Hypertriglyceridemic Waist Phenotype and Associated Lifestyle Factors in a National Population of Youths: CASPIAN Study

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Summary

The objectives of the current study, that is the first of its kind, were to determine the prevalence of the hypertriglyceridemic waist (HW) phenotype in a nationally representative sample of children, as well as the metabolic risk factors identified by HW, and to identify lifestyle habits related to this phenotype. This national survey was conducted on 4811 representative school-students. We assessed the sensitivity and specificity of the HW phenotype for abnormal anthropometric and biochemical factors by using receiver operator characteristic curves. We determined the association of dietary patterns (obtained by factor analysis), physical activity level and some environmental factors with the HW phenotype. Overall, 8.52% of participants had the HW phenotype. Those children with the HW phenotype were more likely to have cardiovascular risk factors, notably for overweight and hypercholesterolemia. The dietary pattern characterized by junk foods increased the odds of having the HW phenotype, OR = 1.426 (95%CI, 1.109, 1.892), whereas the other dietary pattern including healthy foods decreased this odds, OR = 0.874 (95%CI, 0.765, 0.998). The risk of the HW phenotype rose with the consumption of solid hydrogenated fat as well as white-flour bread. Low education of parents and a positive family history of diabetes mellitus, obesity and or premature cardiovascular disease were the other risk factors for the HW phenotype. Low levels of physical activity significantly increased the risk of having the HW phenotype. The HW phenotype can be used as an accurate and easy tool for screening children at metabolic risk in population-based studies.

Key words: Hypertriglyceridemia, abdominal obesity, metabolic risk factors, population-based survey, determinants, lifestyle habits.

Introduction

There are multiple lines of evidence with regard to the strong association between the presence and extent of the risk factors of chronic diseases in childhood and adulthood [1]. Biological risk factors tend to occur more frequently together than expected by chance; a particular phenotype of such clustering is defined as the metabolic syndrome that is associated with various disorders as non-alcoholic fatty liver disease in childhood and chronic diseases in adulthood [2], currently this syndrome has several definitions [3]. Many researchers tried to substitute simple tools for screening of the metabolic

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abnormalities. In order to simplify the clinical practice, at least two parameters should be screened for: One of the most frequent simple inexpensive couples is the measurement of waist circumference (WC) and the other is the fasting plasma triglycerides (TG), or in other words hypertriglyceridemic waist (HW), that might be able to identify the diagnostic triad of the MetS both in adults and in adolescents [4, 5]. Some lifestyle behaviours are associated with metabolic abnormalities in adults [6, 7], but such data is lacking for those youths with HW phenotype.

The objectives of the current study, that is the first of its kind, were to determine the prevalence of the HW phenotype in a nationally representative sample of children and adolescents aged 6–18 years and living in urban and rural areas, as well as the metabolic risk factors identified by this phenotype. In addition, we aimed to identify lifestyle habits related to the HW phenotype among youths.

Methods

The baseline survey of this ongoing national study, entitled *''Childhood & Adolescence Surveillance and Prevention of Adult Non-communicable Disease'':* **CASPIAN** (Caspian is the name of the world's largest lake, located in Northern Iran.) *Study*, was conducted in 2003–2004 in Iran. We have previously described the methods of this study in detail [8, 9], and here we report it in brief. The study population comprised of 4811 school students. Ethics committees and other relevant national regulatory organizations approved the study. The Data & Safety Monitoring Board of the project closely supervised the quality control and quality assurance of the survey at the national level. We obtained written informed consent from parents and oral assent from students. The project team selected students by multistage-random cluster sampling.

We prepared our questionnaires based on the questionnaires of the World Health Organization (WHO) STEPwise approach to non-communicable diseases (Tools ver 9.5) and the WHO Global school-based student health survey (GSHS). In addition, students filled a validated food frequency questionnaire (FFQ), and food items were grouped for analysis [8]. Physical activity was assessed by a scaled questionnaire organized in nine different metabolic equivalent (MET) levels [8–10], we categorized its levels to the tertials.

Physical examination was conducted according to standard protocols [11, 12]. Fasting venous blood was collected, and fasting blood sugar (FBS), total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C) [13] and triglycerides (TG) were measured enzymatically by auto-analysers. Low-density lipoprotein-cholesterol (LDL-C) was calculated in serum samples with $TG \leq 400$ mg/dl according to the Friedewald equation [14]

We defined risk factors according to reference values [12, 15–21]. The prevalence of risk factors in children with and without the HW phenotype was compared between boys and girls by using the Chi-square test. Logistic regression analysis was used for assessment of the age-adjusted odds ratios (95% CIs) for risk factors across participants with the HW phenotype, and for evaluating the age-adjusted associations of the HW phenotype with possible factors.

We used receiver operator characteristic (ROC) curve analyses [22] to calculate the optimal cut-off values, sensitivity and specificity for the HW phenotype and the area under the receiver operating characteristic curve (AUC) for the abnormal anthropometric and biochemical factors, and to determine abnormal lipid levels and anthropometric indices and the HW phenotype by gender and age groups.

Dietary patterns were obtained by exploratory factor analysis of the food groups, and their adjustment was verified using the Kaiser–Meyer–Olkin (KMO) measurement of sample adequacy. Principal component analysis was used for factor extraction. The choice of the number of factors was based on the Kaiser criterion, namely eigenvalues over 1.0, and also on the eigenvalue plot (scree plot), factor loadings were analysed by the Varimax method [23].

Results

Overall, 2248 boys and 2563 girls, with a mean age of 12.07 ± 3.2 years were studied. Most children (72.1%) were breastfed during infancy, and their weaning food was home-made (71.7%). The most frequent type of fat consumed was hydrogenated solid fat (78.2%). Most students (59.2%) consumed breads prepared with white wheat flour. The birth weights of <2500 g and >4000 g were reported in, respectively, 14.3% and 10.1% of participants.

Table 1 presents the prevalence of abnormal biologic and biochemical factors. The highest and lowest prevalence of HW phenotype were, respectively, found in urban girls (9.7%) and rural boys (6.42%).

The prevalence of abnormal factors was higher in those participants with the HW phenotype (Fig. 1), and these children were more likely to have CVD risk factors; the highest OR was documented for overweight and hypercholesterolemia, respectively (Table 2).

Overall, after hypertriglyceridemia, the AUC was largest for hypercholesterolemia, and low HDL-C had the lowest AUC (Fig. 2). All anthropometric indices had nearly similar AUC, except than the waist-to-hip ratio (WHR) that had the lowest AUC (Fig. 3).

In the factor analysis of participants' dietary patterns, the KMO measure of sampling adequacy

TABLE 1
Prevalence of abnormal biologic and biochemical factors by gender, age group and living area: CASPIAN Study

	Boys ($n=2248$)			Girls ($n=2563$)			Total ($n=4811$)		
	Urban (94.5%)	Rural (5.5%)	Total	Urban (93.84%)	Rural (6.16%)	Total	Urban (94.1%)	Rural (5.9%)	Total
6–9.9 years ($n=1616$)									
High LDL-C	11.54	8.53	8.65	13.88	6.06	13.55	11.42	8.47	11.30
Low HDL-C	27.13	7.41	26.32	25.47	8.82	24.74	26.23	8.20	25.47
High TC	7.41	6.31	6.35	8.97	8.82	8.96	8.20	7.75	7.77
High TG	16.25	14.81	16.19	26.47	15.80	16.26	21.31	16.00	16.23
High FBS	1.8	0.4	1.1	1.8	0.2	1.0	1.2	0.1	1.1
High SBP/DBP	5.7	4.8	5.1	7.7	6.4	7.1	9.3	8.9	9.1
High BMI	15.88	12.17	15.27	15.49	13.90	15.28	15.68	12.93	15.28
High WC	28.22	25.29	27.74	28.43	27.80	28.35	28.33	26.39	28.04
HW	7.41	6.96	6.98	14.71	6.84	7.18	11.48	6.89	7.09
10–13.9 years ($n=1887$)									
High LDL-C	9.03	7.27	8.91	18.46	6.56	7.35	13.33	7.68	8.06
Low HDL-C	23.50	14.55	22.90	30.10	16.92	29.23	27.09	15.83	26.34
High TC	12.73	6.52	6.93	13.85	5.56	6.11	13.33	6.00	6.49
High TG	23.64	19.17	19.46	36.92	23.88	24.75	30.83	21.73	22.34
High FBS	5.3	3.8	5.0	4.4	3.1	4.1	4.5	1.2	4.1
High SBP/DBP	6.8	5.2	6.3	4.5	2.9	4.1	5.4	4.1	5.2
High BMI	16.75	7.38	15.34	15.50	12.78	15.07	16.15	10.05	15.21
High WC	29.72	15.27	27.56	28.40	20.67	27.16	29.09	17.94	27.37
HW	10.08	9.09	10.01	10.77	9.45	9.54	10.00	9.74	9.75
14–18 years ($n=1308$)									
High LDL-C	4.05	3.70	4.04	5.57	5.45	5.56	4.88	4.83	4.84
Low HDL-C	29.37	26.01	28.10	23.32	7.27	21.92	28.05	26.62	24.86
High TC	2.93	0.9	2.79	7.27	5.16	5.35	4.88	4.08	4.12
High TG	21.76	11.11	21.25	25.45	18.76	19.34	20.73	20.21	20.25
High FBS	4.3	2.7	4.2	3.4	1.7	3.1	3.8	0.2	3.2
High SBP/DBP	8.2	7.7	8.1	3.4	2.8	3.2	9.1	6.8	8.1
High BMI	15.52	14.09	15.27	15.38	12.33	14.93	15.45	13.26	15.09
High WC	28.58	21.27	27.30	29.36	16.30	27.41	28.99	18.91	27.35
HW	8.49	0.7	8.08	8.98	5.45	8.68	8.74	3.66	8.40
Total ($n=4811$)									
High LDL-C	7.47	7.41	7.46	11.11	8.74	8.89	9.58	8.15	8.23
Low HDL-C	26.61	25.39	25.45	26.81	11.69	25.83	26.15	17.87	25.66
High TC	8.26	5.44	5.59	10.39	6.59	6.84	9.51	7.06	6.26
High TG	18.94	18.35	18.91	30.52	21.87	20.55	25.48	21.44	22.79
High FBS	5.4	3.9	5.1	3.5	1.3	3.0	4.1	0.9	4.1
High SBP/DBP	7.4	6.9	7.2	5.7	5.2	5.4	8.7	7.1	7.4
High BMI	16.12	11.04	15.30	15.46	12.96	15.09	15.79	11.94	15.20
High WC	28.92	20.41	27.54	28.71	21.32	27.62	28.81	20.84	27.58
HW	8.62	6.42	8.50	9.74	8.46	8.54	8.53	8.37	8.52

Note: High TC: levels of >200 mg/dl [16]; high LDL-C: levels of ≥130 mg/dl [16]; high TG: levels of ≥125 mg/dl [16]; low HDL-C: levels of <35 mg/dl [16]; high FBS: levels of ≥100 mg/dl [17]; high BMI: 85th percentile [15]; high WC: levels of >75th percentile [18–21]; high blood pressure: levels of systolic (SBP) and/or diastolic (DBP) blood pressure >90th percentile [12]; HW: hypertriglyceridemic-waist.

was 0.856 that meant that the sample was considered to be adequate for factor analysis. Two dietary patterns were identified through factor analysis, based on the Kaiser criterion and the scree plot (Fig. 4). These two patterns accounted for 52.2% of the variability within the sample. Table 3 shows the factor loadings after varimax rotation. The first factor that accounted for 28.04% of the total variance included fast foods, carbohydrates, salty/fat snacks and sweets/candies. Vegetables, fruits and dairy product groups were negatively associated to this factor. The second factor explained 24.1% of the total variance, and was characterized by dairy products, plant protein, vegetables and fruits. Sweets/candies and fast foods had negative association with this factor.

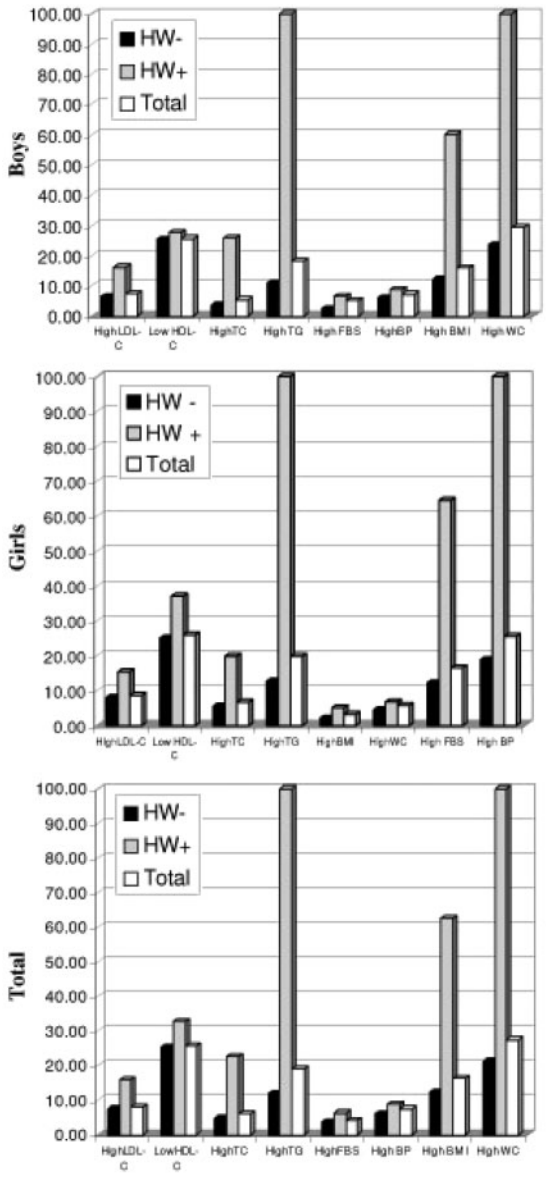

FIG. 1. Prevalence of risk factors in children with and without the hypertriglyceridemic waist phenotype: CASPIAN study.

The unhealthy dietary pattern increased the odds of having the HW phenotype, OR = 1.426 (95%CI, 1.109, 1.892), $p = 0.03$. Whereas the other dietary pattern decreased this odds OR = 0.874 (95%CI, 0.765, 0.998), $p = 0.04$.

Age-adjusted logistic regression analysis of factors associated with the HW phenotype showed that a birth weight of >4000 g in boys and <2500 g in girls increased the risk of having the HW phenotype [OR, 95%CI: 1.4(1.1, 2.05), 1.2 (1.09, 1.7), respectively]. Low education of parents and a positive family history of diabetes mellitus, obesity and or premature CVD were the other risk factors for the HW phenotype in both genders. Low levels of physical activity significantly increased the risk of having the HW phenotype [in boys: 1.4 (1.2, 2.5) and in girls: 1.3 (1.1, 1.9)].

The risk of the HW phenotype rose with the consumption of solid hydrogenated fat [in boys: 1.5 (1.1, 1.7) and in girls: 1.4 (1.1, 1.8)] as well as white-flour bread [in boys: 1.5 (1.1, 2.7) and in girls: 1.4 (1.1, 2.5)].

Discussion

We found that the HW phenotype was present in 8.5% of Iranian children, and was detected in children as young as 6 years of age. The prevalence of the HW was not significantly different in terms of gender and living area; however the highest and lowest prevalence was documented among rural boys and urban girls, respectively. Our findings suggest that the HW phenotype can be used as an accurate and simple screening tool for identifying those children and adolescents who might be at metabolic risk. In addition, our study revealed that birth weight, family history of some chronic diseases, education level of parents, dietary and physical activity habits had diverse effects on the risk of having the HW phenotype.

Although limited experience exists on the usefulness of HW phenotype in identifying those youth at risk of the metabolic abnormalities, it is documented that among adults, the triad of high apolipoprotein B, hyperinsulinemia and high serum small-dense LDL-C that is significantly associated with CVD is predictable by the HW phenotype [4]. Those adults with HW phenotype are at higher risk to have angiographically defined CVD [24, 25]. Overall, this phenotype is considered as a simple tool for prediction of the CVD risk clustering in adult populations of different ethnicities [4, 26–27].

The findings of the current study complement some recent observations of the usefulness of WC and WSR in predicting metabolic risk factors among children of different ethnicities [21, 28], and emphasize on the importance of routine measurement of WC in the pediatric population. In addition, it is suggested that in population-based studies, the two measures of WC and TG can be used instead of difficult and expensive indicators for identifying children at risk of metabolic disorders.

Although some genes are found to be attributable to HW phenotype [29], environmental factors might influence these programmed effects. Of special interest in the context of our findings is that healthy

TABLE 2
Age-adjusted odds ratios (95%CIs) for risk factors across participants with the hypertriglyceridemic waist phenotype: CASPIAN Study

	Boys		Girls	
	Odds ratio (95% CI)	*p*	Odds ratio (95% CI)	*p*
High LDL-C	2.81 (1.80,4.41)	<0.0001	2.25 (1.48,3.40)	<0.0001
Low HDL-C	1.21 (1.09,1.57)	0.04	1.78 (1.32,2.41)	<0.0001
High TC	9.37 (6.23,14.09)	<0.0001	4.41 (2.99,6.51)	<0.0001
High FBS	1.22 (1.04,2,8)	0.02	1.31 (1.14,2.85)	0.04
High SBP/DBP	1.54 (1.21,2.93)	0.04	1.24 (1.04,2.91)	0.03
High BMI	10.99 (7.92,15.26)	<0.0001	12.98 (9.50,17.73)	<0.0001

dietary habits decreased the risk of having the HW phenotype, and unhealthy dietary habits characterized mostly by junk foods increased this risk. Usually, the largest portion of Iranian meals consists of bread and or rice. As indicated in the current study, the popular habit of eating white-flour bread increased the risk of having the HW phenotype. Furthermore, most families consumed hydrogenated solid fat, rich in saturated and trans fatty acids [30], that raised the probability of having the HW phenotype; similarly to its effects on dyslipidemia and high BP of youths [31, 32].

Low levels of physical activity significantly increased the risk of having the HW. Although no previous study has evaluated this association, but it is documented that low physical activity is related to clustering of risk factors in the normal children [7, 33].

It is now well established that the origins of many chronic diseases trace back to the foetal period [34]. Underlying genetic tendency or early-life adverse events may contribute to the high prevalence of chronic disease and their risk factors in developing countries [35]. Birth weight reflects the pattern of intrauterine growth, and might have long-term impacts on chronic diseases [36, 37]. In the current study, the history of high and low birth weight increased the risk of having the HW phenotype, this is consistent with the findings of some cohort studies on the components of the metabolic syndrome [38, 39]. In addition to this U-shaped relationship, post-natal weight gain should be considered, as well [40] Future longitudinal research is needed in this regard.

Although our previous studies in the same population confirmed the protective role of breast feeding on overweight [6] and high BP [32], this association was not confirmed with the HW phenotype. As supposed previously, the association between breast feeding and some risk factors, but not with their cluster may be because childhood represents a transition period between the infancy and the adult effects of breast feeding [41].

There is growing evidence that childhood socioeconomic position can influence chronic diseases in adulthood [42]. Education level, as an indicator of socioeconomic position, is reported to be negatively correlated with the relative risk of the metabolic syndrome among adults [43]. In the current study, lower education of parents increased the odds of having the HW phenotype in children. The lower education level of parents seems to be associated with the adoption of unhealthy lifestyle habits among family members.

A positive family history for chronic diseases is related to the metabolic syndrome [44, 45]. In the current study, while a positive parental history of obesity and or premature CVD increased the risk of having the HW phenotype, a positive history of diabetes either in parents or in second relatives, such as grandparents, uncles and or aunts, increased the odds of having the HW in the youths. This emphasizes on the necessity of high-risk approach for prevention of chronic disease among.

Study strengths and limitations: Our study has three novelties: (i) it is the first population-based study about HW phenotype conducted in a nationally representative sample of youths living in urban and rural areas, the previous study [5] included only the urban area of one district in a metropolitan population; (ii) our study population comprises those children as young as 6 years, the previous study [5] has been conducted among adolescents; and (iii) there is no report in the current literature regarding the association of lifestyle factors and the HW phenotype in the pediatric population.

One of the limitations of the current study is the presumptions on the recall bias for the process of recalling and recording the food intake and the physical activity habits. Considering the large number of participants, only a quantitative FFQ was used in the present survey and the pubertal stages were not examined. We cannot infer causality because of the cross-sectional nature of the associations.

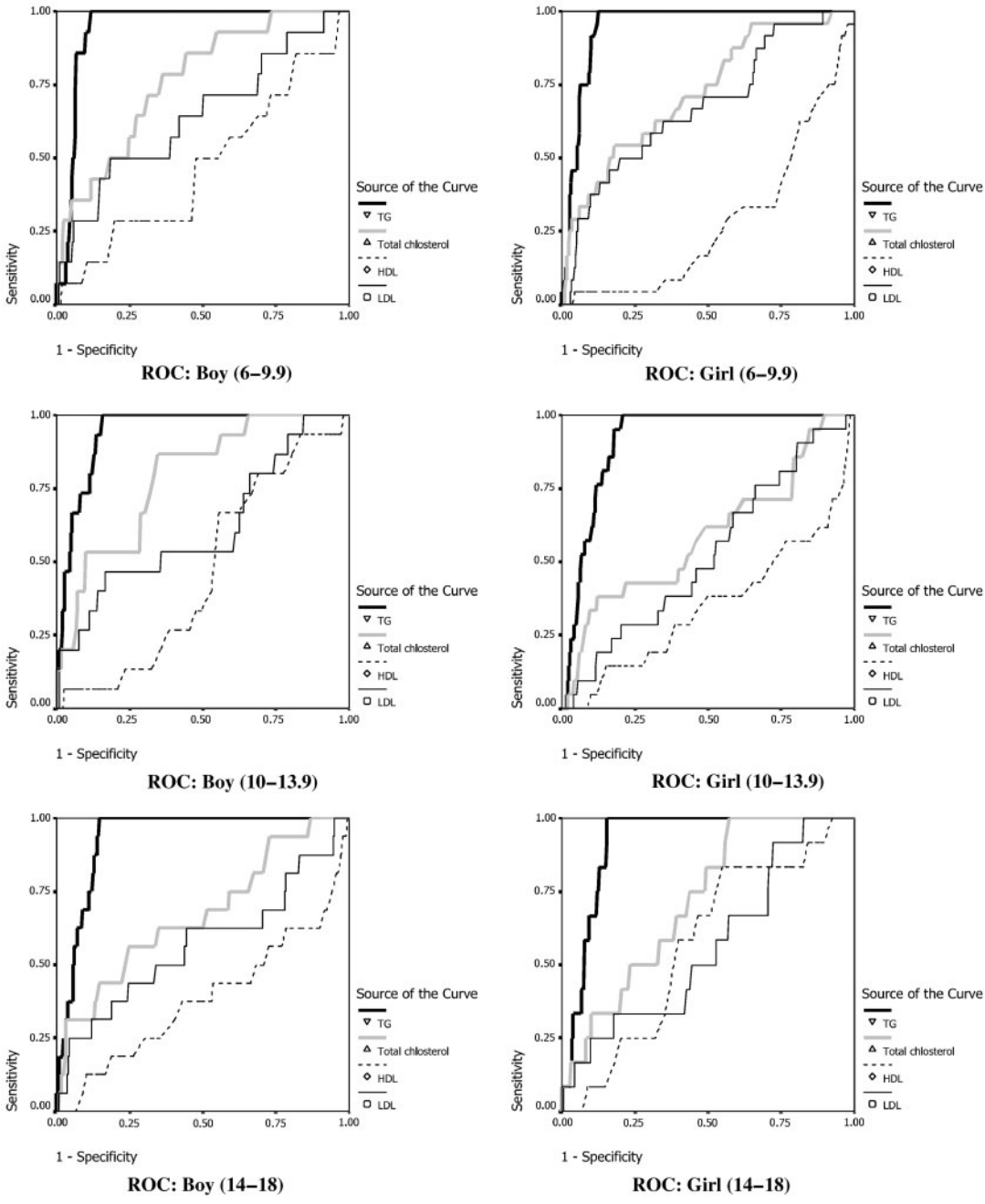

FIG. 2. ROC curves for abnormal lipid levels and the hypertriglyceridemic waist phenotype according to gender and age group: CASPIAN study.

The HW can be used as an accurate, easy and non-expensive tool for screening children at metabolic risk in population-based studies. Considering the effect of modifiable factors on the HW phenotype and its late consequences, public health approaches should be directed toward primordial and primary prevention of chronic diseases.

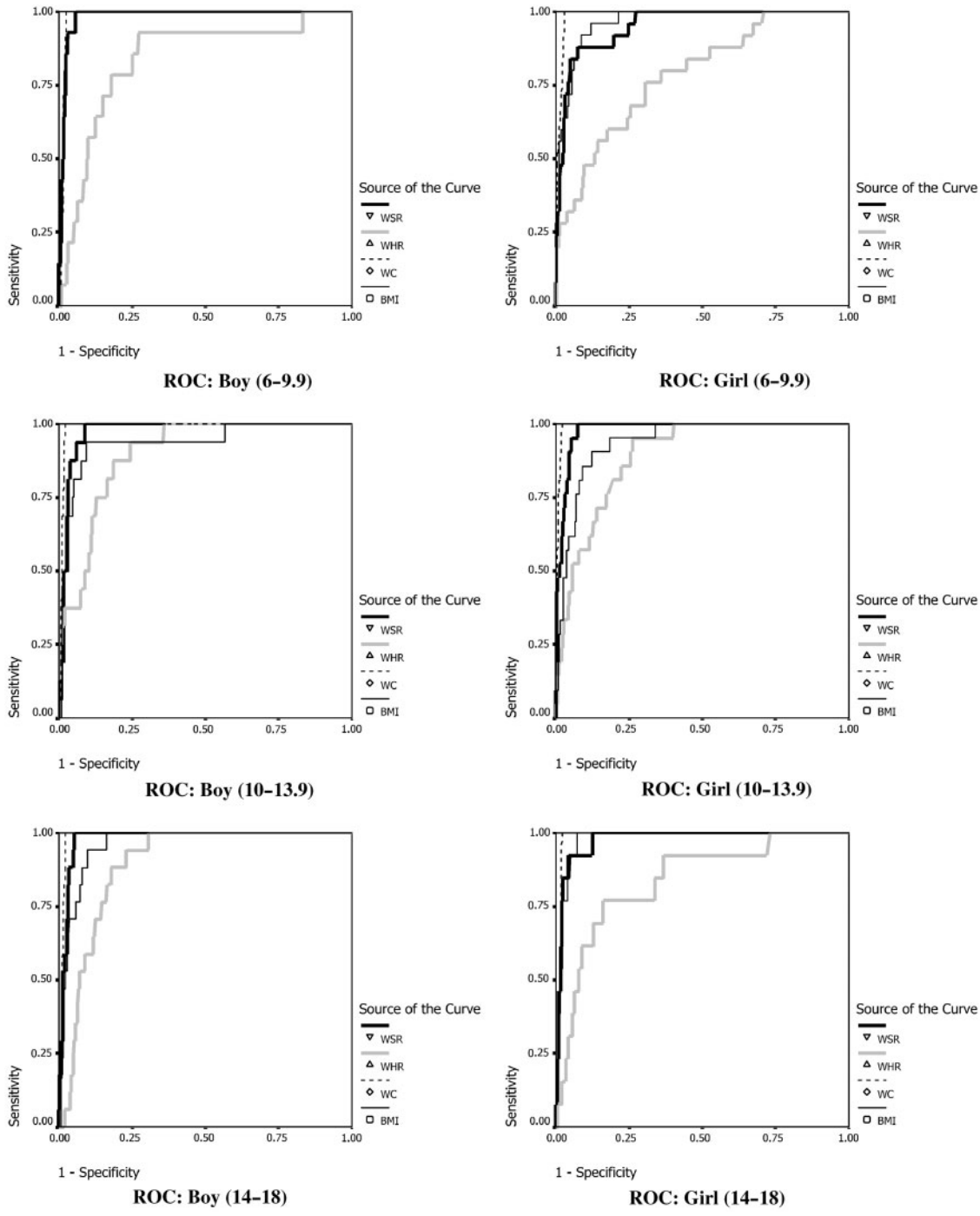

FIG. 3. ROC curves for anthropometric indices and the hypertriglyceridemic waist phenotype according to gender and age group: CASPIAN study.

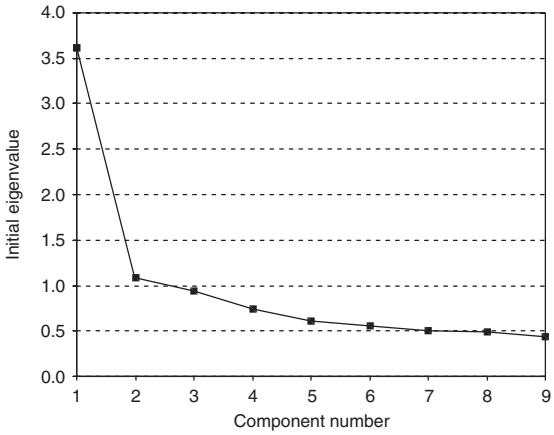

FIG. 4. Scree plot showing eigenvalues for each component in factor extraction of data obtained from a food frequency questionnaire: CASPIAN study.

TABLE 3
Food group factor loading for two dietary patterns identified by factor analysis after Varimax rotation of principal components analysis: CASPIAN study

Food group	Dietary patterns	
	Pattern 1	Pattern 2
Dairy products	−0.296	0.643
Animal protein	0.633	0.321
Plant protein	0.450	0.559
Fast foods	0.774	−0.169
Salty/fat snacks	0.657	0.142
Sweets/candies	0.526	−0.425
Vegetables	−0.340	0.612
Fruits	−0.406	0.624
Carbohydrate foods	0.775	0.148
% Explained variance	28.040	24.184
Cumulative%	28.040	52.225

Note: Bold values represent variables with a factor loading. Carbohydrate foods: rice, bread, pasta, potato; vegetables: fresh/frozen vegetables but potato and French fries not included; fruits: fresh, dried, juice; dairy products: milk, cheese, yogurt; animal protein: red meat, poultry, fish, egg; plant protein: beans, soy, nuts; fast foods:pizza, hamburgers, sausages; snacks: salty/fat potato chips and cheese puffs;sweet/candies: cake, biscuits, candies, cookies.

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