# RELATIONSHIP BETWEEN PARENTAL AND CHILD CARDIOVASCULAR RISK FACTORS

A Fesharak Nia MD<sup>(1)</sup>, A Zarban Ph.D. <sup>(2)</sup>, T Kazemi MD<sup>(3)</sup> Gh R Sharifzadeh M.Sc. <sup>(4)</sup>

#### **Abstract**

INTRODUCTION: Adult cardiovascular disease has its root in childhood. Cardiovascular disease aggregates in families, so determination of high-risk families and early screening and control of cardiovascular risk factors in offspring will help in efforts to prevent cardiovascular disease. This study was performed to determine the relationship between cardiovascular risk factors in parents with a positive history of premature myocardial infarction and their offspring.

METHODS: This cross-sectional study was conducted in 2004 on 91 parents and their offspring (91children). The parents were randomly selected from among patients hospitalized in the critical care unit of Vali-e-Asr hospital with premature myocardial infarction. Important indicators such as systolic blood pressure (SBP), diastolic blood pressure (DBP), body mass index (BMI), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) were measured in both groups.

RESULTS: There was no significant relation of systolic and diastolic blood pressure between parents and their offspring. Thirty-three percent of the parents had hypertension. No cases of hypertension were found in children. Mean systolic and diastolic blood pressure were significantly higher in the children of hypertensive parents. Significant relations were seen between BMI and obesity in parents and their children. There was no significant relation between serum lipids, high TC, high LDL-C and low HDL-C levels in parents and their children. The commonest lipid disorder in parents and their offspring was low HDL-C.

CONCLUSIONS: The results of this study show a significant relation between hypertension, obesity and blood lipid disorders between parents with positive history of premature myocardial infraction and their children. Hence, screening programs in these children for detection of cardiovascular risk factors are recommended.

**Keywords:** Cardiovascular risk factors, parental, relationships, offspring, premature myocardial infarction.

ARYA Journal, 2006, 2(2): 97-101

## Introduction

Adult cardiovascular disease (CVD) has its root in childhood.<sup>1</sup> Although CVD does not manifest itself until adulthood, its risk factors such as elevated blood pressure, excess weight and abnormalities in plasma lipid levels are present in childhood and persist into adulthood.<sup>2</sup>

Prospective studies have shown that cardiovascular disease aggregates in families.<sup>3,4</sup> This is probably due in part to familial aggregation of important cardiovascular risk factors such as hypertension,<sup>5,6</sup> obesity<sup>7</sup> and high total serum cholesterol (TC) and

low-density lipoprotein cholesterol (LDL-C).8 Therefore, early detection and control of these risk factors in childhood may help in efforts to prevent cardiovascular disease, especially in high risk families.9 The relationship between cardiovascular risk factors in parents and their children differs by ethnicity.10 This study was performed in the city of Birjand, northeastern Iran, to characterize the parent-child CVD risk factor relationship in parents with a positive history of premature myocardial infarction (MI) and their offspring.

<sup>(1)</sup> Azita Fesharak Nia MD, Assistant professor of pediatric nephrology, Birjand University of Medical Sciences, Iran Tel: +98 915 561 3254, E-mail: fesharakinia@yahoo.com

<sup>(2)</sup> Asghar Zarban Ph.D., Assistant professor of biochemistry, Birjand University of Medical Sciences

<sup>(3)</sup> Toba Kazemi MD, Assistant professor of cardiology, Birjand University of Medical Sciences

<sup>(4)</sup> Gholam Reza Sharifzadeh M.Sc., Methodological and statistical advisor, Birjand University of Medical Sciences

#### Materials and methods

The studied population consisted of 91 parents, randomly selected from among patients who had suffered premature myocardial infarction (<55 years) and were hospitalized in the coronary care units (CCU) of the Vali-e-Asr Hospital affiliated to Birjand University of Medical Sciences. Only one child (age 2-14 years) was randomly selected from every family. Parents who were taking anti-hypertensive medications were excluded. None of the parents were taking cholesterol-lowering medications at the time of examination.

Height and weight were measured in light clothing and no shoes. Weight (Seca Beam Balance) and height (Seca stadiometer) were measured to the nearest 0.1 kg and 0.1 cm, respectively. Body mass index (BMI) was measured as weight (kg) divided by weight (kg) squared.¹¹ Parents with 25 ≤BMI ≤29.9 and BMI ≥30 were considered as overweight and obese, respectively.¹² Children with BMI ≥ 95th percentile and 85th percentile ≤ BMI < 95th percentile were considered as overweight and at risk of overweight, respectively.¹³

Blood Pressure was measured on the subjects' right arm of subjects in a relaxed, sitting position using a mercury sphygmomanometer with suitable cuff size. The mean of two measurements of Korotkoff phase I and the mean of two values of phase IV (in children) and phase V (in parents) were recorded for systolic blood pressure (SBP) and diastolic blood pressure, respectively.

Based on the WHO definition, parents with SBP ≥140 mmHg or DBP ≥90 mmHg were considered as hypertensive. 12 Children with systolic or diastolic

blood pressure greater than the 95th percentile were considered as hypertensive.<sup>14</sup>

Subjects had been instructed to fast for 12 to 14 hours. Antecubital venous blood was collected. Biochemical tests, including measurement of TC, triglyceride (TG), High- density lipoprotein cholesterol (HDL-C) and LDL-C were carried out. TC and TG were measured by German made Ependrof Elan 2000 autoanalyzer using the enzymatic method.

HDL-C was measured using heparin-manganese precipitation method.¹⁵ LDL-C was measured in samples containing TG ≤400 mg/dl using the Friedwald formula.¹⁶ It was otherwise measured using a special test kit. Parents with TG ≥200, TC ≥240, HDL-C ≤40, or LDL >100 (mg/dl) were considered as dyslipidemic.¹७ In children, high levels of TC and LDL-C were defined as those >200 mg/dl and >130 mg/dl, respectively.¹৪

TG level ≥130 mg/dl was considered high and HDL-C level <35 mg/dl was considered low. 19,20 Statistical analysis was performed by the SPSS statistical package using independent t-test and partial Pearson correlation coefficients. P values less than 0.05 were considered as significant.

### Results

Ninety-one parents (85 fathers, 6 mothers) with positive history of premature MI and 91 offspring (45 girls, 46 boys) were studied. Parents and children had mean ages of 44.4±4.5 and 11.2±2.6 years, respectively. Our study showed no significant relation of systolic and diastolic blood pressure between parents and their children (Table 1).

**TABLE 1.** Mean levels of BP, BMI and serum lipids in parents and their children and relation between them

Variable	Children (n=91) Mean ± SD	Parents (n=91) Mean ± SD	Partial correlation (r)	Pearson coefficients (p)
SBP (mmHg)	97.5±9.6	122.6±13.5	0.13	0.23
DBP (mmHg)	64±8.6	80.5±9	0.21	0.06
BMI $(kg/m^2)$	17.4±3.2	26.9±4.6	0.31	0.04*
TC (mg/dl)	143.6±24.9	168.9±39.3	0.04	0.73
TG (mg/dl)	89±35.6	159.2±100.5	0.04	0.7
HDL (mg/dl)	38.5±11	39.4±11.4	0.16	0.17
LDL (mg/dl)	82±16.9	99.4±24.9	0.12	0.31

**TABLE 2.** Mean blood pressure in children of hypertensive and normotensive parents

Variable	BP (mmHg) Children of hypertensive	BP (mmHg) Children of normotensive	P value
	$(Mean \pm SD)$	$(Mean \pm SD)$	
SBP	$100 \pm 10.2$	$95.9 \pm 8.3$	0.05*
DBP	$66.3 \pm 8.5$	$62.3 \pm 7.6$	0.03*

Variable (mg/dl)	Children (n=91) Mean ± SD		Parents (n=91) Mean ± SD		P value
	N	0/0	N	%	
High – cholesterol	1	1.1	18	19.8	< 0.001*
High TG	32	35.2	23	23.1	0.15
High LDL	1	1.1	31	34.1	< 0.001*
Low HDL	34	37.4	47	51.6	< 0.05*

**TABLE 3.** Prevalence of lipid disorders in parents and their children

Thirty parents (33%) had hypertension. No cases of hypertension were found in children. Mean systolic and diastolic blood pressure were significantly higher in children of hypertensive parents (Table 2). There was a significant relation of BMI between parents and their children (Table 1).

28.6% (26 subjects) and 27.5% (25 subjects) of parents were obese and overweight, respectively. 3.3% (3 subjects) and 9.9% (9 subjects) of children were overweight and at risk of overweight, respectively. There was a significant relation of obesity between parents and their children (P<0.001). There was no significant relation of lipid profile between parents and their children (Table 1). 19.8% of parents and 1.1% of children had high TC levels. 23.1% of parents and 35.2% of children had high TG levels. 34.1% of parents and 1.1% of children had high LDL-C levels. 51.6% of parents and 37.4% of children had low HDL levels. There was a significant relation of lipid disorders (except for high TG level) between parents and their children (Table 3). Low HDL was the commonest lipid disorder in both parents and children.

#### **Discussion**

Familial aggregation of cardiovascular risk factors including blood pressure, serum lipids and obesity has been extensively investigated.<sup>21,22</sup>

It has been demonstrated that both genetic and environmental factors contribute to the variability of risk factors and their familial aggregation.<sup>23,24</sup> Our study did not show a significant relation of systolic and diastolic blood pressure levels between parents and their children. This supports the findings reported by Bao et al.<sup>2</sup> and Jago et al.<sup>10</sup> but contradicts the conclusions reported by Fuentes et al.<sup>25</sup> and Stamler et al.<sup>26</sup>

The data in the present study regarding higher mean systolic and diastolic blood pressure in the offspring of hypertensive parents are in line with many other reports, such as the studies by Kelishadi et al.,<sup>27</sup> Elias et al.<sup>28</sup> and Richard et al.<sup>29</sup> Thus it would be advisable

to track childhood blood pressure, especially in the offspring of hypertensive parents.

Our study showed a significant relation of BMI and obesity between parents and their children. In a study of CVD in Bogalusa, the most significant relationships between parents and their children were for height and weight.<sup>30</sup> In another study, there was a significant association of BMI (P<0.05) between Hispanic mothers and their children, but not in other ethnic groups.<sup>10</sup>

Child obesity increases the risk of obesity in adulthood and is associated with CVD risk factors,31 hence preventive work should begin in early childhood, particularly in children of families at high risk for coronary artery disease (CAD). Our study did not show a significant relation of serum lipids between parents and their children. This supports the findings reported by Bao et al.<sup>2</sup> and Shear et al.,<sup>32</sup> but Jago et al.<sup>10</sup> found that HDL and LDL levels were significantly associated between African American mothers and their children, but not in other ethnic groups. Parent-child associations of serum cholesterol were observed in a cohort of 440 children (from birth to 7 years of age) and their parents in Bogalusa.30 Adult dyslipidemia may reveal familial and therefore, offspring dyslipidemia.33 Our study showed a significant relation of dyslipidemia except for high TG between parents and their children. Another study showed that increased parental lipid levels are associated with persistently and substantially higher lipid levels in their offspring.8

The commonest lipid disorders both in parents and children in our study was low HDL that is an independent risk factor for CAD.<sup>34</sup>

Epidemiologic studies have suggested that multiple risk factors increase the probability of cardiovascular events, since CVD risk factors tend to reinforce each other in their influence on morbidity and mortality.<sup>35</sup> A family history of premature MI is a risk factor for CVD<sup>2</sup> and detecting other risk factors such as hypertension, obesity and dyslipidemia is very important.

This study showed a significant relation of CVD risk factors between parents with a positive history of premature MI and their offspring, so we recommend serial measurement of BP, BMI and lipids in these children from early childhood through young adulthood.

#### References

- 1. Tershakovec AM, Rader DJ. Disorders of lipoprotein metabolism and transport. In: Behrman RE, Kilegman RM. Nelson text book of pediatrics: from WB Saunders Company. Philadelphia: USA, 16th ed,2004: 445.
- 2. Bao W, Srinivasan SR, Valdez R, Greenlund KJ, Wattigney WA, Berenson GS. Longitudinal changes in cardiovascular risk from childhood to young adulthood in offspring of parents with coronary artery disease: The Bogalusa Heart Study. JAMA, 1997; 278(21): 1749-1754.
- 3. Colditz GA, Stampfer MJ, Willett WC, Rosner B, Speizer FE, Hennekens CH. A prospective study of parental history of myocardial infarction and coronary heart disease in women, Am J Epidemiol, 1986;123: 48-
- 4. Myers RH, Kiely DK, Cupples LA, Kannel WB. Parental history is an independent risk factor for coronary artery disease (the Framingham study), Am Heart J, 1990;120:
- 5. Wang X, Wang B, Chen C, et al. Familial aggregation of blood pressure in a rural Chinese community. Am J Epidemiol, 1999;179: 412-420.
- 6. Zinner SH, Levy PS, Kass EH. Familial aggregation of blood pressure in childhood. N Engl J Med, 1971;284:
- 7. Garn SM. Family-line and socioeconomic factors in fatness and obesity. Nutr Rev, 1986;44: 381-386.
- 8. Uiterwaal CS, Witteman JC, De Bruijn AM, Hofman A, Grobbee DE. Families and natural history of lipids in childhood (an 18-year follow-up study). Am J Epidemiol, 1997;145: 777-785.
- 9. Szamosi T, Murber A, Szamosi T, Tory V, Kosztolicz A, Sztankits K. Atherosclerosis risk factors in children of high risk families. Acta Physiol Hung. 1999; 86 (93-4): 185-90.
- 10. Jago R, Baranowski T, Watson K, et al. Relationships between maternal and child cardiovascular risk factors: Ethnic differences and lack of influence of physical activity. Archives of pediatrics & adolescent medicine. Chicago, Dec 2004;158 (12): 1125-1131.
- 11. National Institutes of Health. The practical guide identification, evaluation and treatment of overweight and obesity in adults. NIH publication 2000: 9.
- 12. Azizi F, Raizadeh P, Salehi M, et al. Determinates of serum HDL-C level in a Tehran urban population: The Tehran Lipid and Glucose Study. Nutr metab Cardiovas Dis 2002; 12: 80-89.
- 13. Needlman R. Assessment of Growth. In: Behrman RE, Kilegman RM, Nelson text book of pediatrics: from WB Saunders Company. Philadelphia: USA, 16th ed,2004: 61.
- 14. Bernstein D. Evaluation of the cardiovascular system. In: Behrman RE, Kilegman RM., Nelson text book of pediatrics: from WB Saunders company. Philadelphia: USA, 16th ed,2004: 1486-1487.

- 15. Warnick GR, Benderson J, Albers JJ. Dextran sulfate Mg2+ precipitation procedure for quantification of high density lipoprotein cholesterol. Clinical Chemistry 1982; 28 (6): 1379-1388.
- 16. Friedewald WT, Levy RI, Fredrichson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clinical Chemistry 1972; 18: 499-502.
- 17. Azizi F, Raiszadeh P, Salehi M, et al. Determinates of serum HDL-C level in a Tehran urban population: The Tehran Lipid and Glucose Study, Nutr Dis 2002; 12: 80-
- 18. Perusse L, Chagnon YC, Dionne FT, Bouchard C. The human obesity gene map: the 1996 update. Obes Res 1997; 5: 49-61.
- 19. Schappert SM. National Ambulatory Medical Care Survey: 1994 Summary, Advance data from vital and health statistics. No. 273, Hyattsville, Md: National center for health statistics, 1996, DHHS Publication No (PHS); 96-1250.
- 20. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard growth study of 1922 to 1935. N Engl J Med 1992; 327: 1350-1355.
- 21. Hunt SC, Hasstedt SJ, Kuida H, Stults BM, Hopkins PN, Williams RR. Genetic heritability and common environmental components of resting and stressed blood pressure, lipids and body mass index in Utah pedigrees and twins. Âm J epidemiol 1989;(129): 625-638.
- 22. Vizcajno VM, Aguilar FS, Gutierrez RF, Crespo YJ, Navalon RG, Rojas VD. Familial aggregation of cardiovascular disease risk factors: The Cuenca Study. Prev Med 1999; (28): 131-137.
- 23. Breslow JL. Genetics of lipoprotein disorders. Circulation 1993; 87 Suppl III: 16-21.
- 24. Faith MS, Pietrobelli A, Nunez C, Heo M, Heymsfield SB, Allison DB. Evidence for independent genetic influences on fat mass and body mass index in a pediatrics twin sample. Pediatrics 1999;104: 61-67.
- 25. Fuentes RM, Notkola IL, Shemeikka S, Tuomilehto J, Nissinen A. Familial aggregation of blood pressure a population-based family study in eastern Finland, J Hum Hypertents 2000;(14): 441-445.
- 26. Stamler R, Stamler J, Riedlinger WF, Algera G, Roberts RH. Family (parental) history and prevalence of hypertension, results of a nation wide screening program. JAMA 1979; (241): 43-46.
- 27. Kelishadi R, Sarraf Zadegan N, Nadery Gh.A, Asgary S, Bashardoust N. Atherosclerosis risk factor in children and adolescents with or without family history of premature coronary artery disease. Med Sci Monit, 2002; 8(6): 425-429.
- 28. Elias MC, Bolivar MSM, Fonseca FAH, et al. Comparison of the lipid profile, blood pressure and dietary of adolescents and children descended from hypertensive and normotensive individuals. Arg Bras Cardiol, Vol.82, No.2.
- 29. Richard M, Schieken M: Genetic factors that predispose the child to develop hypertension. Pediatr Clin North Am, 1993; 40 (1).
- 30. Rosenbaum PA, Elston RC, Srinivasan SR, Webber LS. 7 year of age: The Bogalusa Heart Study. Pediatrics 1987;(80:5): 807-816.

- 31. Chu NF, Rimm EB, Wang DJ, Lious HS, Shieh SM. clustering of cardiovascular disease risk factors among obese school children: The Taipei Children Heart Study. American journal of clinical nutrition 1998;(67): 1141-1146.
- 32. Shear CL, Frerichs RR, Weinberg R, Berenson GS. Childhood sibling aggregation of coronary artery disease risk factors in a biracial community. Am J epidemiol 1978;(107): 522-528.
- 33. Lapinleimu J, Nuotio IO, Lapinleima H, Simell OG, Rask- Nissila L, Viikari JSA. Recognition of familial dyslipidemias in 5- year- old children using the lipid
- phenotypes of parents. The STRIP Project. Atherosclerosis 2002;(160): 417-423.
- 34. Wissler RW,for the PADY Research Group.An overview of quantitative influence of several risk factors or progression of atherosclerosis in young people in the united states. Am J Med Sci 1995;310:29-36.
- 35. Kannel WB, Wolf PA, Garrison RJ. The Framingham study: an epidemiological investigation of cardiovascular disease. Bethesda, Md. National Heart, Lung and Blood Institute 1987 (NIH) Publication; (87): 2703.

