

## LIPID PROFILE, HYPERGLYCAEMIA, SYSTEMIC INFLAMMATION AND ANTHROPOMETRY AS CARDIOVASCULAR RISK FACTORS AND THEIR ASSOCIATION WITH DIETARY INTAKES IN CHILDREN FROM RURAL COFIMVABA, EASTERN CAPE, SOUTH AFRICA

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### ABSTRACT

**Introduction:** The aim of this study was to estimate the mean prevalence of dyslipidaemia, hyperglycaemia, systemic inflammation and anthropometry as CVD risk factors as well as dietary intakes and to investigate associations between these CVD risk factors and dietary intakes among apparently healthy school-aged girls and boys, attending five purposively selected schools in rural Cofimvaba, situated in the Eastern Cape Province of SA.

**Methods:** A random sample of 233 children, aged 6-18 years, was used for dietary intake, anthropometric and fasting biochemical measurements. Data were analysed on SPSS, version 22.0, for descriptive analyses as well as Levene's test for equality of variances, one-way analysis of variance and Pearson correlations.

**Results:** Only 1.3% of the children presented with hypercholesterolaemia ( $TC \geq 5.18$  mmol/l) and 2.1% and 7.3% had elevated low density lipoprotein-cholesterol (LDL-C) and triglyceride levels respectively whereas 42.5% had low high-density lipoprotein-cholesterol (HDL-C) levels. No specific trends were observed among the age groups or genders. The highest prevalence of abnormal lipid markers was among the 6-8 year old girls. Hyperglycaemia was observed in 10.3% and systemic inflammation in 20.2% of the children. Most of the dietary intake variables showed normal intakes, except for total energy and dietary fibre that showed a low intake.

**Conclusion:** CVD risk is a problem in these children and adolescents as an undesirable lipid profile of high prevalence of low HDL-C despite a low prevalence of hypercholesterolaemia, hypertriglyceridaemia and high LDL-C, was found, with younger children being more at risk. Hyperglycaemia and systemic inflammation was also prevalent, but no obesity was observed. Healthy lifestyles, including physical exercise, should be promoted and nutrition education and awareness programmes implemented.

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## INTRODUCTION

South Africa (SA) is a country in nutritional transition, characterised by a “double burden of malnutrition” with under- and over-nutrition occurring in the same population (Shisana et al. 2013). Although the prevalence of infectious diseases has decreased similarly to other developing countries, the emerging epidemic of non-communicable diseases (NCDs) remains a challenge (Shisana et al. 2013). Of these NCDs, cardiovascular disease (CVD), is one of the main contributors of poor health (Shisana et al. 2013). Socio-economic challenges such as low income contribute to the risk for CVD with the most disadvantaged being more at risk (Ochoa-Avilés et al. 2014). CVD is the leading cause globally, of mortality and morbidity and it was estimated that by 2013, 23.3 million deaths would have been caused by CVD (Alwan 2011). At the South African Summit on the Prevention and Control of NCDs in 2011, consensus was reached that an intensified national strategic plan was needed to prevent and treat NCDs, including CVD. This plan should include the early detection of NCDs and strategies for behavioural changes (DoH 2013).

Childhood obesity is increasing globally (Martin et al. 2015) and in SA the prevalence of overweight and obesity is 16.5% and 7.1% for girls and 11.5% and 4.7% for boys aged 2-14 years respectively (Shisana et al. 2013). Childhood and adolescent obesity is associated with CVD risk factors such as dyslipidaemia, compromised vascular function, hypertension (Boodai et al. 2014), inflammation (Willerson & Ridker 2004) as well as insulin resistance (Boodai et al. 2014) and abnormal glucose metabolism (Boodai et al. 2014; Amutha & Mohan 2016). A high prevalence of dyslipidaemia, overweight and abdominal obesity have been reported in adolescents and/or children from developing countries such as: Ecuador (Ochoa-Avilés et al. 2014), Iranian (Kelishadi et al. 2004), Mexico (Posadas-Sanchez et al. 2007) and Kuwait (Boodai et al. 2014), but also from developed European countries (Martin et al. 2015). The incidence of elevated serum cholesterol levels in children from different countries have been associated with elevated serum cholesterol levels in adults in the same countries and also with higher CVD mortality rates (American Academy of Pediatrics 1992). Dyslipidaemia, an abnormality in circulating serum cholesterol, triglycerides, high-density and low-density lipoproteins, is thus one of the co-morbidities of obesity and childhood

obesity and CVD and it has been found that the prevalence of CVD risk factors, especially dyslipidaemia and obesity, has increased among children and adolescents since the 2000's (Kelishadi et al. 2004; Boodia et al. 2014; Martin et al. 2015). Furthermore, dietary quality, specifically the intakes of fresh vegetables and fruit, whole grains and fish, has been found to have a protective effect on CVD whereas a high intake of added sugar, sodium and refined carbohydrates (Kell et al. 2014; Ochoa-Avilés et al. 2014) and dietary fat (Kelishadi et al. 2004) has been identified as possible risk factors for the development of CVD in adults (Ochoa-Avilés et al. 2014) and children (Kelishadi et al. 2004).

Against this background and the fact that information about the lipid profile and prevalence of dyslipidaemia and hyperglycaemia in children and adolescents is scarce in SA, the aim of the present study was, therefore, to estimate the mean prevalence of dyslipidaemia, hyperglycaemia, systemic inflammation and anthropometry as CVD risk factors as well as dietary intakes and to investigate associations between these CVD risk factors and dietary intakes among apparently healthy school-aged girls and boys, attending five purposively selected schools in rural Cofimvaba, situated in the Eastern Cape Province of SA.

## METHODS

This study was undertaken in one of the eleven poorest municipalities in the Eastern Cape, namely the Intsika Yethu municipality (Chris Hani District Municipality 2011) where 87.1% of the adult population is unemployed and living in poverty (Intsika Yethu Local Municipality 2011). This cross-sectional study was performed as the baseline evaluation of a larger project with the aim of piloting a model for addressing nutritional problems and micronutrient deficiencies resulting from food insecurity among school children and a breakfast school feeding programme that is linked to gardening programmes and provision of locally grown food in rural schools.

## Ethics

The study protocol was approved by both the Senate Research and Innovation Ethics Committee of the Vaal University of Technology (20130520-3) and the Council of Scientific and Industrial Research (CSIR) (75/2013).

### Sampling and respondents

The following sample size calculation (The Survey System, 2013) was used to determine the sample size for a representative sample:

$$ss = \frac{Z^2 * (p) * (1-p)}{c^2}$$

Where:

Z = Z value of 1.96 for 95% confidence level

p = 80 percent expressed as decimal (0.8 used for sample size needed)

c = confidence interval of 6.5, expressed as 0.065.

A statistically representative sample size of 218 children was required. The Department of Basic Education purposively selected five schools (n=1 250 learners) out of a total of 21 schools (N=5 250 learners 6 to 18 years old in Cofimvaba) for inclusion in the study. These five schools thus represented 24% of the school-aged children in the study area. The researchers visited the selected schools before the study commenced to explain the objectives of the project and acquire informed consent from the school management, the parents/caregivers of the children attending these schools and the children aged 12 years and older (HPCSA 2008). Assent from the younger children was also obtained in writing. A total of 556 informed consent forms were signed. The inclusion criteria included: all school children of both genders aged 6-18 years of age with no known allergies attending the five purposively selected schools. After screening for the inclusion and exclusion criteria, 523 children, for whom the caregivers and children had signed informed consent and assent respectively, were eligible for inclusion in the study. The sample was stratified for gender and age and 240 children selected from those for whom informed consent/assent had been obtained. Measurements were done on different days over a period of four days in August 2013.

### Data collection

Four students from the Department of Food and Consumer Sciences at the Walter Sisulu University were recruited and trained during two eight-hour sessions by a registered dietitian and public health nutritionist as fieldworkers. Training included sessions on ethical and general research philosophies applicable to research in human beings (2 hours), the importance of accurate measurements for obtaining correct and achievable objectives (2

hours), as well as completing the 24-hour recall questionnaires without interviewer bias (2 hours) according to the four-stage, multiple-pass interviewing procedure described by Gibson (2005). Role play (n=10 hours) was employed in the training sessions whereby the fieldworkers had to interview and complete multiple 24-hour recall questionnaires with each other. This process was observed by the researchers to ensure that the correct data collection procedures were followed and incorrect procedures rectified.

A 24-hour recall questionnaire from a previous study (Oldewage-Theron et al. 2006) in a similar community was administered by the fieldworkers for a week day and a weekend day in two sequential weeks for the children aged 10 years and older. The same procedures were followed for the younger children with the assistance of the fieldworkers through one-on-one interviews with the parents/legal guardians. A four-stage, multiple-pass interviewing procedure was used (Gibson 2005) to ensure that 24-hour data were correctly captured. Food models and locally used household utensils were available to assist the fieldworkers for quantifying the portion sizes of the foods consumed.

The age of the children was recorded from school records. Body weight and height were measured by a registered dietician (SA) and public health nutritionist (United States of America [USA]) according to standard procedures (Lohman 1988) with a calibrated Philips electronic scale, model HF350 (135kg/100g) with a two point decimal precision and a Scales 2000 stadiometer respectively. All measurements, which were not to vary by more than 0.1 kg and 0.1 cm respectively, were taken twice and the average of the two measurements recorded.

The children were requested to fast from going to bed the previous evening and the parents/legal guardians were reminded of this by Short Message Service (SMS) the day before blood was drawn. A venous blood sample was drawn before 10h00 using a vacutainer needle with minimal use of tourniquets by registered nursing practitioners. Thereafter breakfast was served immediately. The blood was placed on ice and protected from direct sunlight until separation within two hours of blood collection. Serum was stored at -80 °C for a maximum of two weeks before analyses to prevent changes in lipoprotein composition (Hodson et al. 2008). All blood parameters were analysed according to

standard protocol in the biochemical analysis laboratory at the tertiary institution. Total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C) and triglycerides (TG) were analysed on a Konelab™ analyser by means of the colorimetric method. A coefficient of variation (percent CV) between runs of 1.2–2.8% was obtained for all serum variables analysed. The Friedewald formula was used to calculate low-density lipoprotein-cholesterol (LDL-C) (Warnick et al. 1990). Although SA has specific guidelines for specifying dyslipidaemia (SA Heart & Lassa 2012), these were not applied as the guidelines pertained to adults. For this study, the cut-off points used for dyslipidaemia were the presence of one or more of the following: TC  $\geq 5.18$  mmol/l, LDL-C  $\geq 3.37$  mmol, HDL-C  $< 1.04$  mmol/l, and TG  $\geq 1.12$  mmol/l (children 0-9 years old) and 1.47 mmol/l (children 10-19 years old) (US Department of Health and Human Services [HHS] 2012).

A colorimetric method was used for analysis of high-sensitive C-reactive protein (HS-CRP) and serum glucose on the Konelab 20i analyser. The cut-off point used for HS-CRP was  $> 3.0$  mg/L (Rietszell & De Buyzere 2012) and  $> 6.1$  mmol/l for fasting serum glucose (WHO 2006). HS-CRP was included as a reliable measure of underlying systemic inflammation and a strong predictor of future vascular events. Elevated HS-CRP is associated with an increased CVD risk, even in the absence of dyslipidaemia (Willerson & Ridker 2004). Serum fasting glucose levels were also included as it has been identified as a risk factor for CVD (Park et al. 2013).

### Data analyses

The data were captured on an Excel spreadsheet and stored in a computer database. After checking for missing values and capturing errors, a complete database existed for 233 respondents, 115 girls and 118 boys. The Statistical Package for Social Sciences (SPSS) program (Version 22) was used for all statistical analyses and  $p < 0.05$  was considered significant for all statistical tests. Normal probability plot and Kolmogorov-Smirnov tests were used to test for the normal distributions of all variables. Most variables were not normally distributed and, therefore, medians with interquartile ranges (IQRs) were used for data analyses.

The 24-hour recall data was coded, captured and analysed, supervised and controlled by a registered nutritionist. Food Finder dietary analysis software program version 3, developed

by the Medical Research Council and based on the South African Food Consumption Tables (Langenhoven et al. 1991) was used to translate the consumed foods into nutrients. Since maize meal and bread have been fortified since 2002, codes for these two food products, including the fortification levels, were included in the software program. Medians were calculated for daily macronutrient and for the various dietary fat intakes, including total fat, saturated fatty acids (SFA), mono-unsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), trans fatty acids (TFA), linoleic acid, linolenic acid and dietary cholesterol, per person per day (total of two 24-hour recall/2) and compared with the Dietary Reference Intakes (DRIs) for the age groups, 4-8, 9-13 and 14-18 years for total energy intakes and Adequate Intake (AI) for dietary fibre (IoM 2002). Both EAR and AI form part of the DRIs and the EAR is used for dietary intakes of groups and whereas the AI is used where the EAR is not available (IoM 2000). The Food and Agriculture Organization (FAO) and World Health Organization (WHO) guidelines for prevention of disease for the dietary fat variables and sodium intakes (FAO/WHO 2010), and the Acceptable Macronutrient Distribution Ranges (AMDR) for total protein, fat and carbohydrate intakes (Senekal et al, 2016) were used.

Anthropometric data were analysed using the WHO AnthroPlus version 1.0.2 statistical software (WHO, undated). Stunting was defined as height-for-age z score (HAZ)  $< -2SD$  (severe stunting,  $< -3SD$ ), underweight as weight-for-age z-score (WAZ)  $< -2SD$  (severe underweight,  $< -3SD$ ), thinness as BMI-for-age z score (BMIZ)  $< -2SD$  (severe thinness,  $< -3SD$ ) and overweight as BMIZ  $> +1 SD$  (obesity,  $> +2SD$ ) (WHO 2007).

Medians and quartiles were calculated for the biochemical variables and compared to the cut-off points provided for each variable (HHS 2012). The percentage of respondents with abnormal values was also calculated. Two-tailed independent t-test for equality of variances was performed to measure significant differences between the anthropometric and biochemical variables of the boys and girls. One-way analysis of variance (ANOVA) was performed to measure significant differences between the anthropometric and biochemical variables of the different age groups. Pearson correlations were used to determine significant relationships between dietary intake- and biochemical variables. Only significant relationships are

**TABLE 1: PREVALENCE OF UNDERWEIGHT, STUNTING, THINNESS, OVERWEIGHT AND OBESITY ACCORDING TO THE WHO GROWTH STANDARDS FOR THE TOTAL GROUP OF CHILDREN**

Classification		Girls (n=114) n (%)	Boys (n=118) n (%)	Total group (n=232) n (%)	Significant differences between gender p
<b>Underweight (WAZ) (0-10 years old) (Girls, n=41 and Boys, n=45)</b>					<b>0.834</b>
<-3 SD	Severely underweight	0	0 (0.0)	0 (0.0)	
≥-3<-2 SD	Underweight	1 (2.4)	1 (2.2)	2 (2.3)	
<b>Stunting (HAZ)</b>					<b>0.649</b>
<-3 SD	Severely stunted	2 (1.8)	0 (0.0)	2 (0.9)	
≥-3<-2 SD	Stunted	3 (2.6)	8 (6.8)	11 (4.7)	
<b>BMIZ (Thinness/Wasting/Overweight/Obesity)</b>					<b>0.031</b>
<-3 SD	Severe thinness	3 (2.6)	1 (0.8)	4 (1.7)	
≥-3<-2 SD	Thinness	2 (1.8)	2 (1.7)	4 (1.7)	
≥-2<+1 SD	Normal weight	83 (72.8)	98 (83.1)	181 (78.0)	
≥+1<+2 SD	Possible risk of overweight	21 (18.4)	12 (10.2)	33 (14.2)	
≥+2 SD<3SD	Overweight	5 (4.4)	5 (4.2)	10 (4.3)	
≥+3 SD	Obesity	0 (0.0)	0 (0.0)	0 (0.0)	

reported in the results.

## RESULTS

The sample consisted of 50.6% (n=115) girls and 49.4% (n=118) boys with 23.2% of the children in the 6-8 year old, 35.2% in the 9-13 year old and 41.6% in the 14-18 year old group. The mean±SD age of the children in the 6-8 year old group was 7.0±0.8 years and 11.0±1.4 years and 16.0±1.4 years in the 9-13 year and 14-18 year old groups respectively.

The results are summarised in five tables. In Table 1 the prevalence of the respondents' nutritional status according to the WHO growth standards for gender is given. In Table 2 the median and interquartile range for the anthropometric indices and biochemical parameters are summarised according to age group and gender. The prevalence of respondents with abnormal anthropometric indices and biochemical parameters is summarised in Table 3. In Table 4 the median and interquartile range for the nutrient intake parameters are summarised according to age group and gender and Table 5 is a summary of the top 40 most frequently foods consumed by the participants. The anthropometric results in Table 1 indicated that only 2.3% of the respondents 6-10 year old respondents were underweight. Underweight is usually the result of acute insufficient energy intake (UNICEF 2007). More boys (6.8%) than girls (4.4%) were stunted, however, 1.8% of the girls were severely stunted compared to no boys being

severely stunted in the total sample, thus indicating chronic insufficient food and nutrient intake and/or frequent infections (UNICEF 2007). The median z-scores for all three age groups were within the normal range (Table 2) and no significant differences were observed between the age groups for WAZ (p=0.499) and BMIZ (p=0.903). A significant (p=0.020) difference was observed in HAZ between the age groups, but no significant (p=0.834) difference in HAZ between the boys (-0.54 [-1.23; 0.25]) and girls (-0.33, [-1.00; 0.40]) were observed. The BMIZ results showed a prevalence of 3.4% wasting when both genders are combined. The prevalence of overweight was 4.3% for all the children, however, 14.2% were at risk of overweight. None of the children were obese (Table 1). A significant (p=0.031) difference in BMIZ between the boys and girls were observed. Furthermore, the girls was thinner than the boys when the median was compared - -0.19 (-0.65; 0.38) and 0.06 (-0.55; 0.74) respectively in the 6-8 year old group. However, in both the 9-13 and 14-18 year old groups, the boys were thinner than the girls, but these differences were not significant (p=0.903).

The biochemical results are reflected in Table 2. All the biochemical variables showed normal median levels when compared with the reference values, except for the median HDL-C levels which were elevated irrespective of age and gender. No significant differences were observed among the three age groups and between boys and girls, except for serum glucose that showed a significantly (p=0.024)

**TABLE 2: DESCRIPTIVE PARAMETERS OF THE CHILDREN ACCORDING TO GENDER AND AGE, MEDIAN (25TH AND 75TH PERCENTILES)**

Variable	Normal reference range	Age group 6-8 years		Age group 9-13 years		Age group 14-18 years		Significant difference between boys and girls <sup>a</sup> and different age groups <sup>b</sup> p
		Girls (n=24, 10.3%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	Boys (n=30, 12.9%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	Girls (n=43, 18.5%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	Boys (n=39, 16.7%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	Girls (n=48, 20.6%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	Boys (n=49, 21.0%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> percentile)	
HAZ	>-2 (WHO 2007)	-0.15 (-0.96; 0.59)	-0.28 (-0.19; 0.35)	-0.41 (-1.04; 0.25)	-0.41 (-0.95; 0.55)	-0.37 (-0.94; 0.25)	-0.68 (-1.52; 0.19)	0.834 <sup>a</sup> 0.020 <sup>b</sup>
WAZ	>-2 (WHO 2007)	-0.26 (-0.74; 0.52)	-0.06 (-0.41; 0.46)	0.06 (-0.68; 0.85)	-0.47 (-1.10; 0.77)			0.649 <sup>a</sup> 0.499 <sup>b</sup>
BMIZ	≥-2<+1 (WHO 2007)	-0.19 (-0.65; 0.38)	0.06 (-0.55; 0.74)	0.31 (-0.37; 0.97)	-0.31 (-1.09; 0.19)	0.29 (-0.48; 1.10)	-0.50 (-1.26; 0.17)	0.031 <sup>a</sup> 0.903 <sup>b</sup>
Serum cholesterol	<5.18 mmol/l (HHS 2012)	3.36 (2.87; 3.88)	2.98 (2.80; 3.36)	3.41 (2.78; 3.79)	3.22 (2.91; 3.69)	3.13 (2.72; 3.78)	3.16 (2.70; 3.85)	0.338 <sup>a</sup> 0.938 <sup>b</sup>
HDL-cholesterol	>1.04 mmol/l (HHS 2012)	1.07 (0.76; 1.42)	1.06 (0.78; 1.24)	1.15 (0.86; 1.39)	1.10 (0.81; 1.35)	1.11 (0.90; 1.29)	1.15 (0.89; 1.28)	0.411 <sup>a</sup> 0.740 <sup>b</sup>
LDL-cholesterol	≤3.37 mmol/l (HHS 2012)	1.74 (1.51; 2.19)	1.55 (1.36; 1.91)	1.84 (1.37; 2.21)	1.72 (1.50; 2.06)	1.74 (1.41; 2.13)	1.73 (1.25; 2.15)	0.248 <sup>a</sup> 0.909 <sup>b</sup>
Triglycerides	<1.12 (0-9 years)/1.47 (10-19 years) mmol/l (HHS 2012)	0.72 (0.58; 1.29)	0.75 (0.52; 1.15)	0.68 (0.55; 1.03)	0.90 (0.66; 1.12)	0.62 (0.50; 0.84)	0.82 (0.59; 1.08)	0.145 <sup>a</sup> 0.334 <sup>b</sup>
Serum glucose	<6.1 mmol/l (WHO 2006)	5.16 (4.57; 5.64)	5.41 (4.89; 5.77)	5.32 (4.96; 5.84)	5.25 (4.95; 5.51)	5.05 (4.20; 5.50)	5.37 (4.79; 6.00)	0.024 <sup>a</sup> 0.585 <sup>b</sup>
HS-CRP	≤3 mg/dl (Rietszell & De Buyzere 2012)	1.27 (0.73; 3.31)	1.85 (1.03; 2.62)	1.23 (0.70; 2.62)	1.00 (0.71; 2.39)	1.27 (0.78; 2.20)	1.54 (0.75; 2.83)	0.864 <sup>a</sup> 0.568 <sup>b</sup>

**TABLE 3: PREVALENCE OF RESPONDENTS WITH ABNORMAL BIOCHEMICAL BLOOD VALUES ACCORDING TO GENDER AND AGE**

Variable	Cut-off points for abnormal blood values	Age group 6-8 years		Age group 9-13 years		Age group 14-18 years	
		Girls (n=24, 10.3%)	Boys (n=30, 12.9%)	Girls (n=43, 18.5%)	Boys (n=39, 16.7%)	Girls (n=48, 20.6%)	Boys (n=49, 21.0%)
Serum cholesterol	≥5.18 mmol/l (HHS 2012)	1 (4.2)	1 (3.3)	0 (0.0)	0 (0.0)	0 (0.0)	1 (2.0)
HDL-cholesterol	≤1.04 mmol/l (HHS 2012)	12 (50.0)	15 (50.0)	18 (41.9)	18 (46.2)	19 (39.6)	17 (34.7)
LDL-cholesterol	≥3.37 mmol/l (HHS 2012)	1 (4.2)	1 (3.3)	0 (0.0)	0 (0.0)	2 (4.2)	1 (2.0)
Triglycerides	≥1.12 (0-9 years)/1.47 (10-19 years) mmol/l (HHS 2012)	7 (29.2)	7 (23.3)	0 (0.0)	3 (7.7)	4 (8.3)	8 (16.3)
Serum glucose	≥6.1 mmol/l (WHO 2006)	2 (8.3)	2 (6.7)	7 (16.3)	1 (2.6)	2 (4.2)	10 (20.4)
HS-CRP	>3 mg/dl (Rietszell & De Buyzere 2012)	6 (25.0)	7 (23.3)	8 (18.6)	8 (20.5)	7 (14.6)	11 (22.4)

higher median value (IQR) of 5.34 (4.89; 2.75) for the boys (IQR) than 5.17 (4.76; 5.58) for the girls.

Only 1.3% (3/233) of the children presented with hypercholesterolaemia (TC≥5.18 mmol/l) and 2.1% (5/233) and 12.4% (29/233) had elevated LDL-C (>3.37 mmol/l) and TG (≥1.12 and 1.47 mmol/l for children 0-9 and 10-19 years

respectively) levels respectively, whereas 42.5% (99/233) had low HDL-C (≤1.04 mmol/l) levels. Hyperglycaemia was observed in 10.3% (24/233) of the children. HS-CRP was assessed as a marker of inflammation and 20.2% (47/233) of the children had elevated HS-CRP (>3 mg/dl) levels. No specific trends were observed among the age groups or genders, except that hypercholesterolaemia was only observed in the

**TABLE 4: MACRONUTRIENT, SODIUM AND ADDED SUGAR INTAKES PER DAY OF THE RESPONDENTS AS MEASURED BY 24-HOUR RECALL**

Variable (unit of measure)	Age group 6-8 years			Age group 9-13 years			Age group 14-18 years		
	Girls (n=24, 10.3%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=30, 12.9%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)	Girls (n=43, 18.5%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=39, 16.7%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)	Girls (n=48, 20.6%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=49, 21.0%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)
Total energy (TE) (kJ)	6185 (4869; 7693)	6147 (4298; 7654)	7316 (b)/ 6896 (g)	6867 (6227; 8042)	7587 (5769; 9101)	9572 (b) / 6898 (g)	7084 (5679; 9088)	7121 (5795; 9372)	13238 (b)/ 9940 (g)
Total protein (g)	49 (37; 67)	46 (33; 59)	19	53 (43; 63)	55 (45; 68)	34	51 (41; 69)	54 (41; 71)	52 (b)/ 46 (g)
Total protein as %TE	15	14	10-35%TE	12	13	10-35%TE	12	12	10-35%TE
Plant protein (g)	23 (18; 25)	20 (14; 25)		25 (20; 32)	29 (23; 33)		27 (21; 30)	27 (22; 35)	
Animal protein (g)	27 (18; 44)	21 (15; 38)		29 (15; 36)	25 (17; 38)		25 (15; 37)	28 (14; 38)	
Total carbohydrate (g)	212 (168; 255)	209 (157; 250)	100	249 (201; 276)	280 (211; 299)	100	252 (183; 322)	248 (210; 320)	
Total carbohydrate as % TE	54	55	40-75%	57	57	40-75%	56	55	40-75%
Added sugar (g)	28 (14; 50)	31 (24; 40)		39 (26; 48)	35 (22; 51)		40 (21; 69)	31 (24; 48)	
Added sugar as %TE	7	8		9	7		9	7	
Total dietary fibre (g)	12 (8; 16)	9 (7; 12)	25	11 (9; 16)	13 (9; 17)	31 (b)/ 26 (g)#	13 (10; 16)	13 (10; 18)	38 (b)/ 26 (g)#
Total dietary fibre as %TE	3	3		3	3		3	3	
Total fat (g)	40 (29; 60)	42 (23; 55)	20-35%TE	46 (32; 60)	48 (31; 63)	20-35%TE	51 (27; 65)	49 (39; 71)	20-35%TE
Fat as %TE	24.2	25.8	20-35%TE	25.3	23.9	20-35%TE	27.2	26.0	20-35%TE
SFA (g)	14.12 (9.14; 19.72)	12.53 (6.75; 18.86)		14.75 (9.87; 18.14)	14.94 (11.06; 24.42)		14.74 (9.03; 20.59)	15.17 (9.78; 20.54)	

# AI = Adequate intake where no EAR available  
b= boy, g= girl

**TABLE 4: MACRONUTRIENT, SODIUM AND ADDED SUGAR INTAKES PER DAY OF THE RESPONDENTS AS MEASURED BY 24-HOUR RECALL - Continued**

Variable (unit of measure)	Age group 6-8 years			Age group 9-13 years			Age group 14-18 years		
	Girls (n=24, 10.3%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=30, 12.9%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)	Girls (n=43, 18.5%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=39, 16.7%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)	Girls (n=48, 20.6%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	Boys (n=49, 21.0%) Median (25 <sup>th</sup> ; 75 <sup>th</sup> per- centile)	EAR (IoM 2000, IoM 2002) / FAO and WHO guidelines (2010) / AMDR (Senekal et al. 2016)
SFA as %TE	8.6 13.96 (10.66; 21.47)	7.7 14.68 (8.29; 19.81)	< 10% total TE	8.1 16.35 (11.48; 21.12)	7.4 15.59 (10.43; 23.03)	< 10% total EI	7.9 16.36 (9.98; 21.88)	8.1 15.12 (11.74; 22.63)	< 10% total TE
MUFA (g)	8.5 9.1 (6.25; 14.98)	9.0 10.42 (5.35; 12.71)	Balance [total fat (SFA + TFA + PUFA)	9.0 11.66 (6.66; 17.44)	7.8 11.54 (6.04; 17.56)	Balance [total fat (SFA + TFA + PUFA)	8.7 11.27 (6.24; 19.97)	8.0 14.47 (8.81; 23.79)	Balance [total fat - (SFA + TFA + PUFA)
MUFA as %TE	5.5 1.63 (0.63; 2.64)	6.4 1.13 (5.35; 12.71)	6-11% total EI	6.4 1.65 (1.01; 2.23)	5.7 1.59 (0.78; 2.73)	6-11% total EI	6.0 1.70 (0.75; 2.52)	7.7 1.25 (0.37; 2.63)	6-11% total TE
TFA (g)	0.9 7.10 (4.24; 12.81)	0.7 8.73 (4.67; 11.59)	< 1% total EI	0.9 8.60 (5.04; 13.90)	0.8 8.90 (5.28; 14.58)	< 1% total EI	0.9 8.86 (4.43; 16.54)	0.7 11.26 (6.27; 21.50)	< 1% total TE
Linoleic acid (n-6) C:18:2	4.3 0.27 (0.20; 0.37)	5.4 0.26 (0.18; 0.36)	2.5-9% total EI	4.7 0.29 (0.21; 0.42)	4.4 0.32 (0.22; 0.50)	2.5-9% total EI	4.7 0.25 (0.18; 0.41)	6.0 0.22 (0.16; 0.39)	2.5-9% total TE
Linolenic acid (n-3) C:18:3	0.2 148.71 (78.68; 285.71)	0.2 130.18 (72.13; 190.09)	0.5-2% total EI	0.2 141.80 (104.63; 214.50)	0.2 134.75 (68.00; 224.25)	0.5-2% total EI	0.1 154.01 (85.28; 259.94)	0.1 162.45 (88.51; 324.76)	0.5-2% total TE
Dietary cholesterol (mg)	1358.12 (883.05; 1928.72)	1068.60 (710.11; 1401.85)	<300	1905.86 (1073.80; 2510.70)	2252.16 (1034.23; 2993.13)	<300	1595.00 (827.02; 3221.04)	1445.94 (839.34; 2203.37)	<300
Sodium (mg)			<2500			<2500			<2500

# AI = Adequate intake where no EAR available  
b= boy, g= girl

**TABLE 5: TOP 40 MOST COMMONLY CONSUMED FOOD ITEMS AS MEASURED BY THE 24-HOUR RECALL**

Food item	Total intake (g) for the group per day	Per capita intake (g) per day
Rice, white, cooked	46802.5	195
Sugar-sweetened beverages	27508	115
Tea, brewed	21820	91
Maize meal, stiff porridge	17861	74
Maize meal, crumbly porridge	17321	72
Chicken	17238	72
Bread/rolls, white	15835	66
Samp	12045	50
Maize meal, soft porridge	8880	37
Vetkoek, homemade	8833	37
Dried beans	8118	34
Potatoes	8053	34
Maas / sour milk	7960	33
Cabbage, cooked	7575	32
Mahewu/magou, Liquid	6090	25
Savoury snacks	4714	20
Biscuits	4552	19
Vegetable soup	4375	18
Milk, fresh	3955	16
Sugar, white, granulated	3676	15
Coffee, brewed/instant	3380	14
Apple, raw	3225	13
Egg, scrambled	2550	11
Carrot, boiled	2525	11
Processed meat	2183	9
Orange, raw (peeled)	2050	9
Gravy, meat	1825	8
Hardboiled sweets	1760	7
Spinach, boiled	1575	7
Yoghurt, fruit, low fat, sweetened	1200	5
Onions	1164	5
Pilchards in tomato sauce	993.5	4
Mutton, cooked	948	4
Pork	930	4
Breakfast cereal	916	4
Sunflower oil	899.5	4
Margarine	865	4
Maltabella	840	4
Beef mince, cooked	783	3
Banana, Raw (peeled)	725	3

6-8 year old group and the group with the highest prevalence of abnormal lipid markers was the 6-8 year old girls (Table 3).

The dietary intake data are shown in Table 4. The median energy and dietary fibre intakes were consistently low compared to adequate median intakes of carbohydrates and protein when compared to the EAR irrespective of gender and age. Compared to the FAO and WHO guidelines for the prevention of chronic

disease, the recorded percentages for the total fat, dietary cholesterol, SFA, MUFA, TFA and linoleic acid intakes were within the recommended ranges for all the groups (FAO/WHO 2010). The PUFA and linolenic intakes were much lower than the recommended goal of 6-11% and 0.5-2% in all the groups respectively. In Table 5, the main fat sources in the diet was indicated as fats forming part of meat as well as a per capita intake of 4 gram (g) of margarine and 4 g of sunflower oil. Median sodium intakes

were within the recommended guidelines, however, this is not a true reflection of the sodium intake as the sodium values in FoodFinder are incomplete, however, the analyses of the consumed food items as measured by the 24-hour recall questionnaires, showed a per capita intake of 2 grams of added table salt on the measuring days. A per capita intake of 20 g of savoury snacks (16th most commonly consumed food item) such as cheese curls and potato chips was also reported (Table 5). Furthermore, the median intakes of added sugar were high in the girls and boys of all ages, contributing a median of 8.3% total energy intake. This is consistent with the top 40 most commonly consumed foods (Table 5) that indicated a per capita intake of 115 g of sugar-sweetened beverages, 5 g of white sugar and 1 g of chocolate. Sugar-sweetened beverages was the second and white sugar the 20th most consumed food item by the respondents. The macronutrient distribution for protein, fat and carbohydrates were all within the AMDR.

The results in Table 5 showed that a mainly carbohydrate-based diet is consumed with small per capita intakes of meat, dairy and vegetables.

A significant positive relationship existed between dietary cholesterol intake and serum cholesterol ( $r=0.165$ ,  $p=0.011$ ) and LDL-C ( $r=0.233$ ,  $p=0.000$ ) respectively, whereas a significant negative correlation was observed between MUFA intakes and triglycerides ( $r=-0.152$ ,  $p=0.021$ ). No other significant correlations were observed between the dietary intake variables and biochemical or nutritional status parameters.

## DISCUSSION

The prevalence of CVDs is increasing significantly in lower-income countries and NCDs are still recognised as the leading cause of death globally (Kelishadi & Poursafa 2014). According to the WHO, NCDs were causing 29% of all deaths in SA in 2008 with 11% due to CVD (WHO 2010). Statistics South Africa, however, reported an even higher proportion of 40% of all deaths being caused by NCDs, with 18% due to CVDs (SSA 2011). NCDs, including CVD, thus have public health consequences in South Africa and a strategic plan was developed to prevent NCDs and promote health and wellness at population and community level, however, most of the strategies focus on adults (DoH 2013). Atherosclerosis was first identified in the youth during the 1990's and subsequently

strong relationships have been found between CVD risk factors such as elevated LDL-C and total serum (TC) cholesterol, as well as low HDL -levels, obesity, hypertension and diabetes mellitus during childhood (HHS 2012). It is also common knowledge that CVD risk factors in childhood will still be present in adulthood, thus creating a cumulative burden on cardiovascular health (Raghuveer 2008; Magnussen et al. 2014). This is one of the first studies to estimate the prevalence of CVD risk factors such as abnormal serum lipids, hyperglycaemia and inflammation and the association thereof with dietary intakes in a group of rural children from a resource-poor community in the Eastern Cape of SA.

As previously mentioned, this study formed part of the baseline survey of a school feeding programme to address under-nutrition, focusing on micronutrient deficiencies and the acute conditions of under-nutrition, namely underweight and wasting. Our results found that 2.3%, 3.4% and 5.6% of the children were underweight, wasted and stunted respectively. These prevalences were lower than the national levels of 5.8%, 3.8% and 15.4% for underweight, wasting and stunting in 0-14 year old children respectively (Shisana et al. 2013). No significant differences in anthropometric indices were observed between the different age groups, however, the boys presented with significantly lower HAZ scores than the girls, thus being more affected by chronic under-nutrition and/or infections (UNICEF 2007). This finding was consistent with the national trend (Shisana et al. 2013). Stunting has recently been associated with an increased risk of CVD, specifically dyslipidaemia, hypertension and glucose intolerance (Kelishadi & Poursafa 2014). This relationship was not confirmed in our results, but may be due to the small number of children that were stunted in this group. There is, however, emerging evidence that stunting is associated with an increased prevalence of risk factors of CVD such as dyslipidaemia, hypertension and glucose intolerance in later life, but these associations are still not well understood and may be the result of the epigenetic responses to increased inflammation and/or poor dietary intakes (Kelishadi & Poursafa 2014).

This study was undertaken in the Intsika Yethu that was identified as one of the eleven poorest municipalities in the Eastern Cape (Chris Hani District Municipality 2011). It has further been established that resource-poor individuals often

have inadequate nutrient intakes (Cunha et al. 2011) which was also evident in the low median energy intakes reported by the children of all age groups in this study. Furthermore, 20.2% of the children had elevated HS-CRP levels, thus the presence of an inflammatory condition. No significant correlations were established between any of the biochemical and anthropometric and most of the dietary intake variables and it can thus not be concluded that the poor dietary intakes or the presence of inflammation could have contributed to stunting.

The prevalence of overweight was 4.3% with 14.2% of the respondents being at risk of overweight. No children were obese. These results found much lower overweight and obesity rates when compared to the SANHANES, that found the prevalence of overweight was 16.5% in boys and 11.5% in girls aged 2-14 years old in South Africa (Shisana et al. 2013). Although the SANHANES found more boys to be overweight than girls (Shisana et al. 2013), this finding was not consistent with our results, however, more girls (18.4%) than boys (10.2%) were at risk of overweight in our study. Findings of a recent meta-analysis confirmed a relationship between overweight in childhood with an increased risk of CVD during adulthood (Friedemann et al. 2012; Juonala et al. 2011). Furthermore, obesity in children has also been linked to a higher prevalence of dyslipidaemia in children (HHS 2012). No significant positive correlation was established between overweight and any of the serum lipid variables among the children in this study and although dyslipidaemia was prevalent, none of the children were obese and the link between obesity and dyslipidaemia could not be confirmed.

The most common dyslipidaemic pattern observed during childhood is obesity with a combined dyslipidaemia pattern expressed as mild increase in TC and LDL-C levels, with a moderate-to-high increase in TG levels and low HDL-C levels (HHS 2012). This was not found in this study when comparing the median biochemical levels. All the biochemical variables showed normal median levels when compared with the reference values, except for the median HDL-C levels which was elevated irrespective of age and gender. The main finding of this study was the high prevalence (42.5%) of low HDL-C levels in both boys and girls in all of the age groups, despite low TC and LDL-C levels. Only 1.3% presented with hypercholesterolaemia and 2.1% and 12.4% had elevated LDL-C and TG

levels respectively. These results were consistent with a study undertaken in 2-14 year old children in Turkey (Toprak et al. 2014), but inconsistent with recent studies undertaken among normal weight adolescents from European countries (Martin et al. 2015) and Kuwait (Boodai et al. 2014) where hypercholesterolaemia and elevated LDL-C levels were more prevalent than low HDL-C. However, the United States National Health and Nutrition Examination Survey (NHANES) of 2011 to 2014 reported that low HDL-C was the most common abnormality found in children aged 6-19 years old. The prevalence of low HDL-C was 13.4%, ranging from 7.7% for 6-8, 10.3% of 9-11, 14% for 12-15 and 18.4% for 16-19 year old United States children respectively. NHANES, however, found 21.0% of the children having hypercholesterolaemia, which was much higher than the prevalence in our study (Nguyen et al. 2014). Furthermore, no significant differences were observed in the median lipid levels between the boys and girls in our study. This was also inconsistent with the United States (Nguyen et al. 2014) findings and those of a study undertaken in Arab adolescents (Al-Daghri et al. 2014) and Turkish children (Toprak et al. 2014) where the girls had significantly higher TC, LDL-C and HDL-C levels than the boys (Al-Daghri et al. 2014; Toprak et al. 2014). A similar finding was observed in the United States (Nguyen et al. 2014). The reasons for these inconsistent findings are not clear. Different living conditions, dietary intake patterns and other factors between the children from the various countries may have explained the inconsistent results and more research should be done to investigate the underlying factors of the dyslipidaemia observed in children. Hypercholesterolaemia was only observed in the 6-8 year old group and this group also had the highest prevalence of abnormal lipid markers, confirming that CVD risk markers are already prevalent at an early age as also found in the United States (Nguyen et al. 2014). Furthermore, these results confirm the findings of other researchers, that although obese children are more at risk for CVD, CVD risk factors may also be prevalent in normal-weight children (Bradshaw et al. 2013; Qorbani et al. 2013). Hyperglycaemia was observed in 10.3% of the children. This is consistent with a recent review article that found that globally, the prevalence of adult type 2 diabetes (T2D) is rapidly increasing in not only adults, but also in adolescents and children. The diagnosis at an earlier age of T2D has significant implications for both the health of youth and the socio-

economic burden on the nation as these youth grow into adults (Amutha & Mohan 2016). No significant correlations between serum glucose levels and any of the biochemical or dietary intake variables were observed, however. These findings might have been useful in designing the intervention before implementation as it seems as if over-nutrition is just as prevalent as under-nutrition in this group of children.

Dietary fat intakes of children and adolescents, both quantity and quality, are not only important for growth and development, but also impact their health in adulthood (Kelishadi et al. 2004). According to the FAO and WHO guidelines for the prevention of chronic disease such as CVD, the total dietary fat, dietary cholesterol, SFA, MUFA, TFA and linoleic acid intakes recorded percentages within the recommended ranges for all the groups (FAO & WHO 2010). This was encouraging as TFA and SFA are known to have the worst effect on serum lipids (Kelishadi & Poursafa 2014). High intakes of SFA are known to raise LDL-levels and raised LDL-levels are positively associated with CVD risk (Raal 2014). The PUFA and linolenic intakes were much lower than the recommended goal of 6-11% and 0.5-2% in all the groups respectively. Sunflower-, soybean and maize oil as well as fatty fish such as mackerel, salmon, herring and trout are the PUFA-rich food sources. Sunflower oil formed part of the top 40 most commonly consumed food items (36th) by the participants whereas none of the other PUFA-rich food sources were consumed. The main food sources of the essential linolenic acid include walnuts, linseed, sunflower and rapeseed oil (AHA 2014). In general, these are the more expensive food items and may not be regularly consumed by resource-poor households as in this study and sunflower oil could thus have been the main contributor to linolenic acid for these participants as a per capita intake of 4 grams was measured. None of the other linolenic acid rich foods were consumed by the participants. A high carbohydrate intake has been associated with hypertriglyceridaemia (Kelishadi et al. 2004). High carbohydrate intakes were evident in our study with intakes of two to almost three times higher than the EAR even though low median energy intakes were reported by boys and girls in all the age groups. The carbohydrate-rich foods (per capita intake) forming part of the top 40 most commonly consumed food items included cooked white rice (195 g) as the most commonly consumed food item, followed by sugar-sweetened beverages (115 g), stiff (74 g) and crumbly (72 g) maize

meal porridge, white bread (66 g), samp (50 g), soft maize meal porridge (37 g), homemade vetkoek (37 g), cooked potatoes (34 g), fermented maize drink (mahewu) (25 g), savoury snacks (20 g), biscuits (19 g) and white sugar (15 g). Furthermore, the median intakes of added sugar were high in the girls and boys, contributing to 8.3% of the total energy intake which was higher than the 7.5% found in rural areas nationally (Maunder et al. 2015). This high intake of added sugar was evident in the top 20 most commonly consumed foods by these children as sugar-sweetened beverages were the second most commonly consumed food item with a per capita intake of 115 g and white sugar 20th with a per capita intake of 15 g. Other sugary foods (per capita intake) forming part of the top 40 most commonly consumed were biscuits (19 g) and hardboiled sweets (7 g) in the 16th and 28th place respectively. These findings were consistent with a national study indicating the top 3 sources of added sugar was white sugar, squash type cold drinks and sugar-sweetened carbonated drinks (Maunder et al. 2015). Increasing sugar intakes is a concern as substantial evidence exists for a relationship between high sugar intakes and micronutrient dilution and the prevalence of non-communicable diseases (Maunder et al. 2015). Kell and co-authors established that added sugars may be associated with triglyceridaemia in children (Kell et al. 2014). Furthermore, the relationship between dyslipidaemia with dietary sodium and fat intakes is well established (Kell et al. 2014). Median sodium intakes among the children in this study were within the recommended guidelines and no significant correlation with the serum lipid variables existed. However, two grams per capita intake of table salt was measured and this may even be higher as the sodium content for the food items in FoodFinder is incomplete. The dietary cholesterol intakes were within the recommended guidelines, a significant positive correlation existed between dietary cholesterol intake with serum cholesterol and LDL-C respectively. Furthermore, a significant negative correlation was observed between MUFA intakes and triglycerides. No significant correlations could be established between any of the other dietary intake and serum lipid variables in this study.

Several limitations should be taken into consideration when interpreting the findings of this paper. Firstly, the purposive selection of the schools can bias the interpretation of the tendencies observed in this study. Furthermore,

although the sample size was calculated to render statistically representative results, the sample of children was drawn from a single area in the Eastern Cape and should, therefore, not be generalised to the rest of SA. Furthermore, some of the dietary intakes are not accurate as FoodFinder has incomplete data for sodium, trans fatty acids and certain nutrient. Besides dietary intakes, environmental factors (family history, physical activity patterns) were not taken into account in the present study to identify factors that may have led to the prevalence of dyslipidemia in these children.

### CONCLUSIONS AND RECOMMENDATIONS

The present study found a prevalence of dyslipidaemia even though most of the dietary fat intake variables were within the recommended intakes. In other studies, dyslipidaemia was associated with obesity, however, in this study dyslipidaemia was found in children with normal weight. Furthermore, about one and two in every ten children were hyperglycaemic and had inflammation respectively. Hyperglycaemia is the pre-cursor for diabetes. Prevention of dyslipidemia and hyperglycaemia in childhood will significantly reduce clinical CVD risk in adulthood. Early identification and screening for dyslipidaemia and hyperglycaemia in childhood are thus recommended as these were present in these apparently healthy children. Modifiable factors of CVD risk emphasize the importance of primary prevention already in early childhood. Healthy lifestyles, including physical exercise, should be promoted from a very young age and nutrition education and awareness programmes implemented with the emphasis on balanced diets based on the food-based dietary guidelines for SA.

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### REFERENCES

AL-DAGHRI, NM, ALJOHANI, NJ, AL-ATTAS, OS, AL-SALEH, Y, WANI, K, ALNAAMI, AM, ALFAWAZ, H, AL-AJLAN, ASM, KUMAR, S, CHOUSOS, GP & ALOKAIL, MS. 2015. Non-high-density lipoprotein cholesterol and other lipid indices vs elevated glucose risk in Arab

adolescents. *Journal of Clinical Lipidology* 9:35-41.

ALWAN A. 2011. Global status report on non-communicable diseases 2010. Geneva:World Health Organization.

AMERICAN ACADEMY OF PEDIATRICS. 1992. National cholesterol education program: highlights of the report of the expert panel on blood cholesterol levels in children and adolescents. *Pediatrics* 89:495-501.

AMERICAN HEART ASSOCIATION (AHA). 2014. Polyunsaturated fats. Available at: [http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyEating/Polyunsaturated-Fats\\_UCM\\_301461\\_Article.jsp](http://www.heart.org/HEARTORG/GettingHealthy/NutritionCenter/HealthyEating/Polyunsaturated-Fats_UCM_301461_Article.jsp). Accessed : 03/06/2015.

AMUTHA, A. & MOHAN, V. 2016. Diabetes complications in childhood and adolescent onset type 2 diabetes – a review. *Journal of Diabetes and its Complications* 30:951-957.

BOODAI, SA, CHERRY, LM, SATTAR, N & REILLY, JJ. 2014. Prevalence of cardiometabolic risk factors and metabolic syndrome in obese Kuwaiti adolescents. *Diabetes, Metabolic Syndrome and Obesity: targets and therapy* 7:505-511.

BRADSHAW, PT, MONDA, KL & STEVENS, J. 2013. Metabolic syndrome in healthy obese, overweight, and normal weight individuals: the atherosclerosis risk in communities study. *Obesity (Silver Spring)* 21(1):203-209.

CHRIS HANI DISTRICT MUNICIPALITY. 2011. Annual report 2010-2011. Cofimvaba:Chris Hani District Municipality.

CUNHA, DB, SICHERI, R, DE, ALMEIDA, RMVR & PEREIRA, RA. 2011. Factors associated with dietary patterns among low-income adults. *Public Health Nutrition* 14 (9):1479-1585.

DEPARTMENT OF HEALTH (DoH). 2013. Strategic plan for the prevention and control of non-communicable diseases 2013-17. Pretoria: Government Publishers.

FOOD AND AGRICULTURAL ORGANIZATION (FAO) and WORLD HEALTH ORGANIZATION (WHO). 2010. Fats and fatty acids in human nutrition. Report of an expert consultation. Geneva Switzerland: FAO.

FRIEDEMANN, C, HENEGHAN, C, MAHTANI, K, THOMPSON, M, PERERA, R, WARD, AM. 2012. Cardiovascular disease risk in healthy children and its association with body mass index: systematic review and meta-analysis. *British Medical Journal* 345:e4759.

GIBSON RS. 2005. Principles of nutritional assessment. 2nd ed. New York: Oxford University Press.

HEALTH PROFESSIONS COUNCIL OF

- SOUTH AFRICA (HPCSA). 2008. Guidelines for good practice in the health care professions seeking patients' informed consent: the ethical considerations. Booklet 9. Pretoria: HPCSA. Available at: [http://www.hpcsa.co.za/downloads/conduct\\_ethics/rules/generic\\_ethical\\_rules/booklet\\_9\\_informed\\_consent.pdf](http://www.hpcsa.co.za/downloads/conduct_ethics/rules/generic_ethical_rules/booklet_9_informed_consent.pdf). Accessed: 12/07/2016.
- HODSON, LA, MURRAY, SKEAFF, C & FIELDING, B. 2008. Fatty acid composition of adipose tissue and blood in humans and its use as a biomarker of dietary intake. *Progressive lipid research* 47:348-380.
- INSTITUTE OF MEDICINE (IoM). 2000. Dietary reference intakes: applications in dietary assessment. Food and Nutrition Board. Washington DC: National Academy Press.
- INSTITUTE OF MEDICINE (IoM). 2002. Dietary reference intakes for energy, carbohydrate, fibre, fat, fatty acids, cholesterol, and protein and amino acids. Food and Nutrition Board. Washington DC: National Academy Press.
- INTSIKA YETHU LOCAL MUNICIPALITY. 2012. Annual report 2011/2012. Intsika Yethu: Intsika Yethu Local Municipality.
- JUONALA, M, MAGNUSSEN, CG, BERENSON, GS, VENN, A, BURNS, TL, SABIN, MA, SRINIVASAN, SR, DANIELS, SR, DAVIS, PH, CHEN, W, SUN, C, CHEUNG, M, VIKARI, JSA, DWYER, T & RAITAKARI, OT. 2011. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *New England Journal of Medicine* 365(20):1876-1885.
- KELISHADI, R, POUR, MH, ZADEGAN, NS, KAHBAZI, M, SADRY, G, AMANI, A, ANSARI, R, ALIKHASSY, H & BASHARDOUST, N. 2004. Dietary fat intake and lipid profiles of Iranian adolescents: Isfahan Healthy Heart Program – heart health promotion from childhood. *Preventive Medicine* 39:760-766.
- KELISHADI, R & POURSAFA, P. 2014. A review of the genetic, environmental, and lifestyle aspects of the early-life origins of cardiovascular disease. *Current Problems in Pediatric and Adolescent Health Care* 44:54-72.
- KELL, KP, CARDEL, MI, BOHAN BROWN MM & FERNÁNDEZ JR. 2014. Added sugars in the diet are positively associated with diastolic blood pressure and triglycerides in children. *The American Journal of Clinical Nutrition*, 100(1): 46-52.
- LANGENHOVEN, ML, KRUGER, ML, GOUWS, E & FABER, M. 1991. Food composition tables. Parow: Medical Research Council.
- LOHMAN, TG, ROCHE, AF & MARTORELL, M. 1988. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics.
- MAGNUSSEN, CG, SMITH, KJ & JUONALA, M. 2014. What the long term cohort studies that began in childhood have taught us about the origins of coronary heart disease. *Current Cardiovascular Risk Report* 8:373-385.
- MARTIN, L, OEPEN, J, REINEHR, T, WABITSCH, M, GLAUSSNITZER, G, WALDECK, E, INGRISCH, S, STACHOW R, OELERT, M, WIEGAND, S & HOLLE, R. 2015. Ethnicity and cardiovascular risk factors: evaluation of 40 921 normal-weight, overweight or obese children and adolescents living in Central Europe. *International Journal of Obesity* 39: 5-51.
- MAUNDER, EMW, NEL, JH, STEYN, NP, KRUGER, HS & LABADARIOS, D. 2015. Added sugar, macro- and micronutrient intakes and anthropometry of children in a developing world context. *PLOS One* 10(11):e0142059. Doi:10.1371/journal.pone.0142059.
- NGUYEN, D, KIT, B & CARROLL, M. 2014. Abnormal cholesterol among children and adolescents in the United States, 2011-2014. NCHS Data Brief 228. Washington DC: Centers for Disease Control and Prevention and National Center for Health Statistics.
- OCHOA-AVILÉS, A, VERSTRAETEN, R, LACHAT, C, ANDRADE, S, VAN CAMP, J, DONOSO, S & KOLSTEREN, P. 2014. Dietary intake practices associated with cardiovascular risk in urban and rural Ecuadorian adolescents: a cross-sectional study. *BMC Public Health* 14:939.
- OLDEWAGE-THERON, WH, DICKS, EG & NAPIER, CE. 2006. Poverty, household food insecurity and nutrition: coping strategies in an informal settlement in the Vaal Triangle, SA. *Public Health* 120(9):795-804.
- PARK, C, GUALLAR, E, LINTON, JA, LEE, DC, JANG, Y, SON, DK, HAN, EJ, BAEK, SJ, YUN, YD, JEE, SH & SAMET, JM. 2013. Fasting glucose level and the risk of incident atherosclerotic cardiovascular diseases. *Diabetes Care* (36): 1988-1993.
- POSADAS-SÁNCHEZ, R, POSADA-ROMERO, C, ZAMORA-GONZÁLEZ, J, MENDOZA-PÉREZ, E, CARDOSO-SALDAÑA, G & YAMAMOTO-KIMURA, L. 2007. Lipid and lipoprotein profiles and prevalence of dyslipidemia in Mexican adolescents. *Metabolism Clinical and Experimental* 56:1666-1672.
- QORBANI, M, KELISHADI, R, FARROKHI-KAJEH-PASHA, Y, MOTLAGH, M, AMINAE, T, ARDALAN, G, ASAYESH, H, SHAFIEE, G, TASLIMI, M, POURSAFA, P, HESHMAT, R & LARIJANI, B. 2013. Association of anthropometric measures with cardiovascular risk factors and metabolic syndrome in normal-

- weight children and adolescents: the CASPIAN III study. *Obesity Facts* 6(5):483-492.
- RAAL FJ. 2014. The cardioprotective diet: carbohydrates versus fat. *Cardiovascular Journal of Africa* 25(6):302.
- RAGHUVEER G. 2008. Assessment of atherosclerotic cardiovascular risk and management of dyslipidemia in obese children. *Progress in Pediatric cardiology* 25:167-176.
- RIETZELL E & DE BUYZERE M. 2012. Biomarkers in medicine. *Future Medicine* 6 (1):19-34.
- SENEKAL, M, NAUDE, C & WENTZEL-VILJOEN, E. The Nutrition Society of South Africa fact sheet: Dietary recommendations for health. Available: [www.nutritionociety.co.za/index.php/useful-information/37-fact-sheet-dietary-recommendations-for-health](http://www.nutritionociety.co.za/index.php/useful-information/37-fact-sheet-dietary-recommendations-for-health). Accessed: 12/07/2016.
- SHISANA, O, LABADARIOS, R, REHLE, T, SIMBAYI, L, ZUMA, K, DHANSAY, A, REDDY, P, PARKER, W, HOOSAIN, E, NAIDOO, P, HONGORO, C, MCHIZA, Z, STEYN, NP, DWANE, N, MAKORAE, M, MALULEKE, T, RAMLAGAN, S, ZUNGU, N, EVANS, MG, JACOBS, L, FABER, M & SANHANES-1 TEAM. 2013. South African national health and nutrition examination survey (SANHANES-1). Cape Town: HSRC Press.
- SOUTH AFRICAN HEART ASSOCIATION (SA HEART) AND THE LIPID AND ATHEROSCLEROSIS SOCIETY OF SOUTHERN AFRICA (LASSA). 2012. South African dyslipidaemia guideline consensus statement. *South African Medical Journal* 102 (3):178-188.
- STATISTICS SOUTH AFRICA (SSA). 2011. Mortality and causes of death in South Africa, 2008: Findings from death notification. Pretoria: SSA.
- THE SURVEY SYSTEM. SAMPLE SIZE CALCULATOR. Available at: [www.surveysystem.com/sscalc.htm](http://www.surveysystem.com/sscalc.htm). Accessed: 31/03/2013.
- TOPRAK, D, BUKULMEZ, A, DOGAN, N, OZTEKIN, O & KOKEN, T. 2014. Evaluation of serum lipid profiles in Turkish children aged two to eighteen years. *West Indian Medical Journal* 63(6):588.
- UNITED NATIONS CHILDREN'S FUND (UNICEF). 2007. Progress for respondents. A world fit for children - statistical review. Number 6. New York: UNICEF.
- UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES (HHS), NATIONAL INSTITUTES OF HEALTH, NATIONAL HEART, LUNG AND BLOOD INSTITUTE. EXPERT PANEL ON INTEGRATED GUIDELINES FOR CARDIOVASCULAR HEALTH AND RISK REDUCTION IN CHILDREN AND ADOLESCENTS. 2012. "Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report." NIH Publication No.12-7486. Washington DC: US Department of Health and Human Services.
- WARNICK, GR, KNOPP, RH, FITZPATRICK, V & BRANSON, R. 1990. Estimating low-density lipoprotein cholesterol by the Friedewald equation is adequate for classifying patients on the basis of nationally recommended cut points. *Clinical Chemistry* 36:15-19.
- WILLERSON, JT & RIDKER, PM. 2004. Inflammation as a cardiovascular risk factor. *Circulation* 109:II-2-II-10. Available at: [http://circ.ahajournals.org/content/109/21\\_suppl\\_II/II-2](http://circ.ahajournals.org/content/109/21_suppl_II/II-2).
- WORLD HEALTH ORGANIZATION (WHO). Anthro plus version 1.0.2. Available at: [www.who.iny/growthref](http://www.who.iny/growthref). Accessed: 02/02/2013.
- WORLD HEALTH ORGANIZATION (WHO). 2006. Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia: a report of the WHO/IDF consultation. Geneva:WHO.
- WORLD HEALTH ORGANIZATION (WHO). 2007. The WHO child growth standards. Available at: <http://www.who.int/childgrowth/standards/en/>. Accessed: 02/02/2013.
- WORLD HEALTH ORGANIZATION (WHO). 2010. Global status report on NCDs. Geneva:WHO.
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