cholesterol and degree of fat unsaturation on plasma lipid levels, lipoprotein composition, and fecal steroid excretion in normal young adult men. *Am J Clin Nutr.* 1985;42:399–413.
43. Becker N, Illingworth DR, Alaupovic P, Connor WE, Sundberg EE. Effects of saturated, monounsaturated, and omega-6 polyunsaturated fatty acids on plasma lipids, lipoproteins, and apoproteins in humans. *Am J Clin Nutr.* 1983;37:355–360.
44. Jackson RL, Glueck CJ, Mathur SN, Spector AA. Effects of diet and high density lipoprotein subfractions on the removal of cellular cholesterol. *Lipids.* 1980;15:230–235.
45. Dreon DM, Vranizan KM, Krauss RM, Austin MA, Wood PD. The effects of polyunsaturated fat vs monounsaturated fat on plasma lipids. *JAMA.* 1990;263:2462–2466.
46. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins: a meta-analysis of 27 trials. *Arterioscler Thromb.* 1992;12:911–919.
47. Hegsted DM, Ausman LM, Johnson JA, Dallal GE. Dietary fat and serum lipids: an evaluation of the experimental data. *Am J Clin Nutr.* 1992;57:875–883.
48. Morrison JA, Kelly K, Mellies M, et al. Cigarette smoking, alcohol intake, and oral contraceptives: relationships to lipids and lipoproteins in adolescent school-children. *Metabolism.* 1979;28:1166–1170.
49. Croft JB, Freedman DS, Cresanta JL, et al. Adverse influences of alcohol, tobacco, and oral contraceptive use on cardiovascular risk factors during transition to adulthood. *Am J Epidemiol.* 1987;126:202–213.

# Aerobic Fitness, Blood Lipids, and Body Fat in Children

*Ronald L. Hager, MS, Larry A. Tucker, PhD, and Gary T. Seljaas, MS*

## ABSTRACT

This study examined the association between aerobic fitness and serum cholesterol and the effects of controlling for gender, body composition, abdominal fat, and dietary saturated fat in 262 children. The 1-mile run was used to estimate fitness. Skinfolds were used in assessing body fat. Fit children had lower total cholesterol, low-density lipoprotein cholesterol, and triglyceride levels and higher high-density lipoprotein cholesterol levels than unfit children, except after adjustment for body fat and/or abdominal fat. Unfit children appear to be at an increased risk of unhealthy levels of serum cholesterol due primarily to increased levels of body fat. (*Am J Public Health.* 1995;85:1702–1706)

## *Introduction*

Atherosclerosis has been shown to begin in infancy.[1] Results from the International Atherosclerosis Project identified the presence of fatty streaks in the aortas of many children by 3 years of age.[2] Moreover, fatty streaks observed in the coronary arteries of 10-year-olds have been found to be associated with adult arteriosclerosis.[3]

Research clearly shows that elevated serum lipid levels promote the development of atherosclerosis and are a principal cause of cardiovascular disease.[4–6] Because atherosclerosis can begin to develop in childhood, the early years of life are a good time to intervene to reduce the risk of cardiovascular disease.[7] Research indicates that regular physical activity and subsequent high levels of aerobic fitness can be a valuable method of intervention in the prevention and treatment of hypercholesterolemia in adults.[8–20] Unfortunately, the extent to which these lifestyle factors are associated with cholesterol levels in children is much less clear.

Research has shown that physical activity, sports participation, and training have favorable effects on both high-density lipoprotein (HDL) cholesterol and total/HDL ratio levels in children; however, results have been mixed depending on gender and age.[21–26] Results also have varied regarding training effects on total cholesterol.[22–24]

Although some cholesterol investigations have studied the extent to which blood lipid levels are related to physical activity and exercise in children, little research to date has examined the relation between measured aerobic fitness and blood lipids in children.[24] Yet, study of aerobic fitness and blood lipids is probably a more valid approach than measurement of self-reported physical activity and blood lipids,[8,27] particularly in children. Physical activity is usually a subjective measurement requiring accurate recall. Children's accuracy in reporting their physical activity is likely to be poor and may denote inaccurate perceptions.[8]

The purpose of this study was to determine the extent to which aerobic fitness was associated with blood lipid levels in 262 Utah children 9 and 10 years of age. An ancillary objective was to determine the extent to which demographic, physiological, and lifestyle factors confounded the fitness–cholesterol relation.

## *Methods*

### *Subjects*

A total of 262 children (162 boys and 100 girls) volunteered for participation in the study. Subjects were recruited by newspaper advertisements and word of mouth. Ninety-five percent of the subjects

The authors are with the Department of Physical Education, Brigham Young University, Provo, Utah.

Requests for reprints should be sent to Larry A. Tucker, PhD, Department of Physical Education, 237 SFH, Brigham Young University, Provo, UT 84602-2208.

This paper was accepted June 29, 1995.

were White; other descriptive information and demographic data are displayed in Table 1.

*Data Collection and Measurement*

Subjects and parents completed questionnaires requesting information on demographics and dietary intake. Body weight and height were assessed with the children wearing light exercise clothes but not wearing shoes.

The 1-mile run/walk test was used for the measurement of aerobic fitness. This test is recommended for the measurement of aerobic fitness in children in kindergarten through fourth grade.[28] The test has been shown to have good intraclass reliability ($.83 < r < .90$) for both boys and girls in grades 3 and 4.[29]

Aerobic fitness was indexed in two ways: (1) by time on the 1-mile run/walk test only and (2) by estimated oxygen consumption per kg body weight ($VO_2$). In the latter index, a regression equation that included three variables—gender, sum of three skinfolds (triceps, calf, subscapula), and 1-mile run/walk time—was used. The standard error of estimate for the $VO_2$ measure is 3.96 for children 6 to 13 years of age.[30]

To determine percentage of body fat, three sites on the right side of the body—triceps, subscapula, and calf—were assessed with Harpenden calipers. All skinfolds were assessed by the same researcher to eliminate intertester variability. The test–retest intraclass correlations on a random sample of 30 subjects were greater than .99. The three skinfold measurements were used in two separate formulas (one using triceps and calf and the other using triceps and subscapula) described by Lohman.[31] The standard error of estimate for the two equations are 3.8 and 3.6 to 3.9, respectively.[31] The average of the two results was calculated to index the percentage of body fat. The protocol for precise skinfold locations (as outlined by Allsen et al.[32]) was followed.

Abdominal fat was assessed by taking a skinfold measure at a location 3.8 cm (1.5 in) from the umbilicus. Research has shown that a positive relationship exists between body fat distribution, particularly abdominal fat, and blood lipids.[33,34] Furthermore, amount of abdominal fat has been found to have a negative correlation with levels of high-density lipoprotein cholesterol.[35,36]

Dietary intake was assessed with the food frequency component of the Health Habits and History Questionnaire developed by the National Cancer Institute.[37] This food frequency instrument, when used to calculate nutrients from a diet record, has yielded correlations of greater than .70 in comparisons with actual nutrient intake, and field administration has produced mean values comparable to national data.[37] The subjects' parents completed the questionnaire with input from the subjects.

As a means of determining serum lipid levels, blood was drawn from an antecubital vein after subjects had fasted for 12 hours. A certified laboratory analyzed the blood using the enzymatic method.[38]

**TABLE 1—Descriptive and Demographic Data: 262 Utah Children**

| | All Subjects (n = 262) | | Boys (n = 162) | | Girls (n = 100) | |
|---|---|---|---|---|---|---|
| | Mean | SD | Mean | SD | Mean | SD |
| General | | | | | | |
| Age, y | 9.79 | 0.48 | 9.88 | 0.44 | 9.63 | 0.51 |
| Height, cm | 141.95 | 7.69 | 142.47 | 7.72 | 141.43 | 7.67 |
| Weight, kg | 35.67 | 8.35 | 35.85 | 8.39 | 35.37 | 8.31 |
| Blood lipids | | | | | | |
| Total cholesterol, mmol/L | 4.37 | 0.80 | 4.42 | 0.86 | 4.31 | 0.70 |
| Total cholesterol, mg/dl | 169.12 | 30.99 | 170.77 | 33.23 | 166.53 | 27.02 |
| High-density lipoprotein cholesterol, mmol/L | 1.25 | 0.31 | 1.31 | 0.33 | 1.16 | 0.25 |
| High-density lipoprotein cholesterol, mg/dl | 48.36 | 11.81 | 50.51 | 12.63 | 44.93 | 9.50 |
| Low-density lipoprotein cholesterol, mmol/L | 2.72 | 0.72 | 2.73 | 0.77 | 2.71 | 0.64 |
| Low-density lipoprotein cholesterol, mg/dl | 105.15 | 28.00 | 105.50 | 29.94 | 104.61 | 24.78 |
| Triglycerides, mmol/L | 2.03 | 1.09 | 1.91 | 1.06 | 2.22 | 1.13 |
| Triglycerides, mg/dl | 78.52 | 42.03 | 73.81 | 40.95 | 85.95 | 43.83 |
| Fitness | | | | | | |
| $VO_2$, ml/kg/min | 51.35 | 7.17 | 53.43 | 7.21 | 47.99 | 5.70 |
| Time on 1-mile run/walk test, min | 10.37 | 2.40 | 9.96 | 2.45 | 11.03 | 2.16 |
| Body fat | | | | | | |
| Total body fat, % | 20.90 | 8.08 | 19.15 | 8.67 | 23.73 | 6.07 |
| Abdominal skinfold, mm | 11.60 | 8.69 | 11.61 | 9.34 | 11.57 | 7.24 |
| Dietary fat intake | | | | | | |
| Saturated fat intake, %[a] | 13.13 | 2.33 | 12.92 | 2.21 | 13.48 | 2.50 |

[a]Percentage of total energy intake derived from saturated fat.

*Data Analysis*

Serum cholesterol levels and aerobic fitness were treated as continuous variables. Pearson product–moment correlation coefficients were calculated to determine the extent and direction of the bivariate associations between the blood lipids and the two measures of aerobic fitness. Trend analysis, using the multiple regression technique, was computed up to the cubic level to ascertain the extent of curvilinear relations between each of the cholesterol measures and fitness. Partial correlation was used to determine the extent of the association between serum cholesterol and aerobic fitness, with potential confounders controlled statistically.

## Results

As can be seen in Table 2, regression analysis showed that both estimated $VO_2$ and time on the 1-mile run/walk test individually accounted for a significant percentage of the variance in all of the blood lipids, particularly triglycerides, without control for any of the potentially confounding variables. After differences in gender and dietary saturated fat intake had been controlled, $VO_2$ and time on the 1-mile run/walk test remained significant contributors to all of the blood lipid measures. However, after adjustment for differences in abdominal fat, the significant associations between both measures of fitness and the various blood lipid measures were eliminated. Similarly, when body fat percentage was controlled, there

**TABLE 2—Results of Multiple Regression Analysis**

| Blood Lipid and Variable(s) Controlled | F | $R^2$ | P |
|---|---|---|---|
| **Estimated $VO_2$** | | | |
| Total cholesterol | | | |
| None | 10.32 | .039 | <.01 |
| Gender | 15.52 | .058 | <.01 |
| Gender, abdominal skinfold | 0.27 | .001 | .61 |
| Gender, % body fat | 0.06 | .000 | .80 |
| Gender, saturated fat intake | 15.54 | .058 | <.01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 0.04 | .000 | .85 |
| High-density lipoprotein cholesterol | | | |
| None | 12.95 | .049 | <.01 |
| Gender | 5.73 | .021 | .02 |
| Gender, abdominal skinfold | 0.65 | .002 | .42 |
| Gender, % body fat | 2.41 | .009 | .12 |
| Gender, saturated fat intake | 7.76 | .027 | <.01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 1.97 | .007 | .16 |
| Low-density lipoprotein cholesterol | | | |
| None | 10.64 | .040 | <.01 |
| Gender | 13.17 | .050 | <.01 |
| Gender, abdominal skinfold | 0.09 | .000 | .77 |
| Gender, % body fat | 0.41 | .002 | .55 |
| Gender, saturated fat intake | 13.70 | .052 | <.01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 0.05 | .000 | .83 |
| Triglycerides | | | |
| None | 42.11 | .143 | <.01 |
| Gender | 36.10 | .123 | <.01 |
| Gender, abdominal skinfold | 0.26 | .001 | .61 |
| Gender, % body fat | 1.47 | .005 | .23 |
| Gender, saturated fat intake | 40.61 | .134 | <.01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 0.63 | .002 | .43 |
| **Time on 1-mile run/walk test** | | | |
| Total cholesterol | | | |
| None | 4.32 | .017 | .04 |
| Gender | 5.82 | .022 | .02 |
| Gender, abdominal skinfold | 0.06 | .000 | .80 |
| Gender, % body fat | 0.06 | .000 | .81 |
| Gender, saturated fat intake | 5.77 | .022 | .02 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 0.03 | .000 | .87 |
| High-density lipoprotein cholesterol | | | |
| None | 8.97 | .034 | <.01 |
| Gender | 4.87 | .018 | .03 |
| Gender, abdominal skinfold | 1.34 | .005 | .25 |
| Gender, % body fat | 2.12 | .008 | .15 |
| Gender, saturated fat intake | 6.14 | .022 | .01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 1.76 | .006 | .19 |
| Low-density lipoprotein cholesterol | | | |
| None | 5.89 | .023 | .02 |
| Gender | 6.55 | .025 | .01 |
| Gender, abdominal skinfold | 0.06 | .000 | .81 |
| Gender, % body fat | 0.55 | .002 | .46 |
| Gender, saturated fat intake | 6.73 | .026 | .01 |
| Gender, % body, fat, abdominal skinfold, saturated fat intake | 0.13 | .001 | .72 |
| Triglycerides | | | |
| None | 16.69 | .062 | <.01 |
| Gender | 13.28 | .049 | <.01 |
| Gender, abdominal skinfold | 0.05 | .000 | .82 |
| Gender, % body fat | 0.50 | .002 | .48 |
| Gender, saturated fat intake | 14.75 | .054 | <.01 |
| Gender, % body fat, abdominal skinfold, saturated fat intake | 0.10 | .000 | .76 |

*Note.* Values for F, $R^2$, and P represent not the total model but the contribution of the blood lipid after control for the other potential confounders.

was no link between the fitness measures and the blood lipid variables. None of the curvilinear relationships between the two fitness measurements and the blood lipid levels were significant.

## *Discussion*

The sample of children studied in the present investigation displayed higher than average measures of total cholesterol (169.12 mg/dl), low-density lipoprotein cholesterol (105.15 mg/dl), and triglycerides (78.52 mg/dl) and lower than average results for high-density lipoprotein cholesterol (48.36 mg/dl) in comparison with data on children of similar ages who participated in the Lipid Research Clinics Prevalence Study (values of 163 mg/dl, 99.5 mg/dl, 73 mg/dl, and 55.5 mg/dl, respectively).[39] In terms of the current cut points of 170 mg/dl for increased risk in children and 200 mg/dl (95th percentile) for high-risk total cholesterol,[40] results for 26.5% of the boys and 37.0% of the girls in the present study corresponded to the increased risk category, and results for 17.3% of the boys and 8.0% of the girls corresponded to the high-risk category (see Table 1).

According to the regression results, level of aerobic fitness is a significant predictor of blood lipid levels in children. However, in the present study, this relationship was influenced strongly by levels of abdominal fat and percentage of body fat. Hence, it appears that aerobic fitness and physical activity are related to blood lipids in children as a function of body fat variation.

The interrelationships among the triad of physical activity/fitness, body fat, and blood lipid levels have been well established in adults. Many studies have shown that regular physical activity and a high fitness level can have favorable effects on total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides, particularly the latter two.[8,21–26,41] In addition, a number of studies have indicated that obesity is closely linked to undesirable blood lipid levels.[35,42–45] Furthermore, research indicates that there is a significant relationship between body fat distribution, particularly abdominal fat, and blood lipids.[33–36] Finally, abundant data show a strong connection between physical activity level and body fat percentage.[46,47] Hence, it is not surprising that people who have sedentary lifestyles also have low levels of fitness, excess body fat, and undesirable blood lipid levels.

In the present study of children, fitness, body fat, and blood lipid levels were significantly interconnected. Post hoc analyses indicated that, after gender had been controlled, time on the 1-mile run/walk test was closely tied to body fat ($r = .50$, $P = .0001$), body fat levels were linked to low-density lipoprotein cholesterol ($r = .23$, $P = .0002$), and low-density lipoprotein cholesterol was correlated significantly with aerobic fitness ($r = .16$, $P = .0111$). If high levels of aerobic fitness and low levels of body fat actually contribute to more favorable blood lipid concentrations in children, it may be most beneficial to encourage increased levels of physical activity in children, thus reducing body fat.

Given that this study was cross sectional in design, cause-and-effect conclusions are not warranted. However, if a causal relation is assumed, it appears that the goal of favorably altering blood lipids in children should begin with increasing physical activity and fitness, which in turn will lead to reductions in body fat. Moreover, because children who are physically unfit or who carry excess body fat are more likely to suffer from unhealthy blood lipid levels than their physically fit counterparts, it seems that these high-risk children should be screened for possible blood lipid problems more frequently than normal. □

## References


1. Cresanta JL, Hyg RT, Burke GL, Downey AM, Freedman DS, Berenson GS. Prevention of atherosclerosis in childhood. *Pediatr Clin North Am.* 1986;33:835–858.
2. McGill HC Jr. Morphologic development of the atherosclerotic plaque. In: Lauer RM, Shekelle RR, eds. *Childhood Prevention of Atherosclerosis and Hypertension.* New York, NY: Raven Press; 1980:41–49.
3. McGill HC Jr, Arias-Stellen J, Carbonnell LM, et al. Physical fitness: its contribution to serum high-density lipoprotein. *Atherosclerosis.* 1983;48:173.
4. *Lipid Research Clinics Program.* Washington, DC: The Lipid Research Clinics; 1984.
5. Expert Panel on Blood Cholesterol Levels in Children and Adolescents. *Report of the Expert Panel on Blood Cholesterol Levels in Children and Adolescents.* Washington, DC: US Dept of Health and Human Services; 1991. NIH publication 91-2732.
6. Stamler J. Lifestyles, major risk factors, proof and public policy. *Circulation.* 1978; 58:3.
7. Byrne KP. *Understanding and Managing Cholesterol: A Guide for Wellness Professionals.* Champaign, Ill: Human Kinetics Books; 1991.
8. Tucker LA, Bagwell M. The relation between aerobic fitness and serum cholesterol levels in a large employed population. *Am J Health Promo.* 1991;6:17–23.
9. Paffenbarger RS, Hyde RT, Wing AL, Hsieh CC. Physical activity, all-cause mortality and longevity of college alumni. *N Engl J Med.* 1986;314:605–613.
10. Paffenbarger RS, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. *Am J Epidemiol.* 1978;108:161–175.
11. Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical activity and the incidence of coronary heart disease. *Annu Rev Public Health.* 1987;8:253–287.
12. Garcia-Palmieri MR, Costas R, Cruz-Vidal M, Sorlie PD, Havlik RJ. Increased physical activity: a protective factor against heart attacks in Puerto Rico. *Am J Cardiol.* 1982;50:749-755.
13. Kannel WB, Sorlie P. Some health benefits of physical activity. The Framingham Study. *Arch Intern Med.* 1979;139:857–861.
14. Leon AS, Connett J, Jacobs DR, Rauramaa R. Leisure-time physical activity and risk of coronary heart disease and death: the multiple risk factor intervention trial. *JAMA.* 1987;258:2388–2395.
15. Haskell WL. Exercise-induced changes in plasma lipids and lipoproteins. *Prev Med.* 1984;13:23–36.
16. Joseph JJ, Bena LL. Cholesterol reduction: a long term intense exercise program. *J Sports Med Phys Fitness.* 1977;17:163–168.
17. Straja D, Mymin D. Moderate exercise and high-density lipoprotein-cholesterol: observations made during a cardiac rehabilitation program. *JAMA.* 1979;242:2190–2192.
18. Tran ZV, Weltman A. Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight. A meta-analysis. *JAMA.* 1985;254:919–924.
19. Bennett PN. Effect of physical exercise on platelet adhesiveness. *Scand J Haematol.* 1972;9:138.
20. Wood PD, Haskell WL, Klein H, Lewis S, Stein MP, Farquhar JW. The distribution of plasma lipoproteins in middle-aged runners. *Metabolism.* 1976;25:1249–1257.
21. Viikari J, Välimäke I, Telama R, et al. Atherosclerosis precursors in Finnish children: physical activity and plasma lipids in 3- and 12-year-old children. In: Ilmarinen J, Välimäke I, eds. *Children and Sports.* Berlin, Germany: Springer-Verlag; 1984: 231–240.
22. Välimäke I, Hursti ML, Pihlaskoski L, Viikari J. Exercise performance and serum lipids in relation to physical activity in school children. *Int J Sports Med.* 1980;1: 132–136.
23. Durant RH, Linder CW, Harkess JW, Gray RG. The relationship between physical activity and serum lipids and lipoproteins in Black children and adolescents. *J Adolesc Health Care.* 1983;4:55–60.
24. Gilliam TB, Freedman PS, MacConnie SE, Geenen DL, Pels AE. Comparison of blood lipids, lipoproteins, anthropometric measures, and resting and exercise cardiovascular responses in children, 6–7 years old. *Prev Med.* 1981;10:754–764.
25. Thorland WG, Gilliam TB. Comparison of serum lipids between habitually high and low active pre-adolescent males. *Med Sci Sports Exerc.* 1981;13:316–321.
26. Wanne O, Viikari J, Välimäke I. Physical performance and serum lipids in 14–16-year-old trained, normally active, and inactive children. In: Ilmarinin J, Välimäke I, eds. *Children and Sports.* Berlin, Germany: Springer-Verlag; 1984:241–246.
27. Blair SN, Jacobs DR, Powell KE. Relationships between exercise or physical activity and other health behaviors. *Public Health Rep.* 1985;100:172–180.
28. Ross JG, Delpy LA, Christenson GM, Gold RS, Damberg CL. Study procedures and quality control. *J Phys Educ Recreation Dance.* 1987;58:57–62.
29. Rikli RE, Petray C, Baumgartner TA. The reliability of distance run tests for children in grades K–4. *Res Q Exerc Sport.* 1992;63: 270–276.
30. Damitz SR, Ebbling CE, Ward A, Freedson P, Rippe JM. Validity of the one mile run/walk test in children ages 6 to 13 years. *Med Sci Sports Exerc.* 1994;26(suppl):S209.
31. Lohman TG. *Advances in Body Composition Assessment.* Champaign, Ill: Human Kinetics Books; 1992.
32. Allsen PE, Harrison JM, Vance BV. *Fitness for Life. An Individualized Approach.* Dubuque, Iowa: Wm C Brown; 1989.
33. Larsson B, Svärdsudd K, Welin L, Wilhemsen L, Björntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13-year follow-up of participants in the study of men born in 1913. *BMJ.* 1984;288: 1401–1404.
34. Donahue RP, Abbott RD, Bloom E, Reed DM, Yano K. Central obesity and coronary heart disease in men. *Lancet.* 1987;1:821–824.
35. Webber LS, Srinivasan SR, Wattigney WA, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to adulthood: the Bogalusa Heart Study. *Am J Epidemiol.* 1991;133:884–899.
36. Foster CJ, Weinsier RL, Birch R, Norris DJ, Bernstein RS, Wang J. Obesity and serum lipids: an evaluation of the relative contribution of body fat and fat distribution to lipid levels. *Int J Obes.* 1987;11:151–161.
37. Block G, Hartmen AM, Dresser CM, Carroll MD, Gannon J, Gardner L. A data-based approach to diet questionnaire design and testing. *Am J Epidemiol.* 1986; 124:453–469.
38. *Manual of Laboratory Operations.* Washington, DC: US Dept of Health, Education, and Welfare; 1974;1. DHEW publication NIH 75-628.
39. National Heart, Lung and Blood Institute. *The Lipid Research Clinics Population Studies Data Book.* Washington, DC: US Dept of Health and Human Services; 1980. NIH publication 80-1527.
40. Expert Panel of the National Cholesterol Education Program. Report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults. *Arch Intern Med.* 1988;148:36–39.
41. Yamamoto A, Horibe H, Sawada S, et al. Relationship between body fat distribution and blood lipids in obese adolescents. *Int J Obes.* 1988;14:271–277.
42. Khoury P, Morrison JA, Kelly K, Mellies, Horvitz R, Glueck CJ. Clustering and

interrelationships of coronary heart disease risk factors in schoolchildren, ages 6–19. *Am J Epidemiol.* 1980;112:524–538.
43. Freedman DS, Burke GL, Harsha DW, et al. Relationship of changes in obesity to serum lipid and lipoprotein changes in childhood and adolescence. *JAMA.* 1985; 154:515–520.
44. Becque MD, Katch VL, Rocchini AP, Marks CR, Moorehead C. Coronary risk incidence of obese adolescents: reduction by exercise plus diet intervention. *Pediatrics.* 1988;81:605–612.
45. Berenson GS, Frank GC, Hunter SM, Srinivasan SR, Voors AW, Webber LS. Cardiovascular risk factors in children: should they concern the pediatrician? *Am J Dis Child.* 1982;136:855–862.
46. King AC, Tribble DL. The role of exercise in weight regulation in nonathletes. *Sports Med.* 1991;11:331–349.
47. Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a U.S. national cohort. *Int J Obes.* 1993;17:279–286.

# The Reporting Sensitivities of Two Passive Surveillance Systems for Vaccine Adverse Events

*Steven Rosenthal, MD, MPH, and Robert Chen, MD, MA*

ABSTRACT

To evaluate reporting sensitivities for vaccine adverse events, reporting rates were estimated by dividing the number of events reported to the Monitoring System for Adverse Events Following Immunization and the Vaccine Adverse Event Reporting System in a given period by the number of doses administered or distributed during the same period. Reporting sensitivity was calculated as the ratio of the rates at which events were reported to each passive surveillance system (numerator) and occurred in controlled studies (denominator). Reporting sensitivities were generally better in the public sector than in the private sector. The significant underreporting of known outcomes, together with the nonspecific nature of most adverse event reports, highlights the limitations of passive surveillance systems in assessing the incidence of vaccine adverse events. (*Am J Public Health.* 1995;85: 1706–1709)

## *Introduction*

Vaccines are one of the most cost-effective public health measures.[1] But while their benefits far outweigh their risks and costs, no vaccine is perfectly safe. Vaccine safety is initially assessed in prelicensure clinical trials. However, such trials usually have sample sizes that are insufficient to detect rare adverse events. In addition, vaccine trials are usually carried out in well-defined, homogeneous populations with relatively short follow-up periods, which may limit their generalizability. Postlicensure drug evaluations have relied on passive surveillance systems to monitor adverse events. Such systems are more practical and less expensive than controlled trials; however, their data are usually inadequate to determine causality.[2]

Passive surveillance systems for vaccine adverse events have been useful for evaluating contraindications to the diphtheria-tetanus-pertussis (DTP) vaccine[3] and for assessing the safety of simultaneous or combined vaccinations.[4] Reporting sensitivities allow the utility of such systems for detecting and analyzing rare adverse events to be evaluated. In this paper, we assess the reporting sensitivities of two passive vaccine adverse event reporting systems for selected adverse events.

From 1978 through 1990, the Centers for Disease Control and Prevention (CDC) and the Food and Drug Administration (FDA) divided the responsibility for post-marketing surveillance of vaccines in the United States. The FDA received reports of adverse events after vaccines were administered in the private sector; events occurring after the administration of vaccines purchased with public funds were reported to the Monitoring System for Adverse Events Following Immunization.[5]

The monitoring system was a stimulated passive surveillance system. In other words, when vaccines purchased with federal funds were administered in the public sector, "Important Information" forms were given to recipients or their parents or guardians instructing them to report any illnesses requiring medical attention that occurred within 4 weeks of vaccination. System coordinators at each immunization project/grantee site and at the state health department completed standardized forms that were reviewed for consistency and completeness and then forwarded to the CDC for data entry and analysis.[5]

In response to the National Childhood Vaccine Injury Act of 1988, which required health workers to report vaccine adverse events, the CDC and the FDA collaborated in 1990 to implement the Vaccine Adverse Event Reporting System to monitor the safety of vaccines in both sectors.[6] Health care professionals and parents/caretakers are encouraged to report all clinically significant vaccine ad-

The authors are with the National Immunization Program, Centers for Disease Control and Prevention, Atlanta, Ga.

Requests for reprints should be sent to Steven Rosenthal, MD, MPH, National Immunization Program, Centers for Disease Control and Prevention, Mailstop E61, 1600 Clifton Rd, Atlanta GA 30333.

This paper was accepted April 13, 1995.