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IMPACT OF URBANISATION ON SERUM LIPID PROFILES — THE THUSA SURVEY

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Objective. To examine the impact of urbanisation on lipid profiles of black South Africans, stratified for HIV status.

Design. Cross-sectional population-based survey.

Setting. North West province of South Africa.

Subjects. A representative sample of 1 854 apparently healthy volunteers aged ≥ 15 years, was recruited from 37 randomly selected sites throughout the province. Subjects were stratified into five urbanisation strata (S): S1 rural villages, S2 farms, S3 informal housing or 'squatter camps', S4 urban townships, and S5 surburban housing.

Outcome measures. Demographic, physical activity and dietary intake information was collected using validated and culture-sensitive questionnaires. Anthropometric measurements and lipid analyses were determined using standardised methodology.

Results. The results revealed significantly lower mean (95% confidence interval) total serum cholesterol (TC) levels in HIV-negative men in S1 - S4 compared with S5 (S1 3.91 (3.77 - 4.05) v. S5 4.79 (4.54 - 5.04) mmol/l). In HIV-negative women, TC levels were significantly lower in S1 - S3 than in S4 and S5 (S1 4.05 (3.94 - 4.17) v. S5 4.79 (4.59 - 5.00) mmol/l). The same trends were seen for serum low-density lipoprotein cholesterol (LDLC) and triglycerides and in HIV-positive subjects. Binary logistical analysis indicated that the main factor responsible for the increased TC levels seemed to be increased body mass index (BMI) due to decreased physical activity.

Conclusions. Serum lipid levels increased with urbanisation although they remained within levels recommended for

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other populations. This may, however, become an important health problem in future if preventive strategies are not implemented. Culturally sensitive physical activity programmes to decrease BMI, targeted at professional men and women, and women in urban townships, seem to constitute the most appropriate intervention.

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Urbanisation in developing countries, changing from a rural to an urban dwelling and often from a traditional to a more Westernised lifestyle, is associated with an increased risk of chronic diseases such as ischaemic heart disease (IHD).[1] As a result of political change there is currently a rapid process of urbanisation among black South Africans. In 1994, 48.8% of the total South African population was urbanised, compared with 53.7% in 1996. During the period 1993 - 1996, the percentage of urbanised black South Africans increased from 35.8% to 43.3%.[2] One would expect the incidence of IHD to have increased in the black population, but it is still virtually absent in this population group.[3] Results from two studies in 1990 on black South African populations showed a low prevalence of dyslipidaemia,[4,5] one of the major risk factors for IHD.[6] This may in part have been ascribed to a prudent diet.[7] The present situation regarding serum lipid profiles and risk of IHD among blacks is not known. Information is urgently needed for the design of appropriate intervention programmes to try to prevent an escalation of IHD in this population. Serum lipid levels are also decreased by HIV infection.[8,9] In South Africa the HIV/AIDS epidemic is prevalent mostly among the black population and it is estimated that 4 million people are at present infected with HIV, with a predicted increase to 6 million in less than 10 years.[10] The objective of this paper is to report on the impact of urbanisation on serum lipid profiles and the associated factors in South African men and women in North West province who participated in the THUSA (Transition in Health during Urbanisation of South Africans) survey, stratified for HIV status and level of urbanisation. THUSA, a cross-sectional, comparative, multidisciplinary study conducted from 1996 to 1998, was designed to measure the impact of urbanisation on the health risks of blacks in transition. *Thusa* means 'help' in the Setswana language.

METHODS

Subjects and study design

A representative sample of 1 854 apparently healthy volunteers in North West province, aged 15 years and older, participated in the survey. The design, sampling procedures and methods of the survey have been reported elsewhere.[11] Briefly, volunteers were recruited from 37 randomly selected sites in North West province. Pregnant and lactating women, subjects with any known diagnosed disease, subjects on chronic medication, those with oral temperatures > 37°C and inebriated subjects were excluded. Subjects were classified into five strata of urbanisation based on occupation and living (housing) circumstances, namely stratum 1 (S1) deep rural villages, stratum 2 (S2) farms, stratum 3 (S3) informal housing areas or 'squatter camps', stratum 4 (S4) urban townships or stratum 5 (S5) suburban housing. The following relevant information for this particular paper was obtained from each subject. Structured, standardised questionnaires were administered during individual interviews in the language of the subject's choice to yield demographic information, including type of housing, income, education, health history and smoking habits. Dietary information was obtained using a quantitative food frequency questionnaire, specially designed and validated against 7-day weight records and 24-hour urinary nitrogen excretion for this population,[12] and administered by trained fieldworkers. Nutrient intakes were analysed using a computer program based on the South African Food Composition Tables.[13] Anthropometric measurements (heights and weights) were made in duplicate, with subjects in their underclothes. These measurements were taken by postgraduate biokinetics students, and standardised by a level III internationally accredited anthropometrist (JHdR) using calibrated equipment. Body mass index (BMI) (kg/m^2) was calculated. Physical activity information was obtained using a quantitative questionnaire measuring physical activity index (PAI), based on the Baecke physical activity questionnaire,[14] specially adapted and validated for this population.[15] Adaptations from the Baecke questionnaire included changing the item on commuting to provide information on time, as well as pace of walking or cycling. Using a test-retest design to assess reliability, an intra-class correlation coefficient of 0.880 was obtained between PAI obtained at the first and second interview. The PAI was validated against a 24-hour activity record of a subsample of subjects and correlated positively with the calculated energy cost of activities ($r = 0.556$, $P = 0.009$).[15]

Blood samples and biochemical analyses

A venous blood sample was drawn by a registered nursing sister from the vena cephalica of each patient using a sterile butterfly infusion set and sterile syringes. Subjects had to be in a fasted state for the blood sampling, but some of the subjects had had a snack beforehand. Record was kept of subjects not in the fasted state. Serum was prepared in the field by centrifuging the blood samples within 30 minutes, using a Universal 16R™ Hettich centrifuge with cooling facilities. Aliquots of serum were stored in the field at –18°C and after 2 - 4 days in the laboratory at –84°C. Serum lipids were

determined using the DAX system (discrete analyser, Technicon DAX 48; Miles Inc., Diagnostic Division, Tarrytown, NY, USA) in the laboratory of the Chemical Pathology Department, University of Pretoria. HIV status was determined anonymously using an enzyme-immunological method (Enzymum-Test, anti-HIV 1 and 2 and subtype ø, Boehringer Mannheim, Germany, cat. no. 1557319). Appropriate internal and external standards were used for all analyses.

Ethical approval and informed consent

The North West Department of Health and the Ethics Committee of the Potchefstroom University for Christian Higher Education (PUCHE) (approval no. HHK 4M5-95) gave permission for the survey. The subjects gave written informed consent to participate in the survey. Ethical approval to test anonymously for HIV infection was obtained from the same ethics committee.

Blinding

The study was double blinded. All subjects and researchers, except those responsible for administration of the demographic questionnaire (used to collect information on housing circumstances and occupation), were blinded to the classification of subjects into strata.

Statistical analysis

The SPSS programme was used. Data that were not normally distributed were logarithmically transformed. The data are presented as means (95% confidence intervals (CIs) or standard deviations (SDs)), stratified for gender, HIV status and stratum. The effect of stratum on serum lipid variables in subjects stratified for gender and HIV status were analysed using multivariate analyses while controlling for age and fasting state in the case of triglycerides (TGs). Known predictors of cholesterol levels were identified and their association with total serum cholesterol (TC) was examined using stepwise regression analyses. These factors included: *(i)* dietary factors, namely total energy (E), %E fat, %E saturated fatty acids, %E polyunsaturated fatty acids, %E monounsaturated fatty acids, polyunsaturated/saturated ratio, trans fatty acids, %E total carbohydrate, total dietary fibre and alcohol intake; *(ii)* socio-economic factors, namely income and education; *(iii)* PAI; *(iv)* BMI; and *(v)* smoking habit. The most important factors that were significantly associated with TC were expressed as means (95% CI) or percentages per quartile of TC. Odds ratios (ORs) were calculated per stratum for subjects to have a cholesterol level greater than the median (3.84 mmol/l for men and 4.13 mmol/l for women) adjusted for age, BMI and HIV status. The median TC level had to be used and not the IHD risk cut-off point of 5 mmol/l[16] because of the low cholesterol levels in this population group.

RESULTS

Table I summarises some of the study population characteristics. More detailed information regarding the physical, physiological and mental characteristics of the population are reported elsewhere.[11]

Table I. Mean (SD) or percentages of population characteristics

Characteristics	Men	Women
Number of subjects	710	989
Age (yrs)*	37.37 (15.41)	37.80 (14.19)
Body mass index (kg/m²)	21.16 (4.09)	26.92 (6.82)
Education (%)		
No education	24.4	19
< 8 years of schooling	27.6	34.8
8 - 10 years (± trade)	19.8	21.6
11 - 12 years (± trade)	20.3	17.4
11 - 12 years (+ tertiary education)	7.9	7.1
Smokers (%)	56.5	17.1
Snuff takers (%)	2.8	21.8
Alcohol intake (g/day)	18.45 (40.13)	2.87 (10.63)
Physical activity index (%)		
Inactive	23.8	35.7
Moderately active	24.9	31.3
Active	51.3	33.0
Energy distribution of diet		
% from total fat	24.87 (7.55)	25.84 (7.45)
% from total carbohydrate	64.95 (9.86)	63.93 (9.92)
% from protein	11.96 (2.10)	11.87 (2.21)
Fibre intake (g/day)	18.29 (9.34)	16.37 (7.59)

* Range 15 - 65 years.
SD = standard deviation.

The effect of urbanisation on lipid profiles, stratified for gender and HIV status, is illustrated in Table II. Multivariate analysis, controlled for age, showed that in HIV-negative men, TC and low-density lipoprotein cholesterol (LDLC) levels in S1 - S4 did not differ, but were all significantly lower than in S5. In HIV-negative women, TC and LDLC levels did not differ in S1 - S3 but were significantly lower than in S4 and S5. Levels in S4 were significantly lower than in S5. The same trends were seen in HIV-positive subjects. The mean TC levels of HIV-positive subjects were significantly lower than in HIV-negative subjects.

Men in S5 had a 3.4-times higher risk (OR) (Table III) of having a cholesterol level greater than the median compared with men in S1. The risk for women in S4 and S5 of a cholesterol level greater than the median was 1.6 and 2.7 times, respectively, higher than for women in S1.

Urbanisation had no effect on high-density lipoprotein cholesterol (HDLC) levels in men. HIV-negative women in S5 had higher HDLC levels than women in S1 - S4, although this

Table II. Serum lipid profiles of black men and women stratified for HIV status and level of urbanisation (means (95% CI))

Variable	HIV status	Level of urbanisation: strata 1 - 5 S1	S2	S3	S4	S5	Total
Men							
Number of subjects	+	14	8	14	50	4	93
Number of subjects	–	176	104	115	171	54	638
TC (mmol/l)	+	3.57	3.65	3.44[a]	3.85	4.44[a]	3.76*
		3.12 - 4.02	3.05 - 4.24	2.98 - 3.89	3.61 - 4.08	3.59 - 5.29	3.56 - 3.95
	–	3.91[a]	4.07[b]	3.88[c]	4.00[d]	4.79[abcd]	4.03*
	3.77 - 4.05	3.89 - 4.24	3.71 - 4.05	3.86 - 4.14	4.54 - 5.04	3.96 - 4.10	
HDLC (mmol/l)	+	1.11	1.19	0.98	1.17	1.10	1.14
		0.91 - 1.31	0.93 - 1.46	0.78 - 1.18	1.06 - 1.27	0.73 - 1.48	1.05 - 1.22
	–	1.22	1.18	1.23	1.22	1.23	1.21
		1.16 - 1.28	1.10 - 1.25	1.15 - 1.30	1.16 - 1.28	1.12 - 1.34	1.18 - 1.25
LDLC (mmol/l)	+	2.17	2.10	2.11	2.31	2.88	2.27
		1.74 - 2.60	1.54 - 2.67	1.68 - 2.54	2.08 - 2.53	2.08 - 3.69	2.07 - 2.47
	–	2.33[a]	2.45[b]	2.22[c]	2.37[d]	3.10[abcd]	2.41
		2.19 - 2.47	2.27 - 2.63	2.05 - 2.39	2.23 - 2.51	2.85 - 3.35	2.33 - 2.48
TG (mmol/l)	+	0.81	1.15	0.83[ab]	1.08[a]	-	1.01
		0.53 - 1.09	0.58 - 1.71	0.62 - 1.03	0.95 - 1.21	-	0.82 - 1.20
	–	1.02	1.02	1.10	1.19	1.38	1.10
		0.85 - 1.18	0.75 - 1.28	0.90 - 1.26	1.04 - 1.33	0.88 - 1.88	1.02 - 1.17
Women							
Number of subjects	+	27	13	29	37	9	116
Number of subjects	–	258	130	142	245	87	873
TC (mmol/l)	+	3.48[abc]	4.07[a]	3.73[de]	4.21[bd]	4.39[ce]	3.98*
		3.16 - 3.81	3.61 - 4.53	3.42 - 4.03	3.94 - 4.48	3.83 - 4.95	3.80 - 4.15
	–	4.05[ab]	4.12[cd]	4.21[ef]	4.47[aceg]	4.79[bdfg]	4.28*
		3.94 - 4.17	3.95 - 4.29	4.05 - 4.37	4.35 - 4.60	4.59 - 5.00	4.21 - 4.34
HDLC (mmol/l)	+	1.01	0.98	0.99	1.07	1.10	1.04*
		0.89 - 1.13	0.81 - 1.15	0.88 - 1.10	0.97 - 1.17	0.89 - 1.30	0.98 - 1.09
	–	1.18	1.17	1.15[a]	1.15[b]	1.24[ab]	1.17*
		1.14 - 1.21	1.12 - 1.23	1.10 - 1.20	1.11 - 1.19	1.17 - 1.30	1.15 - 1.19
LDLC (mmol/l)	+	2.13[abc]	2.70[a]	2.33[d]	2.77[bd]	2.84[c]	2.56
		1.81 - 2.45	2.24 - 3.16	2.02 - 2.63	2.50 - 3.04	2.28 - 3.40	2.39 - 2.73
	–	2.50[ab]	2.54[cd]	2.61[ef]	2.89[ace]	3.05[bdf]	2.68
	2.38 - 2.61	2.38 - 2.71	2.46 - 2.77	2.77 - 3.01	2.85 - 3.25	2.62 - 2.75	
TG (mmol/l)	+	0.87	1.02	1.09	1.03	-	1.06
		0.70 - 1.03	0.79 - 1.24	0.92 - 1.26	0.86 - 1.20	-	0.92 - 1.20
	–	0.98[a]	1.09	1.04	1.17[a]	0.87	1.06
		0.87 - 1.10	0.94 - 1.24	0.89 - 1.19	1.07 - 1.28	0.35 - 1.39	1.01 - 1.12

a, b, c, d, e, f, g: Means with the same symbol differ significantly within strata (multivariate analysis, controlling for age (TG also controlled for fasting state), $P < 0.05$).
* Means differ significantly between HIV-positive and -negative subjects (multivariate analysis, controlling for age and stratum (TG also controlled for fasting state), $P \leq 0.05$).
S1 = rural, S2 = farms, S3 = informal housing areas (squatter camps), S4 = urban (townships), S5 = suburban housing, TC = total cholesterol, HDLC = high-density lipoprotein cholesterol; LDLC = low-density lipoprotein cholesterol, TG = triglycerides.

was only significant for S4. HIV-negative women had significantly higher HDLC levels than HIV-positive women.

Multivariate analysis, controlling for age and fasting state showed no effect of urbanisation on TG levels in men. In women, TG levels of urban dwellers (S4) were significantly higher than for rural subjects (S1). TG levels did not differ between HIV-negative and -positive subjects.

Irrespective of stratum, the mean TC, LDLC and TG levels were within the normal levels of < 5.0 mmol/l, < 3.0 mmol/l and < 1.7 mmol/l, respectively, that are recommended for South Africans.[16] Although the mean HDLC levels were below the recommendation of > 1.2 mmol/l,[16] the levels were still relatively high, especially when expressed as a percentage of TC.

Stepwise regression analysis showed significant correlations between TC and PAI, BMI, fibre intake (only in men) and education (only in women). Testing using a binary logistical

Table III. Risk estimations (odds ratios) per stratum with regard to subjects having cholesterol levels greater than the median

	Men			Women		
Stratum	Odds ratio	95% CI	*P*	Odds ratio	95% CI	*P*
S1	-	-	0.000	-	-	0.000
S2	1.44	0.87 - 2.39	0.159	1.25	0.81 - 1.93	0.323
S3	0.767	0.49 - 1.20	0.241	1.16	0.78 - 1.73	0.462
S4	1.001	0.70 - 1.43	0.997	1.62	1.18 - 2.24	0.003
S5	3.442	1.79 - 6.61	000	2.67	1.67 - 4.26	0000

S1 = rural, S2 = farms, S3 = informal housing areas (squatter camps), S4 = urban (townships), S5 = suburban housing; CI = confidence interval, *P* = significance. Median cholesterol levels: men = 3.84 mmol/l, women = 4.13 mmol/l. (Binary logistical regression analysis adjusting for age, body mass index and HIV status. The subjects in S1 were regarded as the reference group.)

Table IV. Means (95% confidence intervals) of body mass index and % men and women in physical activity categories stratified for quartile of total serum cholesterol

	Quartile of total serum cholesterol			
Gender and variable	Q1	Q2	Q3	Q4
Men				
Total cholesterol	2.86	3.57	4.14	5.37
(mmol/l)	2.82 - 2.91	3.54 - 3.59	4.11 - 4.18	5.27 - 5.47
PAI (% of total)				
Inactive	5.1	5.6	4.8	8.2
Moderately active	5.6	6.3	6.8	6.5
Active	18.0	15.7	9.7	7.3
BMI (kg/m²)	19.8	20.3	21.1	22.9
	19.5 - 20.2	19.9 - 20.8	20.5 - 21.6	22.2 - 23.5
Women				
Total cholesterol	3.00	3.81	4.48	5.70
(mmol/l)	2.97 - 3.05	3.78 - 3.83	4.45 - 4.50	5.60 - 5.80
PAI (% of total)				
Inactive	7.1	8.1	9.2	11.2
Moderately active	7.7	8.6	8.4	6.1
Active	12.3	10.4	7.5	3.1
BMI (kg/m²)	25.1	26.2	27.1	29.0
	24.3 - 25.9	25.3 - 27.0	26.4 - 27.9	28.1 - 30.0

PAI = physical activity index.

regression model to predict the risk for increased TC levels with urbanisation indicated that BMI was a highly significant (P = 000) independent predictor of increased TC. PAI and education, however, were indirect predictors for increased TC but directly linked to BMI and stratum, respectively. Fibre intake, in this model, was not an independent predictor for increased TC risk with urbanisation. Other known predictors of TC, namely dietary factors such as E, %E fat, %E saturated fatty acids, %E polyunsaturated fatty acids, %E monounsaturated fatty acids, P/S ratio, trans fatty acids, %E total carbohydrate, total dietary fibre, alcohol intake, income and smoking were not significantly associated with TC (data not shown). Table IV illustrates the increased BMI and decreased number of physically active subjects with increasing quartiles of TC in men and women.

Discussion

The major observation of this study was that serum lipid levels increased with urbanisation in both men and women. The main factor responsible for these increases seems to be increased BMI, probably due to decreased physical activity. The lipid levels in all the strata were, however, within the normal recommended levels.

Van Rooyen *et al.*[17] demonstrated that blood pressure also increased with urbanisation in these subjects. In their investigation blood pressure was also positively correlated with BMI.

Very few studies have examined the effect of urbanisation on lipid levels and only two studies, both in 1990, have been conducted on blacks. Mollentze *et al.*[4] compared a rural with an urban group of blacks in the Free State. This study showed that the TC levels were very comparable between the two groups although there was a tendency towards higher values in the urban group. According to the authors the rural group could already have been in an advanced stage of urbanisation. The study by Steyn *et al.*[18] on the black population in the Cape Peninsula, showed that the degree of urbanisation of subjects living in an urban setting had no effect on TC concentrations. Four other studies done in Puerto Rico,[19] Vanuatu, a Melanesian island country in the south-west Pacific,[20] India[21] and Saudi Arabia[22] showed higher cholesterol levels in urban compared with rural groups.

In the present study the professional men and women as well as women in urban townships were most at risk of increasing TC, LDLC and TG levels. The professional women had increased HDLC levels.

Results from this study showed that the main factor probably responsible for the increasing TC levels with urbanisation was BMI, probably due to decreased physical activity. Taylor *et al.*[20] and García-Palmieri *et al.*[19] also reported lower physical activity and increased weight or prevalence of obesity in urban compared with rural groups. Metabolic studies have documented that obesity results in an increased rate of endogenous cholesterol synthesis and increased very low-density lipoprotein (VLDL) apoprotein B and triglyceride production rates.[23]

The diets in these subjects changed during urbanisation from a very low-fat (approximately 23%E as fat) traditional diet with a small variety of additions (e.g. tomato and onion stew) to maize porridge (the staple eaten at all meals) in S1 and S2, to a more Western type of diet. But the urban dwellers and professionals followed a more adequate diet with a higher intake of fibre and micronutrients. This diet was relatively prudent with ≤ 30% of its energy as fat (data reported

elsewhere).[24] This may explain the lack of association between TC and dietary fat intake. Vorster *et al.*[25] have argued that during the nutrition transition of blacks raised in adverse environments, their risk factors for chronic disease increase when they are exposed in adulthood to affluent diets. The THUSA subjects had mean cholesterol levels still below the generally accepted South African cut-off point,[16] but the upper urban strata had significantly higher levels than those living in more rural areas. These phenomena may reflect the phase or time schedule of the urbanisation and dietary transition processes in South Africa at the moment. Micronutrient and fibre quality of the diet actually improved with urbanisation, while the total energy distribution was within prudent recommendations (fat ≤ 30%E, carbohydrate ≥ 64%E). It should be noted that the mean BMI of the women was in the overweight range (> 26 kg/m^2) despite the low fat intake. It is of concern that despite the prudent diet urban subjects showed significantly increased TC levels. It can be expected that with further transition in diet because of urbanisation and economic development, South Africa may well be on the road towards an IHD epidemic in the black population.

The lower TC and HDLC levels observed in the HIV-positive compared with HIV-negative subjects are in accordance with the literature.[8,9] Possible mechanisms that may explain the hypocholesterolaemia include fat malabsorption, which is commonly found in patients with HIV infection.[26] Increased release of cytokines such as tumour necrosis factor (TNF)-α, as part of the immune response to the virus infection, has been negatively associated with cholesterol.[8] Ly *et al.*[27] have shown reduced plasma lecithin/cholesterol acyltransferase (LCAT) activity and hepatic LCAT mRNA levels in Syrian hamsters after treatment with TNF and endotoxin (mimics infection). LCAT is responsible for the esterification of free cholesterol during the formation of HDL_2.[28] Decreased LCAT levels may therefore result in decreased HDLC levels. The TG levels did not differ between HIV-negative and -positive subjects. Increased TG levels have been reported in HIV-positive subjects[8,9] especially at a late stage of the disease and in cases of concurrent infection.[8] The participants from the current study were asymptomatic and apparently healthy. The majority of HIV-infected subjects were probably at an early stage of the infection.

In conclusion, lipid levels increased with urbanisation in both men and women. The lipid levels were, however, within normal recommended levels in both HIV-negative and -positive groups. Murray and Lopez[29] predict that in 2020, IHD will be one of the leading causes of death in developed and developing countries. The progression to IHD in this population may be slower because of current low lipid levels, but may become an important health problem in the future with continued urbanisation. Strategies will be necessary for the prevention of increased lipid levels with urbanisation. Culturally sensitive programmes should be designed to prevent decreases in physical activity and increases in BMI with urbanisation, targeted at professional men and women and women in urban townships.

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