTT study had collected antenatal, birth and early development information on the children to identify determinants of their growth, health and development. These data would allow for the identification of factors related to the emergence of CVD risk factors in children.

In the South African setting ethnicity is often a proxy for socio-economic differences between groups of people. However, it is important to identify groups that have different biological as well as environmentally determined risk profiles in order to ensure that groups with high risk for chronic diseases of lifestyle are identified and specifically targeted for appropriate interventions. A study by Brancati *et al.*[18] in the USA showed that after correcting for socio-economic indicators being African-American was an independent risk factor for diabetes. However, an interaction was found between being African-American and socio-economic status. If similar results are found in South Africa, then it will be inappropriate to assume that as socio-economic differences between groups are corrected, chronic disease risk profile differences will disappear. The objective of this study was to identify the CVD risk profile and associated factors in 5-year-old children living in an urban environment in South Africa. In addition, multiple regression analyses of the data allow for an assessment of the individual contributions of social class and ethnicity to the observed CVD risk profile of 5-year-old children in a South African city.

METHODS

BTT is a prospective longitudinal cohort study. The cohort was made up of singleton infants born during a 7-week period between April and June 1990 to women who gave their permanent address within the defined area. Identification and enrolment of children born during this 7-week period and living in the defined areas took place throughout the first year of the study. There were 4 029 births enrolled.[15,16] This probably represented more than 80% of all births that occurred in the defined area. The coverage of the cohort was, therefore, virtually complete as earlier pilot studies had found that approximately 20% of mothers who deliver babies in this metropolitan area are from rural areas, travelling to the metropole to give birth and then returning home shortly after delivery.[17]

The children were studied annually. In 1995 when they were 5 years old, the children and their caregivers were invited to attend interviews, which provided a selected cross-sectional sample drawn from the original cohort. The interviews took place at the children's local primary health care centre, where their height and weight were measured using standardised procedures. Blood pressure (BP) was measured in a separate

room, using a Dinamap Vital Signs Monitor (1846SX) and an appropriate cuff size. After 5 minutes at rest, the BP was taken in triplicate, and the lowest diastolic BP recorded with its matching systolic pressure was used in the analysis. Children with severe hypertension (BP ≥ 124/84 mmHg) were referred to their health care provider.

A non-fasting blood sample for lipid determinants was collected with minimal stasis. The children were prepared for the venesection by using local anaesthetic plasters and by playing TV programmes to distract them. A professional nurse interacted with the children in a supportive manner. The blood was placed into ethylene diamine tetra-acetic acid tubes, kept on ice and centrifuged within 6 hours at 4°C to separate the plasma. The plasma was kept at 4°C and analysed within 24 hours. The total cholesterol (TC) and high-density lipoprotein cholesterol (HDLC) levels were measured on a Gilford auto-analyser. HDLC was determined after the precipitation of the apolipoprotein B-containing lipoproteins using manganese chloride/heparin. The high performance CHOD-PAP enzymatic colorimetric kit (Boehringer Mannheim) was used to determine plasma TC and HDLC. Non-fasting triglyceride (TG) levels were determined using the Boehringer Mannheim enzymatic Peridochrom method. In each case the Gilford auto-analyser was calibrated against Preciset Cholesterol. Precinorm L was used as an external control and pooled plasma as an internal control.

Caregivers were interviewed and detailed questionnaires were completed. These contained socio-demographic questions, including questions on living conditions within the child's home, the child's exposure to tobacco smoke and additional health-related factors.

Analyses of the data

The questionnaire responses and data sheets were encoded in duplicate. The Statistical Analysis System (SAS) package was used to calculate the means and standard deviations of variables. The prevalence rates of a variety of conditions and categorisation of data were calculated according to the criteria set out below. Socio-economic status (SES) was assessed using two indicators, namely: (*i*) the mother's level of education; or (*ii*) an SES score defined as follows: no electricity in the house (SES = 1), electricity and access to a car (SES = 2), electricity, access to a car and a washing machine (SES = 3), electricity, access to a car, washing machine and private medical insurance (SES = 4). Children with other combinations of the SES score elements were classified according to the highest element of a reported score.

Body mass index (BMI) was calculated as the ratio of weight (kg) over height (m), squared. The LDLC level was calculated using Friedewald's equation, namely LDLC = TC – HDLC – TG/2.18 mmol/l. This calculation was only done for subjects whose plasma TG levels were below 4.5 mmol/l, a requirement of this equation.[19] The prevalence rates of hypertension, categorised as high/normal blood pressure (≥ 108/70, but < 115/75 mmHg), significantly raised by blood pressure (≥ 115/75, but < 124/84 mmHg) and severe hypertension (≥ 125/84 mmHg), were calculated using the cut-off points for 3 - 5-year-old children as proposed by the fifth Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure.[20] The prevalence of borderline and high TC and LDLC levels was calculated using the levels suggested by the National Cholesterol Education Programme for children and adolescents.[21]

The associations between pairs of categorical variables were examined using the chi-square test. Analysis of variance was applied when the explanatory variable was categorical and the response variable continuous. When significant differences were found, the Student-Newman-Kuels multiple comparison method was used to determine which means differed significantly. The Pearson's correlation coefficient was used to investigate relationships among the continuous variables examined.

Multiple regression analysis was used to test for independent effects of the explanatory variables on a number of CVD risk factors. Forward and backward selection procedures were used in separate analyses. Height, birth weight, BMI, gestational and maternal age and breast-feeding were all treated as continuous independent variables. Socio-economic status, ethnic group, the mother's years of schooling (categorised as 7, 8 - 9, 10 - 11 and 12 years), breast-feeding and number of regular smokers in the household (categorised as no smokers, 1 smoker or more than 1 smoker in the home) were treated as categorical independent variables. The systolic and diastolic BPs, TC, LDLC and HDLC at 5 years were used as continuous dependent variables in separate analyses. All interactions of the continuous independent variables with the categorical independent variables were examined, as well as the interactions between the categorical independent variables. These interactions allow for the relationship of the continuous independent variables with the dependent variables to differ among the levels of the categorical independent variables. A final model describing the data was obtained, using backward elimination of statistically non-significant terms using a 5% significance level.

RESULTS

Demographic and birth characteristics of the 964 5-year-old children (5-year participants) who were investigated are shown in Table I. Characteristics of those children who did not participate at age 5 years (non-participants) are also shown. The Table clearly shows that an overall poor response rate was achieved, particularly for the Indian and white children. Consequently, data for these groups should be interpreted with caution. There were no differences between the birth weights

and gestational ages of the 5-year-old participants and those of the non-participants. However, compared with the non-participating mothers, the mothers of the participating 5-year-old children were significantly younger, had a higher level of education, fewer lived in shacks, hostels or single rooms and fewer lived with their partners at the time of their child's birth . A significant difference between the two groups of mothers at both extremes of the SES indicator suggests that more middle-class mothers participated in the follow-up study in 1995.

No significant differences were found when comparing the birth data of the ethnic groups. However, both black male and female infants had significantly lower gestational ages than the coloured males and females (black and coloured male gestational age, 37.9 v. 38.7 weeks, $P < 0.05$; black and coloured female gestational age, 37.9 v. 39.3 weeks, $P < 0.05$).

The CVD risk factor profile of the 5-year-old children is given in Table II. The mean systolic and diastolic BPs were significantly higher in the black children compared with those of some of the other groups. Of the black children 23.9% had significant or severe hypertension, followed by the coloured children, 14% of whom fell into these categories. The white and Indian children had lower rates of raised BP.

The highest mean TC and LDLC levels were found in the white children; these levels were significantly higher than those of the black children. The coloured children's TC level was also significantly lower than that of the whites, while the LDLC level of the Indian children was significantly higher than that of the blacks. Although the HDLC levels were similar in all the groups, the ratio of % HDLC/TC was significantly higher in the black than the white children. Only 13.7% of black children had LDLC levels that put them at risk of developing CVD in later life, compared with about 30% of the white and Indian children.

The white children were significantly taller and heavier than the other children, while the Indian group had significantly lower BMIs than the other three groups.

The children's exposure to environmental tobacco smoke (ETS) and their own experimentation with smoking are shown in Table III. About 64% of all the children were exposed to ETS, with the white children reported to be the least exposed. The coloured children were most frequently exposed to ETS, with 40.6% of them having primary caregivers who smoked; of these children 42% lived in homes with two or more smokers. The figures for the Indian, black and white communities were 33.4%, 24.4% and 13.6% respectively. Overall, at the age of 5 years, 6.7% of all the children had experimented with cigarettes. The children in the coloured population reported this most frequently.

The results of a number of multiple regression analyses to explain the variation of some of the CVD risk factors are shown in Table IV. All these analyses show the contribution of both the ethnicity of the child and the mother's level of education (reflecting the SES of the child) to the variation of the outcome variable. None of the interaction terms entertained in the initial models proved to be statistically significant.

Table I. Data for 5-year-old participants compared with data of non-participants in the Birth to Ten study

	Black ($N = 852$)	Coloured ($N = 70$)	Indian ($N = 19$)	White ($N = 23$)	Total 5-year-old participants ($N = 964$)	Total non-participants ($N = 3\,075$)
Mean gestational age (weeks) (SD)	37.9 (1.8)	39.0 (2.2)	38.1 (2.5)	39.0 (2.2)	38.0 (1.8)	38.1 (2.1)
Mean birth weight (SD))	3 066 (521)	2 992 (570)	2 908 (452)	3 183 (529)	3 054 (524)	3 060 (528)
Mean age of mother at child's birth	25.4 (6.2)	25.8 (6.3)	27.7 (4.7)	27.4 (4.9)	25.6* (6.2)	26.1* (5.9)
Mother lives with partner (%)	33.4	66.7	100	83.8	38.8*	57.7*
Mothers with ≥ 10 years' schooling (%)	66.9	54.3	92.9	100	67.5*	58.3*
Type of home (%)						
Shack†	9.5	3.13	0	0	8.6*	26.4*
House‡	90.5	96.9	100	100	91.4	73.6
Sample size for social class indicator (N)	428	31	14	12	485 §	1 505 §
Social class indicator (%)						
0 = No electricity in the house	2.8	6.9	0	0	2.9	10.4
1 = With electricity in the house (E)	74.9	51.7	7.1	0	69.5	58.5
2 = Electricity in the house and access to a car (E + C)	19.2	10.3	35.7	8.3	19.2	15.4
3 = Electricity in the house, access to a car and washing machine (E + C + W)	2.8	24.1	28.6	16.7	4.9	5.5
4 = Electricity in the house, access to a car, washing machine and medical aid fund (E + C + W + M)	0.23	6.9	28.6	75.0	3.5	10.2

* $P < 0.05$ when comparing the total number of 5-year-old participants with the non-participants.
† Includes a hostel and single room or garage.
‡ Includes a flat or cottage.
§ Distribution of social class indicators differs significantly between 5-year-old participants and non-participants ($P = < 0.001$).

Table II. Risk factor profile for cardiovascular diseases in 5-year-old children in the Birth to Ten study

	All	Black	Coloured	Indian	White
Blood pressure					
Mean systolic BP (mmHg) (SD)	107.0 (13)	108.0 (12)*†	105.0 (11)	100.0 (8)*	100.0 (11)†
Mean diastolic BP (mmHg) (SD)	62.0 (8)	63.0 (8)*	61.0 (9)	59.0 (8)	56.0 (8)*
% with BP ≥ 108/70 mmHg‡	45.9	48.4	37.9	10.5	17.4
% with high/normal BP ≥ 108/70 but < 115/75 mmHg‡	23.6	24.5	23.9	0	13
% with significant hypertension ≥ 115/75 but < 124/84 mmHg‡	11.6	12.5	7	10.5	0
% with severe hypertension ≥ 124/84 mmHg‡	10.7	11.4	7	0	4.4
Lipid profile					
Mean TC level mmol/l (SD)	3.8 (0.8)	3.7 (0.7)†	3.9 (1)*	4.1 (0.6)	4.4 (0.8)*†
Mean LDLC level mmol/l (SD)	2.2 (0.7)	2.1 (0.6)*†	2.4 (1.0)	2.6 (0.5)*	2.8 (0.8)†
Mean HDLC level mmol/l (SD)	1.1 (0.3)	1.1 (0.3)	1.1 (0.3)	1.1 (0.2)	1.1 (0.3)
Mean % HDLC/TC ratio (SD)	30.4 (8)	30.6 (8)*	28.9 (7)	27 (4.5)	25.8 (5.7)*
% with HDLC level ≤ 1.0 mmol/l§	43.8	43.9	52.1	15.8	47.8
% with LDLC level ≥ 2.8 mmol/l§	14.8	13.7	18.3	31.5	30.4
% with borderline increased LDLC ≥ 2.8 < 3.4 mmol/l§	10.7	10.4	9.9	26.3	13
% with high LDLC ≥ 3.4 mmol/l§	4.1	3.3	8.5	5.3	17.4
% with TC level ≥ 4.4 mmol/l§	19.6	18.3	23.9	21.1	52.2
% with borderline increased TC level ≥ 4.4 < 5.2 mmol/l§	15.6	15.1	15.5	15.8	34.8
% with high TC ≥ 5.2 mmol/l§	4	3.3	8.45	5.3	17.4
% with protective HDLC/TC ratio ≥ 20%§	85	86	73	89	78
Anthropometry					
Mean weight in kg (SD)¶	18.1 (2.3)	18.2 (2.3)	16.9 (2.1)	16.8 (1.9)	19.5 (2.9)
Mean height in cm (SD)II	108.0 (4.7)	108.0 (4.6)	106.0 (4.8)	109.0 (4.0)	112.5 (4.6)
Mean BMI (SD)**	15.5 (1.3)	15.6 (1.3)	14.9 (1.3)	14.2 (1.1)	15.4 (1.7)

*† $P < 0.05$ when comparing two variables marked with either * or †.
‡ Blood pressure cut-off points for children 3 - 5 years according to JNC V;[20] distribution of the blood pressure categories differs significantly between the four ethnic groups.
§ Lipid cut-off points according to the National Cholesterol Education Programme (NCEP),[21] distribution of the lipid categories differs significantly between the four ethnic groups.
¶ Mean weight for Indians and coloureds differs significantly for all other comparisons ($P = 0.0001$).
II Mean height for Indians, blacks and coloureds differs significantly from whites ($P = 0.0001$).
** Mean BMI of blacks, whites and coloureds differs significantly from Indians ($P = 0.0001$).

Table III. Exposure of the 5-year-old children to environmental tobacco smoke

	All ($N = 883$)	Black ($N = 779$)	Coloured ($N = 64$)	Indian ($N = 18$)	White ($N = 22$)
Caregiver does not smoke (%)*	91.8	95.9	54.7	72.7	72.7
Caregiver smokes (%)*	8.2	4.1	45.3	27.8	27.3
Partner lives at home (*N*)†	765	674	53	18	20
Partner does not smoke (%)‡	61.8	62.9	60.4	27.8	60
Partner smokes (%)‡	38.4	37.1	39.6	72.3	40
Nobody in home smokes (%)§	36	36.2	28.8	33.3	54.6
Only 1 member in home smokes (%)§	38.5	39.6	28.8	33.3	31.8
≥ 2 members in home smoke (%)§	25.7	34.4	42.4	33.4	13.6
Children who had experimented with cigarette smoking (%)	6.7	6.7	8	7.1	0

* Significant differences ($P = 0.001$) between the 3 categories of caretaker smoking behaviour across ethnic groups except Indians = whites.
† Denominator for variables marked‡.
‡ Significant differences ($P = 0.045$) between the 3 categories of partners' smoking behaviour across ethnic groups.
§ Significant differences ($P = 0.024$) between the 3 categories of numbers of smokers in the household across ethnic groups.

Only 9.9% and 7.8% of the variation in systolic and diastolic BP, respectively, were explained by the regression models. Systolic BP increases with the current height of the child, decreases with increased birth weight of the child, and is lower if the child is being breast-fed at 1 year. In addition, the ethnic group had a significant impact on the systolic BP. The child's current height, the mother's age at the child's birth and the child's ethnicity were the independent variables explaining the variation of diastolic BP in the children.

Even less of the variation in TC, LDLC and HDLC could be explained by the variables entered into the regression analyses,

Table IV. Multiple regression analyses of CVD risk factors at 5 years

Dependent variable	R^2	Independent variable	*P*-value
Systolic BP	9.9%	Height at 5 years	0.0001
		Birth weight (inverse)	0.0238
		Breast-feeding at 1 year (inverse)	0.0481
		Ethnic group	0.0096
		Mother's education	0.1308
Diastolic BP	7.8%	Height at 5 years	0.0001
		Mother's age at child's birth	0.0381
		Ethnic group	0.0001
		Mother's education	0.8505
Total cholesterol	4.2%	Caregivers smoking at 5 years	0.0002
		Ethnic group	0.0001
		Mother's education	0.2792
LDL cholesterol	5.0%	Caregiver smoking at 5 years	0.0073
		Ethnic group	0.0001
		Mother's education	0.7185
HDL cholesterol	2.6%	Mother's age at child's birth	0.0037
		Caregiver smoking at 5 years	0.0486
		Mother's education	0.0164
		Ethnic group	0.9846

found to be 4.2%, 5.0% and 2.6%, respectively. For TC and LDLC the independent explanatory variables were the smoking of cigarettes by the primary caregiver and the ethnicity of the child. The mother's level of education, as an indicator of SES, was not identified as an independent variable relating to the biological variable in any of the previous results discussed. For the HDLC level, the mother's age, her level of education and the primary caregiver's current smoking status explained the observed variation.

Discussion

The GBD has predicted that in the early part of the 21st century non-communicable diseases will be the major cause of death in developing countries.[5] This implies, owing to the long incubation period, that the risk profile pattern observed in the last decade of the 20th century should reflect populations of children who already have significant levels of risk for chronic diseases. The data presented here, the first available on the risk profile of preschool children for chronic diseases of lifestyle in South Africa, demonstrate substantial differences across ethnic groups, suggesting that children from various communities are at different stages of the epidemiological transition. Indian and white children demonstrate typical risk profiles of industrialised westernised groups, with relatively low rates of raised BP, but high rates of hyperlipidaemia. In contrast, the black children demonstrate the other extreme of the transition, with low rates of hyperlipidaemia and high rates of raised BP. Between these groups are the risk profiles of the coloured children, with significant levels of raised BP as well as relatively high rates of hyperlipidaemia (Table II). Furthermore, these coloured children are exposed to the highest rates of ETS and include the most children to have experimented with cigarette smoking by the age of 5 years. This results in the coloured children having the worst chronic diseases risk profile compared with the other groups described here. Overall, the black children have the most favourable chronic diseases risk profile.

Despite the clear patterns of CVD risk that have emerged from these data, the study has a number of shortcomings that need to be considered when assessing the overall value of the data. The small number of white and Indian children who were willing to participate in the study has probably provided a biased sample, and the data should therefore be viewed in the light of this situation. Furthermore, the fact that we could find only a small proportion of the children who were entered into the study at birth, and that the CVD group of children differed significantly from the non-participants who were entered into the study, will affect the generalisability of the data. As seen in Table I, the non-participants showed significant differences from the participants at both extremes of the socio-economic scale. In the lowest socio-economic group there were fewer participants than non-participants in the
5-year-old study. This could be due to the fact that the poorest people move about more than those who are better off. Again, among the more wealthy participants significantly fewer children participated in the 5-year follow-up study. This is further reflected in the low numbers of white and Indian children who participated in the study. The lack of participation in the highest SES groups may be explained by the fact that people with sufficient resources see little benefit in participating in public health studies, as they normally utilise the private health sector.

Notwithstanding the concern that this study sample may not be representative of 5-year-old South African children, the patterns reported here are similar to those described for older South African children.[13,14] The earliest report on the chronic disease risk profile in South Africa was that of Rossouw.[14] In 1985 she compared the chronic disease risk profile of black, coloured and white children aged about 11 years attending primary schools in the Western Cape in urban and rural settings. The urban black children's BP was found to be higher than that of the urban white and coloured children, while their lipid levels showed the opposite distribution between the groups. In addition, the rural children had a better risk profile than that of their urban counterparts, and this urban/rural difference was not evident in the other groups studied. This high urban BP level compared with the low level of the rural black children was found to be associated with a higher sodium intake in urban children compared with their rural black counterparts. Similarly, the unfavourable lipid profile in the white children occurred in association with the intake of a non-prudent diet compared with the prudent diet of the black children.[14]

Seftel *et al.*[13] described a similar ethnic distribution of chronic disease lipid risk profiles among scholars in South Africa. This study focused on the lipid profile of boys with 11 years' schooling, aged 17 - 19 years. The worst lipid profiles were found among Indian males, but these were similar to the levels of the white and high socio-economic coloured males. The lipid profiles of black males in the rural settings were found to be better than those for young black urban males.

The risk factor profile demonstrated in this group of 5-year-old children reflects the chronic disease pattern in South Africa, where the Indian and white communities have the highest levels of chronic disease mortality, the black community the lowest level, and the coloured community falls between these extremes.[22] That the coloured community's current mortality pattern does not reflect its adverse risk profile at present is probably due to the long incubation period of these chronic diseases. However, the risk factor profile suggests that this group will have markedly raised chronic disease patterns in the near future. This is already evident. Lung cancer rates among coloured women have increased significantly during the last few years following their increased smoking rates.[22]

The reason for the difference in BP levels and high rates of hypertension in the black children compared with other children in the study is uncertain. It is possible that postnatal environmental differences, genetic differences, or interactions between these factors may be responsible. Smith *et al.*[23] studied BP and its determinants in a subgroup ($N = 684$) of 1-year-old BTT children from Soweto. Multivariate analyses identified weight and upper arm circumference to be associated with the BP level at 1 year of age as well as the duration and volume of infant formulas. Salt added to feeds approached statistical significance.[23] A nutrition study conducted by MacKeown *et al.*[24] in the same group of BTT children when they were 5 years old, found that black children consumed higher levels of salt than the other groups of children. Increased salt-sensitivity in people of African origin has frequently been reported and has been postulated to play a role in the development of hypertension. Although this phenomenon has not been studied in detail in the black population in South Africa, these data allow for the hypothesis that there is an interaction between a possible salt sensitivity and the high salt intake that precipitates the high levels of hypertension in these black children. Rossouw's study[14] of the 11-year-old children in 1985 found that rural black children consumed significantly less salt than urban black children. The black community in the country is undergoing a marked degree of urbanisation (3.5% per annum); should this be associated with increased salt consumption in a salt-sensitive community, it will have significant implications for the level of hypertension and its complications in this group. In addition, it has been reported that bread, a staple food for many South Africans, contains much higher levels of sodium chloride in South Africa than elsewhere in the world (Ms N Vorster, SASKO (Suid-Afrikaanse Sentrale Koöperatiewe graanmatskappy), Paarl — personal communication). This highlights the need to study the issue of salt sensitivity in the black community in South Africa in order to determine whether it is appropriate to motivate salt-reducing dietary strategies in the country.

The level of unfavourable lipid profiles in the Indian and white children suggests that the high rates of cardiovascular mortality in these groups are unlikely to decrease significantly in the early part of the 21st century. These children, when adults, will experience the results of their lifelong dietary habits unless they can achieve significant changes in their dietary patterns.

The high level of exposure to ETS identified in these children is of concern, as it has been clearly documented that parents who smoke adversely affect the health of their offspring.[25] Babies who are exposed to ETS are more likely to be hospitalised for bronchitis and pneumonia in the first year of life. Chronic cough and phlegm are also more frequent among children exposed to ETS. Indeed, there is evidence that by enhancing the frequency or severity of respiratory diseases in children, ETS could contribute to the development of respiratory diseases in these individuals when they are adults.[26] Furthermore, it has also been shown that an adult example of smoking in the home leads to an increased risk that children will experiment and adopt a smoking habit later in life.

In order to assess the impact of both genetic and environmental influences on the emergence of risk factors, an attempt was made to consider socio-economic status and ethnicity simultaneously in the multiple regression analyses. In the results shown in Table IV, the socio-economic differences were measured using the mother's level of education. This has

proved to be a valuable proxy for a child's socio-economic status in communities where the income of the family is not determinable.[27] The results in this table show clearly that ethnicity contributes independently to the variation in systolic and diastolic BP, TC and LDLC levels, while the mother's level of education contributes independently to the level of HDLC and ethnicity does not.

The inverse relationship between systolic BP and birth weight supports the Barker hypothesis that low birth weight predisposes to the development of risk factors that will lead to higher rates of chronic diseases of lifestyle in adults.[28,29] When Levitt *et al.*[30] analysed these data exclusively for the black children participating in this study, a mean decline of 3.4 mmHg systolic BP resulted from every 1 000 g increase in birth weight. The finding of an inverse relationship between breast-feeding at 1 year and systolic BP in 5-year-old children is new and may well be due to either dietary differences in children's intake of other foods at 1 year, or to an inherent benefit of breast-feeding itself during the first year of life. The data published by Smith *et al.*[23] on formula feeding in these children, and the possible role of added salt as a determinant of BP at the age of 1 year, again suggests that breast-feeding plays a protective role against hypertension. An association between birth weight and the emergence of diabetes in the BTT children has been found by Crowther *et al.*[31] The influence of maternal smoking pattern on the variation of the lipid profiles is also an unknown association. In fact, the direct association between HDLC levels and the caregiver's smoking is opposite to what would be expected, as it has been described that exposure to tobacco smoke reduces the HDLC levels in adults.[32,33]

The data reported here suggest that urban 5-year-old South African children carry a higher level of risk for adult chronic diseases than was previously suspected. This finding is in line with the GBD predictions of a chronic disease epidemic for developing countries early in the 21st century. This should motivate the South African health services to ensure that appropriate interventions are put into place to prevent the predicted epidemic.

Improved legislation to provide comprehensive tobacco control for South Africa has been passed to protect South Africans against tobacco sale promotions. Once implemented, this will be the first step towards increasing the priority of chronic disease prevention, health promotion and appropriate care for chronic diseases and their risk factors on the South African health policy agenda.

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