The association between dyslipidemia and anthropometric indicators in black and white adolescents residing in Tlokwe Municipality, North-West Province, South Africa: the PAHL study

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Abstract

Background: The dyslipidemia associated with excess weight is a risk for cardiovascular disease. Worldwide and in South Africa adolescent obesity has been reported.

Objectives: To determine the association between dyslipidemia and anthropometric indices in black and white adolescents.

Methods: The study involved 129 black and 69 white adolescents aged 12 to 16 years. Data collected included height, weight, waist circumference (WC) and skinfolds, blood pressure and blood for glucose, insulin, total cholesterol (TC), low density lipoprotein (LDL), high density lipoprotein (HDL), triglycerides (Trig) and C - reactive protein (CRP).

Results: WC correlated negatively with HDL in both blacks ($p=0.042$) and whites ($p=0.008$) and in whites it correlated positively with LDL ($p=0.006$); TC/HDL ($p=<0.001$) and LDL/HDL ratio ($p<0.0001$). WC/Hgt correlated negatively with HDL ($p=0.028$) and positively with LDL/HDL ($p=0.026$ and $p<0.0001$) in both races. In whites positive correlations were between WC/Hgt and TC ($p=0.049$); LDL ($p=0.003$) and TC/HDL ($p<0.0001$). BAZ correlated positively with TC/HDL ratio ($p=0.004$) and LDL/HDL ratio ($p=0.002$). The most common abnormalities were HDL and LDL.

Conclusion: Whites exhibited more associations between dyslipidemia and anthropometric indicators as compared to Blacks, suggesting that there might be differences in the lipid metabolism or even susceptibility to risk factors in adolescents.

Key words: dyslipidemia, anthropometry, adolescents

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Introduction

There are several risk factors for coronary heart diseases, which can act independently or together. Among the most common are arterial hypertension, smoking, a sedentary lifestyle, diabetes, obesity, dyslipidemias, and a positive familial history of cardiovascular disease (CVD). The precocity of these factors signals the need to develop prevention and intervention strategies in pediatric populations.

Atherosclerosis coronary heart disease (CHD) has multifactorial causes. Studies have established that dyslipidemia plays an important role in its development and progression. Even though clinical CHD only occurs in later life it is known that atherosclerosis may already present itself in young adults[1]. It has also been observed that conditions related to altered lipid levels such as unhealthy dietary habits, tobacco smoking and physical inactivity are acquired during childhood and adolescence[2]. Moreover, obesity, dyslipidemia and hypertension in adolescence have been reported to track into adulthood[3]. Other studies have shown that differences in lipid levels among cultures and ethnic groups appear early in childhood[3].

Waist circumference (WC) and waist-to-height ratio (WHtR) during childhood are predictors of the development of risk factors for CVD. Visceral adiposity has a strong impact on CVD due to its association with dyslipidemias, arterial hypertension, insulin resistance and diabetes. High plasma triglycerides (TG) and low concentrations of high-density lipoprotein cholesterol

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(HDL- C) are among the alterations observed in the lipid profile that are primarily related to central fat distribution[4].

Childhood obesity has been on the increase in the past decades[5] and furthermore, it has been shown to be a predictor of increased mortality owing primarily to an increased risk of CVD[6]. In addition, the prevalence of individuals with normal body weight who display one or more obesity related morbidity such as type 2 diabetes and high blood pressure is increasing[7, 8]. There is substantial evidence that the association between obesity and CVD is due to adverse CVD risk factor profile that is seen in obese adults. These include type 2 diabetes, hypertension and dyslipidemia[9, 10]. To date it is known that all these are emerging in children and adolescents [11, 12].

Studies have shown that detection of altered lipid levels in adolescents especially raised serum levels of total cholesterol (TC) and LDL-C accompanied by low HDL-C can be useful in initiating measures for the prevention of atherosclerotic diseases and reduction of mortality rates[11, 12, 13]. The metabolic and physiological changes in the lipid profile of adolescents were found to be more pronounced in males than females due to differences in hormonal changes accompanying puberty[14].

The dyslipidemia associated with excess weight is a risk for cardiovascular disease. In South African adolescents obesity has been reported[15] and as such the aim of this study was to determine the association between dyslipidemia and anthropometric indices in black and white adolescents.

Methods

Study area

This study was conducted in Tlokwe Municipality (previously known as Potchefstroom Municipality) of the Dr Kenneth Kaunda District Municipality in the North West Province, South Africa. Tlokwe Municipality is located between 26° 43' 0" South and 27° 6' 0" East and longitudes 27, 1000 (276'0.000"E). The municipality encompasses several neighboring settlements with a population of 128,357 in a density of 48 km^2, according to the 2007 community survey. The area is primarily inhabited by Black Africans (˜70%), 27.0% White Africans, 3.0% Colored and 0.4% Asians (Stats SA; 2007). The major languages spoken in the area are Setswana, Afrikaans and English. The seat of the local municipality is Potchefstroom.

Study sample

Data on a total of 198 adolescents (129 Blacks and 69 Whites) from six schools out of the eight schools which were purposefully recruited within the Tlokwe municipality with four from Ikageng Township (that mainly consists of people with low socio-economic background) and two in Potchefstroom town (that mainly consists of people with high socio-economic background) participated in the study. This study is part of a five year observational multidisciplinary longitudinal study on Physical Activity and Health Longitudinal Study (PAHLS) that started in 2010. The study conveniently selected grade 8 pupils for baseline so as to make the five-year follow-up feasible; additionally, given the fact that schools are good grounding to conduct research studies which are longitudinal in nature for its logistics. The group of pupils studied may not be considered to be representative of the adolescents' population of either Tlokwe municipality or South Africa in general. Its goal was to describe the development of physical activity and determinants of health risk factors in adolescents attending high schools within Tlokwe municipality areas of the North West Province of South Africa since such information in this region is lacking in the literature, as such information may be of grateful in addressing the abnormalities of health risk factors.

Anthropometric measurements

Anthropometric measurements of height, weight and skinfolds were measured by Level 2 Criteria anthropometrists according to the standard procedures described by the International Society for the advancement of Kinanthropometry: ISAK[16]. Height was measured by the use of stadiometer to the nearest 0.1 centimeters (cm) with participants in a bare feet standing upright position with the head in the Frankfort plane. Weight was measured to the nearest 0.1 kilogram (kg) with an electronic scale with the subject wearing minimal clothing. The triceps and subscapular skinfolds were measured to the nearest 0.2 mm with a Harpenden (British Indicators, UK) skinfold caliper and the average of two measurements were used. The waist circumference (WC) was measured, to the nearest 0.1 cm with a 7-mm-wide flexible steel tape (Lufkin, Cooper Tools, Apex, NC), at the midpoint between the lower rib margin and the iliac crest. The hips were measured to the nearest 0.1 cm at maximum extension of the buttocks. Waist-to-hip ratio (WHR) was calculated from waist and hip circumferences. Body mass index (BMI) as a measure of body composition was calculated as body mass/ stature^2 (kg/m^2). Subsequently, height-for age z-score (HAZ), weight-for-height z-score (WHZ), weight-for-age z- score (WAZ) and as well as BMI z-score (BAZ) were classified according to WHO Multicentre study references[17].

Blood analysis

Participants were requested to fast for 12 hours before blood samples were taken in the morning. Professional nurses took venous blood from the cephalic vein for the preparation of serum. The tubes were kept for approximately 30 min to coagulate and then centrifuged for 15 min at 2000g for the serum. The serum was divided into aliquots and stored at -84°C until analysed at an accredited laboratory (Ampath Laboratories, Pretoria, South Africa). Serum was used for the analyses of total cholesterol (TC), low density lipoproteins (LDL), high density lipoproteins (HDL), triglycerides (Trig) and C-reactive proteins CRP. Serum TC, LDL, HDL, Trig, was measured with a Vitros DT60 II Chemistry Analyser (Ortho-Clinical Diagnostics, Rochester, NY, USA) with Vitros reagents and controls.Serum high-sensitivity C-reactive protein was determined by rate turbidimetry with a High Sensitivity C-Reactive Protein kit (CRPH, IMMAGE, Immunochemistry Systems, Fullerton, (CA, USA) with control serum as an external standard.

Diagnosis of abnormal lipid parameters

Abnormal lipid parameters were defined by using the following criteria: HDL-C: <1.2 mmol/LLDL: >2.5 mmol/LTC: >2.3 mmol/L LDL/HDL ratio: <2.20 TC/HDL ratio: <3.5

Ethical considerations

This study was approved by the ethics committee of North-West University (Potchefstroom campus) and approved by both the North-West Province Department of Health and Social Welfare Research committee and Department of Education. Written informed consent was obtained from the adolescents' parents/ guardians and their verbal assent was obtained.

Statistical analysis

WHO Anthroplus software was used to calculate the adolescents' BAZ-scores. Data was analyzed using SPSS (version 19). Since most of the data were not normally distributed non-parametric tests were computed. Descriptive statistics were computed and data are presented as medians and interquartile ranges. Mann-Whitney U test was used to test for differences between two groups and furthermore differences were computed after adjusting for gender. X2-test was used to compare differences between categorical data and Spearmen's correlation coefficients were used to assess the association between anthropometric indices and measures of iron status. Partial correlations after adjusting for gender were also computed. Linear regression analyses were done to determine anthropometric predictors of lipid parameters. A p-value of <0.05 was considered statistically significant.

Results

Differences were observed in weight, height, BMI, BAZ and WC with Black adolescents recording lower values in these variables even after adjusting for gender differences. With regard to SST ratio it only showed significant differences after adjusting for gender. Biochemical variables that showed differences between the two races before and after adjusting for gender were total cholesterol and LDL with blacks showing significantly lower values than Whites, even though the significant levels dropped after adjustment for gender (Table 1).

Triglyceride and glucose levels were lower in blacks before adjusting for gender as were TC/HDL ratio and LDL/TC ratio with lower values recorded in Black adolescents but these differences were not there after adjusting for gender differences (Table 1).

Table 1: Anthropometric and Biochemical variables of Black and White Adolescents residing in Tlokwe municipality (medians and IQ ranges)

Variable	Total (198)	Africans (n=129)	Whites (n=69)	p-Value	p-Value*
Age (years)	14.89 (0.82)	14.88 (0.94)	14.90 (0.75)	0.581	0.947
Weight (kg)	54.0 (14.0)	51.0 (14)	61.0 (16.0)	<0.0001	<0.0001
Height (m)	1.61 (0.13)	1.58 (11.0)	1.67 (11.0)	<0.0001	<0.0001
BMI (kg/m2)	20.28 (4.75)	19.82 (4.49)	21.39 4.85)	0.004	<0.0001
BAZ	0.23 (1.62)	0.10 (1.50)	0.53 (1.35)	0.010	0.014
TSF + SSF	24.10 (15.45)	23.0 (15.30)	26.0 (16.90)	0.451	0.053
SSF/TSF ratio	0.82 (0.32)	0.82 (0.34)	0.81 (0.29)	0.499	0.061
SST ratio	0.07 (0.05)	0.07 (0.05)	0.07 (0.05)	0.408	0.028
WC (cm)	67.5 (8.48)	65.50 (7.90)	70.35 (8.60)	<0.0001	<0.0001
HC (cm)	89.40 (13.2)	86.85 (12.50)	92.65 (12.30)	<0.0001	0.482
WHR	0.75 (0.08)	0.75 (0.08)	0.77 (0.08)	0.184	0.537
WC/Hgt ratio	0.42 (0.05)	0.41 (0.05)	0.42 (0.05)	0.420	0.320
CRP (mg/L)	2.0 (1.0)	2.0 (1.0)	2.0 (1.0)	0.289	0.495
TC (mmol/L)	4.0 (1.2)	3.80 (1.10)	4.30 (1.30)	<0.0001	0.008
Trig (mmol/L)	0.7 (0.4)	0.60 (0.30)	0.80 (0.60)	<0.0001	0.206
HDL (mmol/L)	1.30 (0.40)	1.30 (0.40)	1.30 (0.30)	0.273	0.207
LDL (mmol/L)	2.40 (1.0)	2.20 (0.80)	2.80 (0.70)	<0.0001	0.002
TC/HDL ratio	3.17 (1.04)	3.06 (0.93)	3.46 (1.0)	<0.0001	0.306
LDL/HDL ratio	1.92 (0.92)	1.75 (0.79)	2.22 (0.89)	<0.0001	0.088

*Adjusted for gender

BAZ-BMI-for age z-score; HAZ-height-for-age z-score, TSF-triceps skin fold, SSF-subscapular skin fold, SST-subscapular- to- triceps, WC-waist circumference, HC-hip circumference, WC/Hgt- waist circumference-to-height, CRP-C-reactive protein, TC- Total cholesterol, Trig-triglycerides, HDL- High density lipoprotein, LDL-low density lipoprotein

Having looked at the measured lipid parameters it was found that the most common abnormalities were HDL 92 (46.5%) [57 (44.2%) Africans and 35 (50.7%) Whites (X2= 0.847; p= 0.245)]; LDL 83 (41.9%) (24 (31%) Africans and 43 (62.3%) Whites (X2= 4.732; p= 0.025)]. TC abnormalities were only observed in Africans 126 (97.5%). With regard to ratios the most common abnormalities were LDL/HDL ratio 135 (68.2%) [101 (78.3%) Blacks and 34 (49.3%) Whites (X2= 2.660; p= 0.080)] and TC/HDL ratio 133 (67.2%) [98 (76%) Blacks and 35 (50.7%) Whites(X2= 1.491; p=0.160)].

Tables 2 and 3 show crude correlation coefficients in black and white adolescents respectively between measured lipid parameters and anthropometric indices and after adjusting for gender differences. In Black adolescents BMI showed a negative correlation with TC and HDL only after adjusting for gender while in Whites there was a positive correlation with LDL (r=0.293; p=0.015), TC/HDL ratio (r=0.412; p<0.0001) and LDL/HDL ratio (r=0.431; p<0.0001) before adjustment and this was even stronger with LDL (r=0.426; r=0.011) and LDL/ HDL ratio (r=0.601; p<0.0001) after adjusting for gender. BAZ correlated positively with all measured lipid parameters in both races except with HDL with which it was negatively correlated but after adjusting for gender it correlated negatively with TC, LDL and HDL in Black adolescents and only positively with LDL/HDL ratio in Whites.

Waist circumference-to-height ratio showed positive correlations with all lipid parameters except with HDL which it was negatively correlated with in both races. After adjusting for gender differences in Black adolescents the correlations were still maintained while in Whites it remained with LDL, HDL and LDL/HDL ratio. WHR in Blacks was positively correlated with Trig (r=0.250; p=0.004); TC/HDL ratio (r=0.283; p=0.001) and LDL/HDL ratio (r=0.271; p=0.002) and negatively with HDL (r=-0.399; p<0.0001) after adjusting for gender the remaining associations were with TC/HDL ratio (r=0.333; p=0.025) and HDL (r=-0.353; p=0.017). WC was negatively associated with HDL in black adolescents (r=0.179; p=0.042) and this was even stronger after adjusting for gender differences (r=-0.442; p=0.002), furthermore, after taking the gender differences into consideration it correlated positively with LDL/HDL ratio. In Whites on the contrary WC was positively correlated with LDL (r=0.483; p=0.003), TC/HDL ratio (r=0.442; p<0.0001) and LDL/ HDL ratio (r=478; p<0.0001) and negatively with HDL (-0.316; p=0.008). After adjusting for gender differences all the correlations remained except with HDL and further WC showed a positive correlation with TC (r=0.360; p=0.034). Skinfolds indices showed varied associations with the lipid parameters in both races (Tables 2 and 3).

Table 2: Spearman's correlation coefficients in Black adolescents and adjusted for gender (n= 129)

		BMI	*BMI	WC	*WC	WHR	*WHR	TSF + SSF	*TSF + SSF	SSF/ TSF	*SSF/ TSF	SST ratio	*SST ratio	BAZ	*BAZ	WC/Hgt ratio	*WC/Hgt ratio
TC	r	-0.079	**-0.370**	-0.092	**-0.303**	-0.171	-0.086	0.086	-0.105	-0.106	0.058	-0.060	0.126	**0.283**	**-0.390**	**0.273**	**0.303**
	p	0.371	**0.012**	0.299	**0.043**	0.053	0.575	0.333	0.493	0.234	0.706	0.501	0.410	**0.001**	**0.008**	**0.002**	**0.043**
Trig	r	0.058	0.150	0.094	0.123	**0.250**	0.182	0.003	-0.002	**0.316**	**0.327**	0.139	0.077	**0.519**	0.140	**0.469**	**0.447**
	p	0.514	0.326	0.288	0.420	**0.004**	0.231	0.975	0.990	**<0.0001**	**0.028**	0.116	0.614	**<0.0001**	0.358	**<0.0001**	**0.002**
LDL	r	-0.025	-0.285	0.012	-0.135	0.018	0.116	0.093	0.052	0.022	0.050	0.064	-0.039	**0.637**	**-0.295**	**0.670**	**0.609**
	p	0.778	0.058	0.891	0.376	0.843	0.446	0.293	0.735	0.806	0.745	0.471	0.800	**<0.0001**	**0.049**	**<0.0001**	**<0.0001**
HDL	r	-0.084	**-0.382**	**-0.179**	**-0.442**	**-0.399**	**-0.353**	0.043	**-0.365**	**-0.335**	-0.063	**-0.194**	**0.341**	**-0.641**	**-0.399**	**-0.626**	**-0.600**
	p	0.345	**0.010**	**0.042**	**0.002**	**<0.0001**	**0.017**	0.626	**0.014**	**<0.0001**	0.680	**0.028**	**0.022**	**<0.0001**	**0.007**	**<0.0001**	**<0.0001**
TC/HDL ratio	r	0.027	0.039	0.118	0.184	**0.283**	**0.333**	0.015	0.275	**0.252**	0.044	0.152	-0.254	**1.000**	0.039	**0.978**	**1.000**
	p	0.763	0.800	0.182	0.227	**0.001**	**0.025**	0.870	0.068	**0.004**	0.775	0.085	0.092	**<0.0001**	0.800	**<0.0001**	**<0.0001**
LDL/HDL ratio	r	0.071	0.126	0.164	**0.223**	**0.271**	-0.127	0.062	0.052	**0.227**	0.126	**0.195**	**-0.183**	**0.978**	0.098	**1.000**	**0.303**
	p	0.423	0.157	0.063	**0.011**	**0.002**	0.155	0.483	0.559	**0.010**	0.157	**0.026**	**0.039**	**<0.0001**	0.271	**<0.001**	**0.001**
CRP	r	**0.276**	0.293	**0.322**	**0.403**	0.039	0.117	**0.275**	**0.397**	0.044	-0.036	**-0.285**	**-0.354**	**0.253**	0.259	**0.307**	**0.323**
	p	**0.002**	0.051	**<0.0001**	**0.006**	0.657	0.443	**0.002**	**0.007**	0.617	0.812	**0.001**	**0.017**	**0.004**	0.086	**<0.0001**	**0.031**

*adjusted for gender

BAZ-BMI-for age z-score; HAZ-height-for-age z-score, TSF-triceps skin fold, SSF-subscapular skin fold, SST-subscapular- to- triceps, WC-waist circumference, HC-hip circumference, WC/Hgt- waist circumference-to-height, CRP-C-reactive protein, TC-Total cholesterol, Trig-triglycerides, HDL- High density lipoprotein, LDL-low density lipoprotein

Table 3: Spearman's correlation coefficients in White adolescents adjusted for gender (n=69)

***adjusted for gender**

BAZ-BMI-for age z-score; HAZ-height-for-age z-score, TSF-triceps skin fold, SSF-subscapular skin fold, SST-subscapular- to- triceps, WC-waist circumference, HC-hip circumference, WC/Hgt- waist circumference-to-height, CRP-C-reactive protein, TC-Total cholesterol, Trig-triglycerides, HDL- High density lipoprotein, LDL-low density lipoprotein

		BMI	*BMI	WC	*WC	WHR	*WHR	TSF +SSF	*TSF SSF	+SSF/ TSF	*SSF/ TSF	SST ratio	*SST ratio	BAZ	*BAZ	WC/Hgt ratio	*WC/Hgt ratio
TC	r	0.185	0.325	0.184	**0.360**	-0.018	0.233	0.285	0.290	0.003	0.015	0.238	-0.277	**0.337**	0.151	**0.317**	0.314
	p	0.134	0.057	0.129	**0.034**	0.880	0.178	0.018	0.091	0.984	0.931	0.049	0.108	**0.005**	0.387	**0.008**	0.066
Trig	r	0.140	0.072	0.166	0.036	0.090	0.067	0.162	-0.031	**0.288**	**0.510**	0.181	0.121	**0.420**	-0.113	**0.362**	0.311
	p	0.251	0.681	0.174	0.837	0.463	0.702	0.183	0.861	**0.016**	**0.002**	0.137	0.489	**<0.0001**	0.519	**0.002**	0.069
LDL	r	**0.293**	**0.426**	**0.329**	**0.483**	0.166	**0.365**	**0.309**	0.313	0.087	0.035	**0.349**	**-0.307**	**0.592**	0.274	**0.604**	**0.586**
	p	**0.015**	**0.011**	**0.006**	**0.003**	0.172	**0.031**	**0.010**	0.067	0.478	0.843	**0.003**	**0.072**	**<0.0001**	0.112	**<0.0001**	**<0.0001**
HDL	r	-0.248	-0.149	**-0.316**	-0.197	**-0.366**	**-0.369**	-0.085	0.015	**-0.254**	-0.171	**-0.286**	-0.013	**-0.670**	-0.048	**-0.680**	**-0.697**
	p	0.040	0.393	**0.008**	0.257	**0.002**	**0.029**	0.488	0.931	**0.035**	0.327	**0.017**	0.943	**<0.0001**	0.785	**<0.0001**	**<0.0001**
TC/HDL	r	**0.412**	**0.371**	**0.442**	**0.415**	**0.289**	**0.458**	**0.316**	0.172	**0.222**	0.174	**0.457**	-0.164	**1 .000**	0.254	**0.983**	**0.983**
ratio	P	**<0.0001**	**0.028**	**0.000**	**0.013**	**0.016**	**0.006**	**0.008**	0.322	**0.067**	0.317	**<0.0001**	0.348	**<0.0001**	0.140	**<0.0001**	**<0.0001**
LDL/HDL	R	**0.431**	**0.601**	**0.478**	**0.605**	**0.342**	**0.422**	**0.292**	**0.531**	**0.253**	**0.282**	**0.469**	**-0.276**	**0.983**	**0.268**	**1.000**	**0.628**
ratio	P	**<0.0001**	**<0.0001**	**<0.0001**	**<0.0001**	**0.004**	**<0.0001**	**0.015**	**<0.0001**	**0.036**	**0.020**	**<0.0001**	**0.023**	**<0.0001**	**0.027**	**<0.0001**	**<0.0001**
CRP	R	0.153	0.141	0.180	0.191	0.133	0.071	0.109	-0.026	0.116	0.024	-0.088	0.056	**0.241**	0.253	0.109	0.022
	P	0.209	0.419	0.138	0.271	0.275	0.684	0.372	0.881	0.342	0.890	0.474	0.748	**0.046**	0.143	0.375	0.902

The predictions of skinfolds indices were inconsistent among the indices in both races (Table 4).

Table 4: Linear regression models for assessing the association between anthropometric indices and dyslipidemia in black and white adolescents

SEE = standard error of the estimate

Total (n=198)				**Africans (n=129)**			**Whites (n=69)**		
Dependent variables	**Independent variables**	**β**	**p-value**	**Independent variables**	**B**	**p-value**	**Independent variables**	**β**	**p-value**
TC	Constant	3.677	<0.0001	Constant	4.881	<0.0001	Constant	5.390	0.002
	SST ratio	0.252	0.003	WC	-0.148	0.095	WHR	-0.283	0.041
	BAZ	-1.191	0.027	TSF+ SSF	0.185	0.037	WC/Hgt ratio	0.422	0.003
	Race	0.325	<0.0001						
R = 0.371, R^2 = 0.124, SEE = 0.853, *p<0.0001*				**R = 0.219, R^2 = 0.033, SEE = 0.801, *p=0.045***			**R = 0.362, R^2 =0.105 , SEE = 0.908, *p=0.010***		
Trig	Constant	-2.539	<0.0001	Constant	-0.027	0.920	Constant	-2.828	<0.0001
	SSF/TSF ratio	0.141	0.017	SSF/TSF	0.236	0.009	BAZ	-0.568	<0.0001
	BAZ	-0.528	<0.0001	WC/Hgt ratio	0.148	0.092	WC/Hgt ratio	0.887	<0.0001
	WC/Hgt ratio	0.714	<0.0001						
	Race	0.315	<0.0001						
R = 0.618, R^2 = 0.369, SEE = 0.369, *p<0.0001*				**R = 0.306, R^2 = 0.079, SEE = 0.335, *p=002***			**R = 0.757, R^2 = 0.561, SEE = 0.393, *p<0.0001***		
LDL	Constant	-0.411	0.492	Constant	0.449	0.578	Constant	0.401	0.577
	WHR	-0.127	0.047	WHR	-0.178	0.039	WC/Hgt ratio	0.383	0.001
	TSF +SSF	0.112	0.082	TSF + SSF	0.167	0.054			
	BAZ	-0.174	0.058	BAZ	-0.283	0.058			
	WC/Hgt ratio	0.320	<0.0001	WC/Hgt ratio	0.314	0.035			
	Race	0.384	<0.0001						
R = 0.480, R^2 = 0.211, SEE = 0.689, *p<0.0001*				**R = 0.311, R^2 = 0.068, SEE = 0.633, *p=0.013***			**R = 0.383, R^2 = 0.134, SEE = 0.769, *p=0.001***		
HDL	Constant	2.230	<0.0001	Constant	2.743	<0.0001	Constant	2.654	<0.0001
	SSF/TSF	-0.148	0.036	SST ratio	-0.306	0.003	SST ratio	-0.336	0.014
	WC/Hgt ratio	-0.252	<0.0001	WC/Hgt ratio	-0.386	<0.0001	WC/Hgt ratio	-0.569	<0.0001
	Gender	-0.249	<0.0001						
R = 0.419, R^2 = 0.163, SEE = 0306, *p<0.0001*				**R = 0.334, R^2 = 0.098, SEE = 343, *p=0.001***			**R = 0.467, R^2 = 0.194, SEE = 250, *p<0.0001***		
TC/HDL ratio	Constant	-2.568	<0.0001	Constant	-1.294	0.198	Constant	-1.347	0.063
	BAZ	-0.364	<0.0001	SSF/TSF ratio	0.143	0.096	WC/Hgt ratio	0.654	<0.0001
	WC/Hgt ratio	0.663	<0.0001	BAZ	-0.457	0.002			
	Gender	0.130	0.032	WC/Hgt ratio	0.567	<0.0001			
	Race	0.292	<0.0001						
R = 0.580, R^2 = 0.323, SEE = 0.756, *p<0.0001.*				**R = 0.389, R^2 = 0.131, SEE = 747, *p<0.0001***			**R = 0.654, R^2 = 0.419, SEE = 0.767, *p<0.0001***		
LDL/HDL ratio	Constant	-3.088	<0.0001	Constant	0.010		Constant	-1.958	0.004
	BAZ	-0.298	0.001	BAZ	0.001		WC/Hgt ratio	0.628	<0.0001
	WC/Hgt ratio	0.622	<0.0001	WC/Hgt ratio	<0.0001				
	Gender	0.130	0.033						
	Race	0.290	<0.0001						
R = 0.567, R^2 = 0.307, SEE =0.704 , *p<0.0001*				**R = 0.380, R^2 = 0.131, SEE = 0.698, *p<0.0001***			**R = 0.628, R^2 = 0.386, SEE = 0.709, *p<0.0001***		

Discussion

In both races the prevalence of abnormal lipid values were high but gender seemed to affect TC, Trig, LDL-C, TC/HDL ratio and LDL/HDL ratio in both races. The most common form of dyslipidemia found in the current study was low HDL-C a finding previously reported in adolescents[18]. These values are however comparable with what has been reported in other developing countries worldwide[13, 19].

Hypercholesterolemia and elevated concentrations of LDL-C in adolescents have been linked to genetic susceptibility. It is a well-known finding that family history and low birth weight contribute to the pathogenesis of CVD[7, 20]. These findings support the hypothesis of fetal origins of cardiovascular and metabolic diseases in later life[21]. Young people with a family history of high blood pressure and Type 2 diabetes, irrespective of their adipocity had significantly higher insulin and abnormal lipid levels[20], and tended to have greater fat mass. However, elevated TC has been found in adolescents with and without familial history of premature CVD events[22].

Unfortunately in the current study family history of CVD in the studied adolescents was not recorded as this could have added valuable information on the observed phenomenon. In order to avoid over speculation it is necessary to keep in mind that a number of interrelated factors are often associated with and may contribute to the development of dyslipidemia in adolescents[20]. But on the other hand it has been previously reported that in South Africa the prevalence of CVDs is increasing at an alarming rate in all races[23, 24]. This has been partly linked to the nutrition transition the country is undergoing[25] with a shift to a more westernised lifestyle including fatty food and an increase in the intake of fast foods by the South African population[26].

It is known that diet is modulated by several effects and it has been established that it is an important determinant of plasma lipids. Serum TC levels have been found to correlate with cholesterol and saturated fat intake[27]. On the other hand replacement of fat by carbohydrates in the diet results in significant reduction of HDL-C concentrations[28]. This could be a possible explanation for the observed increase in low HDL-C levels especially in blacks who even though undergoing the nutrition transition their diet is still largely made up of carbohydrates. Unsurprisingly the same has been reported in studies done on adolescents from populations with a high carbohydrate intake[29].

Evidence shows that elevated TC and LDL-C levels increase the risk of CVD. Others have also revealed that low HDL-C levels are independent risk factors for atherosclerotic vascular disease[30]. It has been reported that individuals with low HDL-C have an abnormal HDL sub-class distribution, with lower levels of large particles and increased levels of small HDL[31]. This abnormality in HDL sub-populations is associated with CHD prevalence[32] and increased recurrence of coronary events[33]. Though the above associations have been reported in adults only, recent evidence show that atherosclerosis begin to manifest itself early in life and its initial stages are associated with adverse lipid profiles in children and adolescents[11,12]. Thus the above can be seen as suggesting that the abnormalities found in the current study's adolescents may predispose them to increased coronary heart disease risk later in life.

Elevated TC levels in childhood have been shown to track into adulthood[3, 11, 34], a phenomenon observed also with measures of adiposity especially BMI[34]. Moreover, previous researchers have reported that when there is risk factor clustering in adolescence as observed in the current study (results not shown),these adolescents are at an increased risk of developing CVDs in adulthood [12, 34].

In both races TC, Trig, LDL, TC/HDL ratio and LDL/HDL ratio were positively associated with both BAZ and WC/Hgt ratio. HDL on the other hand was inversely associated with BAZ and WC/Hgt ratio as well as WC. These findings are congruent with what has been found by Lima et al,[13]. The association between adipocity and abnormal lipid levels have long been established [35], with longitudinal changes in relative weight being associated with changes in these risk factors[36]. In addition these findings are supplemented by the observation that linear regression models revealed WC/Hgt ratio as the most predominant factor predicting most measured lipid parameters. This opens a new window for research into the use of anthropometric indices as surrogate measures to screen for dyslipidemia among other conditions, an area that still requires further research.

On the other hand, the use of other indices such as skinfold thickness still need further research with larger

epidemiological studies. This could be of important public health implication and reduce the risks associated with dyslipidemia if it can be detected early in adolescence especially in individuals with a familial history of dyslipidemia. Thus the current results show that even at this early stage abdominal fat deposition contribute to an adverse lipid profile[18].

Studies have linked the association between hypertriglyceridemia and central obesity to the increased number and size of adipocytes in the abdominal region, which promote insulin resistance and thus intensifying the release of free fatty acids (FFA) into the circulation. The FFA then provide a substrate for triacylglycerol synthesis in the liver, leading to increased hepatic release of Trig rich very low density lipoprotein particles into the circulation[37].

Hyperinsulinaemia is also known to enhance hepatic VLDL synthesis, thus it may directly contribute to the increased plasma Trig and LDL-C levels[38]. Resistance to the action of insulin on lipoprotein lipase in peripheral tissues may also contribute to elevated Trig and LDL- C (39). It has also been suggested that insulin resistance may be involved in the reduced HDL-C levels in type 2 diabetes patients. As such the findings in the present study suggest the need to monitor lipid levels in adolescents.

Gender and pubertal development stage are the other factors that have been shown to influence the lipid profile of individuals[14], while other evidence has shown that BMI influences Trig levels irrespective of age and gender[18]. However, in the current study no data was available on the adolescents' pubertal development stages to can assist in adding to this pool of literature. On the contrary adjusting for gender affected the association between lipid parameters and measures of adiposity supporting the earlier findings that gender does play a role in the adolescents' lipid profile exhibited probably due to differences in hormonal changes[14].

Though it has been shown that, most risk factors do track into adulthood; substantial proportions of young people with high risk levels had no risk levels in adulthood[40]. These discrepancies have been associated with changes in lifestyle habits[40], suggesting that modifiable risk factors in the time between adolescence and adulthood have the potential to shift adolescents with high risk lipid levels into adults with low-risk levels and vice versa[40]. These modifiable risk factors include adipocity, smoking, hormonal contraceptive use[40], physical activity[41], upwards social mobility from lower socio-economic status to higher socio-economic status and adoption of a healthier diet[40]. The above findings show that all is not lost in young children as interventions to change the modifiable risk factors can aid in reducing the adverse effects of impaired lipid tracking from adolescence by reversing them.

Conclusion

The study showed that whites exhibited more associations between dyslipidemia and anthropometric indicators as compared to black adolescents with WC/Hgt ratio being the index associated with most measured lipid parameters, suggesting that there might be differences in the lipid metabolism or even susceptibility to risk factors in adolescents. Furthermore, the association between dyslipidemia and adipocity in this study adds to the current literature that it is necessary to introduce screening and preventative measures at an early age due to the adverse consequences posed by tracking of these risk factors into adulthood, but these results have to be cautiously interpreted as the smaller sample sizes in both populations might have affected the results in one way or another as such warranting larger epidemiological studies in this setting.

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