



Associations of Birth Weight and Length, Childhood Size, and Smoking with Bone Fractures during Growth: Evidence from a Birth Cohort Study

Ianthe E. Jones¹, Sheila M. Williams², and Ailsa Goulding¹

¹ Department of Medical and Surgical Sciences, University of Otago Medical School, Dunedin, New Zealand.

² Department of Preventive and Social Medicine, University of Otago Medical School, Dunedin, New Zealand.

Received for publication May 22, 2003; accepted for publication August 27, 2003.

Little information exists on risk factors associated with bone fractures during childhood and adolescence. This 1972/1973–1990/1991 New Zealand study examined the influence of birth size, height and weight throughout growth, smoking, breastfeeding, and sports participation on the risk of fracture in participants of the Dunedin Multidisciplinary Health and Development Study. Information on height, weight, fracture status, and lifestyle was collected at birth and at ages 3, 5, 7, 9, 11, 13, 15, and 18 years from parents and/or participants. Study members sustained 229 (girls) and 393 (boys) fractures between birth and age 18 years. Fracture risk was elevated (per standard deviation unit increase) in relation to birth length (prepubertal fractures only) (risk ratio (RR) = 1.28, 95% confidence interval (CI): 1.04, 1.58), weight at age 3 years (RR = 1.14, 95% CI: 1.03, 1.27), weight from ages 5 to 18 years (RR = 1.15, 95% CI: 1.03, 1.28), height at age 3 years (RR = 1.13, 95% CI: 1.01, 1.26), and height from ages 5 to 18 years (RR = 1.13, 95% CI: 1.02, 1.24). Birth weight, maternal smoking, breastfeeding, and sports participation had no significant effect on fracture risk. However, for teenagers, personal daily smoking increased the risk of fracture (RR = 1.43, 95% CI: 1.05, 1.95). The authors concluded that tall and heavy children had an increased risk of fracture, as did adolescents who smoked regularly.

body height; body weight; child; cohort studies; growth; risk factors; smoking

Abbreviation: CI, confidence interval.

Approximately 55 percent of all children break at least one bone before the age of 18 years (1). Fractures are the most common injury of childhood requiring hospitalization (2). Thus, childhood and adolescent fractures represent a considerable public health burden. In most populations, fracture incidence peaks in early adolescence and then again during late adult life (3–5). More attention has been given to determining and minimizing the risk of fracturing during later life because of the greater morbidity associated with fractures in this age group. However, less attention has been directed toward determining risk factors for fractures sustained during growth and considering what can be done to minimize them.

A number of groups have found that low bone mass and reduced bone density in childhood or adolescence are associated with clinical risk factors such as maternal smoking during pregnancy (6), reduced rates of breastfeeding (7), low calcium intakes, low physical activity levels (8), reduced birth size, or altered postnatal growth (9). Nevertheless, little

work has been done to elucidate whether these same risk factors play a role in elevating the risk of fracture during childhood and adolescence.

A number of risk factors for fracture during growth have already been identified. We have previously shown that fractures of the distal forearm are associated with low bone density of the total body, forearm, hip, and lumbar spine in both boys and girls (10, 11). Reduced bone mineral density is therefore a risk factor for fracturing during growth, as it is during adulthood (12, 13). Prospectively, low total body bone mineral density has been shown to predict new fractures at any site in girls over a period of 4 years (14). Sustaining at least one previous fracture is also an independent risk factor for further fractures in children (14), similar to adults (15). Whereas, in adults, high body weight acts to increase bone density and reduce fracture risk (16), we and others have found that high body weight during growth is associated with an increased risk of distal forearm fracture (10, 11, 14, 17). Reduced bone size also increases the risk of

Correspondence to Dr. Ailsa Goulding, Department of Medical and Surgical Sciences, University of Otago Medical School, PO Box 913, Dunedin, Otago 9001, New Zealand (e-mail: ailsa.goulding@stonebow.otago.ac.nz).

fracture in overweight girls (17). Other nonbony risk factors identified include high levels of sports participation, lower levels of breastfeeding and inhaled steroid use (18), habitually low calcium intakes (19), and consumption of cola beverages (20).

The aims of this study were to examine whether perinatal growth variables such as birth weight and length have any effect on fracture risk during growth. In addition, we examined the influence of height and weight throughout growth and the influence of breastfeeding, smoking, and sports participation on the risk of sustaining a fracture during growth. The Dunedin Multidisciplinary Health and Development Study provides a unique opportunity to examine risk factors associated with fractures during growth because accurate anthropometric records exist for both birth and later ages (21, 22). Moreover, information regarding fractures was collected regularly throughout growth (1).

MATERIALS AND METHODS

The Dunedin Multidisciplinary Health and Development Study is a longitudinal study of the health, development, and behavior of a sample of children born at the only obstetric hospital in Dunedin, New Zealand, between April 1, 1972, and March 31, 1973. The sample was drawn from all children born at Queen Mary Hospital that year whose mothers had resided in the metropolitan area at the time of the birth and still lived in the Otago area when the children were 3 years of age. Consent to participate was given by the parents of 1,037 of the 1,139 eligible children traced (52 percent male). Members were seen again close to the time of their birthdays at ages 3, 5, 7, 9, 11, 13, 15, and 18 years. The study members' socioeconomic status distribution corresponded to the full range of socioeconomic status distribution in the general population of New Zealand's South Island. The cohort is predominantly Caucasian and, when compared with the New Zealand population, is underrepresentative of Maori and other Pacific peoples. The study is described in detail elsewhere (22).

The Research Ethics Committee of the Otago Hospital Board granted ethics approval for each phase of this longitudinal study. Study members' parents, or later the members themselves, gave informed consent before participating.

Birth weight and length were derived from a larger study in which details of both antenatal and perinatal events were recorded for babies born at Queen Mary Hospital between 1967 and 1973 (23). Duration of pregnancy was measured from the date of the last menstrual period. Infants whose gestational age was known to be 37 weeks or less were excluded from the present study. Weight and height were measured at each visit to the research unit. Weight was recorded to the nearest 0.1 kg by using a Lindell beam balance with the participants wearing only light clothing. Height was measured to the nearest millimeter by using a portable Harpenden stadiometer. Birth weight and length, as well as weight, height, and body mass index at each age thereafter, were standardized to the Centers for Disease Control and Prevention growth charts (24); these values were then used in the present analysis.

We chose an arbitrary cutoff for determining prepuberty of less than age 9 years for the girls and less than age 11 years for the boys (25). Older children were classified as adolescents. We recognize that this cutoff would have misclassified some study members, but no pubertal staging was available for the members.

When children were aged 5 years, the parents were asked to provide information on all injuries suffered by their children during the first 5 years of life. Thereafter, injuries were reported when children were seen at ages 7, 9, 11, 13, 15, and 18 years. Fracture details were derived from this information (1). Bone age was measured at age 7 years, as described previously (26). Information about breastfeeding was obtained at age 3 years and was categorized as more than 4 weeks, yes/no. We also examined the influence of sports participation (reported as minutes per week of physical activity at ages 15 and 18 years) on fracture risk from age 13 years on. When the study members were aged 9 years, maternal smoking was assessed by asking the mothers whether they had smoked during pregnancy. From age 9 years on, the participants were asked about their own personal smoking (27). For the purpose of the current analysis, smoking information collected at ages 15 and 18 years was used because very few members smoked cigarettes daily before this age.

Socioeconomic status was based on the six-point Elley and Irving scale (28) designed for use in New Zealand. We used socioeconomic status information collected at birth and the assessments at ages 9 and 15 years. Participants were described as having a low socioeconomic status if their families were classified as being in the lowest two categories of the scale on at least two occasions and as having a high socioeconomic status if their families were grouped in the highest two categories on at least two occasions; all other combinations were recorded as medium socioeconomic status.

Negative binomial regression with a random effect was used to analyze the data regarding the total number of fractures because we were interested in the number of fractures rather than whether the participant had experienced a fracture. The random effect accounts for the overdispersion in the data and enables it to vary from person to person. The model included terms for interactions between age at which the data were collected and sex, to take into account different fracture rates at different ages for males and females, and terms for the standardized score of the independent variable of interest. Including additional terms for the interaction between age and the variable of interest (such as weight or height) was also considered; however, because these terms were not statistically significant, a single term described the association between standardized weight and height and the risk of fractures over the whole age range. Thus, the estimate for the effect of weight or height was the same for each age. Dividing the group into prepubertal participants or adolescents was used as a more sensitive test of the effect of maturity. Interactions between these two groups and the variable of interest enabled us to describe effects at the different developmental stages. The different exposure times were also taken into account.

TABLE 1. Descriptors of the population studied to assess the association of birth weight and length, childhood size, and smoking with bone fractures during growth, New Zealand, 1972/1973–1990/1991

	Girls		Boys	
	No.	Mean (SD*)	No.	Mean (SD)
Total fractures†	229		393	
Wrist or forearm fractures	55		92	
Prepubertal/adolescent fractures	42/187		114/279	
Birth weight (kg)	468	3.35 (0.49)	500	3.48 (0.48)
Birth length (cm)	468	51.6 (2.08)	500	52.5 (2.06)
Age 5 years	417		436	
Weight (kg)		18.5 (2.30)		19.0 (2.18)
Height (cm)		108.0 (4.38)		108.7 (4.23)
Age 7 years	397		424	
Weight (kg)		23.2 (3.12)		23.5 (2.99)
Height (cm)		120.6 (5.10)		121.4 (4.93)
Age 9 years	364		407	
Weight (kg)		28.6 (4.23)		29.0 (4.11)
Height (cm)		131.7 (5.75)		132.7 (5.46)
Age 11 years	322		353	
Weight (kg)		36.1 (6.28)		35.8 (5.96)
Height (cm)		143.0 (6.57)		142.6 (6.00)
Age 13 years	342		365	
Weight (kg)		49.4 (8.45)		47.0 (8.60)
Height (cm)		156.6 (6.87)		155.4 (8.16)
Age 15 years	388		421	
Weight (kg)		56.1 (8.52)		57.8 (10.46)
Height (cm)		163.2 (5.94)		169.1 (8.20)
Age 18 years	401		432	
Weight (kg)		61.8 (9.42)		70.8 (10.77)
Height (cm)		164.2 (6.13)		176.2 (6.18)

* SD, standard deviation.

† Fractures reported at any skeletal site.

Although the total number of wrist or forearm fractures was small, we wanted to examine the risks of wrist fracture. This fracture is the most common in this cohort, and wrist fractures are often associated with bone fragility. Participants who sustained wrist fractures and participants who experienced other fractures were simultaneously compared with those who did not fracture by using multiple logistic regression with the data from each phase. A person who reported both a wrist fracture and another fracture in the same period was assigned to the wrist fracture group. Because the fracture rate was low, the odds ratios could be compared with the risk ratios generated by negative binomial regression. The observations were not independent because multiple measures for each person were used; therefore, the sandwich estimator was used to adjust the standard errors (29). Inclusion of interaction terms was approached in the same way as that for the negative binomial regression.

Sixty-nine participants were omitted because of prematurity ($n = 41$) or because anthropometric data were missing. The multivariate analyses allowed for the different number of observations at each phase. All data were analyzed by using Stata statistical software, release 7 (Stata Corporation, College Station, Texas, 2001).

RESULTS

Table 1 presents the numbers of participants seen at each age and the means and standard deviations for birth weight, birth length, and weight and height at each age. For girls and boys included in the study, 229 and 393 fractures, respectively, were reported between birth and 18 years of age.

Table 2 shows that birth weight, after adjustments for age and sex, had no effect on the risk of experiencing a fracture throughout growth either prepubertally or during adolescence. By contrast, a standard deviation unit increase in birth

TABLE 2. Risk ratios† for the influence of birth weight and length, body weight, and height and body mass index at different ages on bone fracture occurrence during growth, New Zealand, 1972/1973–1990/1991

	All fractures		Prepubertal fractures		Adolescent fractures	
	Risk ratio	95% CI‡	Risk ratio	95% CI	Risk ratio	95% CI
Birth weight	1.08	0.98, 1.20	1.07	0.90, 1.28	1.08	0.97, 1.21
Birth length	1.08	0.95, 1.22	1.28	1.04, 1.58	1.01*	0.88, 1.16
Weight at age 3 years	1.14	1.03, 1.27	1.27	1.06, 1.52	1.10	0.97, 1.23
Height at age 3 years	1.13	1.01, 1.26	1.35	1.11, 1.64	1.06*	0.94, 1.20
Weight from ages 5 to 18 years§	1.15	1.03, 1.28	1.37	1.12, 1.67	1.09*	0.96, 1.23
Height from ages 5 to 18 years§	1.13	1.02, 1.24	1.31	1.09, 1.57	1.08	0.96, 1.20
BMI‡ from ages 5 to 18 years§	1.01	0.98, 1.23	1.24	1.02, 1.52	1.05	0.92, 1.20

* Significantly different from the prepubertal group, $p < 0.05$.

† All risk ratios were adjusted for sex and age. Results are expressed per standard deviation unit increase.

‡ CI, confidence interval; BMI, body mass index.

§ Terms for the interaction between age and weight, height, and BMI were not statistically significant; thus, the estimate for the effect of weight or height on fracture risk was the same for each age.

length (prepubertal fractures only), weight and height at age 3 years, and weight and height from ages 5 to 18 years were all significantly associated with an increased risk of experiencing a fracture, and this association was stronger for prepubertal than for adolescent fractures. Raised body mass index increased the risk of prepubertal fracture only. We also looked at the difference in z score between birth weight and length and subsequent weight and height at 3 years. Neither

was significant (risk ratios for prepubertal fractures = 1.13, 95 percent confidence interval (CI): 0.96, 1.34 for weight and 1.08, 95 percent CI: 0.90, 1.28 for height).

Table 3 shows that elevated birth weight and birth length were not associated with an increased risk of sustaining either a wrist/forearm fracture alone or any other type of fracture; those who did not fracture were considered the reference group. However, elevated birth length (per stan-

TABLE 3. Odds ratios† for the influence of birth size, weight, and height at age 3 years and from ages 5 to 18 years and of body mass index from ages 5 to 18 years on the risk of wrist fracture or any other fracture, prepubertally or during adolescence, New Zealand, 1972/1973–1990/1991‡

	Wrist fractures only						Other fractures					
	All ages		Prepubertal fractures (16 girls, 25 boys)		Adolescent fractures (39 girls, 67 boys)		All ages		Prepubertal fractures (26 girls, 89 boys)		Adolescent fractures (148 girls, 212 boys)	
	Odds ratio	95% CI§	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI	Odds ratio	95% CI
Birth weight	1.09	0.91, 1.30	1.10	0.78, 1.54	1.08	0.88, 1.33	1.08	0.96, 1.22	1.06	0.87, 1.30	1.09	0.94, 1.26
Birth length	1.22	0.96, 1.55	1.45	0.91, 2.32	1.13	0.87, 1.46	1.06	0.92, 1.22	1.23	0.94, 1.61	0.99	0.84, 1.17
Weight at age 3 years	1.03	0.85, 1.26	1.18	0.81, 1.72	0.97	0.78, 1.22	1.21	1.05, 1.40	1.31	1.00, 1.71	1.17	0.99, 1.38
Height at age 3 years	1.11	0.90, 1.36	1.31	0.96, 1.81	1.03	0.80, 1.32	1.18	1.02, 1.35	1.38	1.01, 1.88	1.10	0.94, 1.27
Weight from ages 5 to 18 years¶	1.00	0.83, 1.21	1.41	0.93, 2.14	0.89	0.73, 1.09	1.23	1.08, 1.40	1.33	1.03, 1.71	1.19	1.02, 1.38
Height from ages 5 to 18 years¶	1.11	0.93, 1.33	1.29	0.91, 1.83	1.05	0.85, 1.29	1.13	0.99, 1.28	1.32	1.00, 1.75	1.06	0.92, 1.21
BMI from ages 5 to 18 years¶	0.93	0.77, 1.14	1.40*	0.96, 2.03	0.81	0.66, 1.01	1.20	1.05, 1.36	1.17	0.94, 1.45	1.20	1.02, 1.42

* Significantly different from the adolescent group, $p = 0.013$.

† All odds ratios were adjusted for sex and age.

‡ Results are expressed as per standard deviation unit increase.

§ CI, confidence interval.

¶ Terms for the interaction between age and weight, height, and body mass index (BMI) were not statistically significant; thus, the estimate for the effect of weight or height on fracture risk was the same for each age.

TABLE 4. Risk ratios* for the influence of maternal smoking during pregnancy and personal smoking on bone fracture occurrence during growth compared with nonsmokers, New Zealand, 1972/1973–1990/1991

	Maternal smoking during pregnancy		Participant smoking occasionally†		Participant smoking daily†	
	Risk ratio	95% CI‡	Risk ratio	95% CI	Risk ratio	95% CI
All fractures	1.15	0.93, 1.42	1.08	0.72, 1.61	1.43	1.05, 1.95
Wrist/forearm fractures only	1.24	0.84, 1.85	0.89	0.39, 2.03	0.90	0.43, 1.87
Prepubertal fractures	1.38	0.97, 1.98	NA‡		NA	
Adolescent fractures	1.07	0.84, 1.36	NA		NA	

* All risk ratios were adjusted for sex and age.

† Limited to fractures and smoking habits reported at ages 15 and 18 years.

‡ CI, confidence interval; NA, not applicable.

dard deviation unit) was associated with a nonsignificant increased risk of 45 percent for prepubertal wrist fractures. In addition, increased weight and height at age 3 years and from ages 5 to 18 years were associated with an increased risk of prepubertal wrist fracture (nonsignificant) and all other fractures prepubertally, which reached significance mostly in the “other fractures” category because of larger numbers in this group ($n = 26$ for girls and $n = 89$ for boys) compared with prepubertal wrist fractures ($n = 16$ for girls and $n = 25$ for boys). Body mass index from ages 5 to 18 years increased the risk of other fractures at all ages and in adolescence.

The risk ratios for maternal smoking during pregnancy and for personal daily smoking at ages 15 and 18 years are presented in table 4. A total of 258 (26.6 percent) mothers reported smoking during pregnancy, 485 mothers did not smoke, and smoking status was unavailable for 225 mothers. Maternal smoking was associated with a trend toward an increased risk of childhood fractures sustained throughout the 18 years of the study, particularly of the wrist/forearm and for fractures reported prepubertally. Adjusting for socioeconomic status did not alter the results appreciably (risk ratio for all fractures = 1.13, 95 percent CI: 0.91, 1.40). Analysis for personal smoking was related to 92 fractures reported in the girls and 194 fractures reported in the boys when the participants were aged 15 and 18 years (encompassing the period 13–18 years). At age 15 years, 105 study participants reported smoking daily and 106 reported smoking occasionally; at age 18 years, 261 study participants reported smoking daily and 98 reported smoking occasionally. Daily cigarette smoking was associated with a 43 percent increased risk of fracture, whereas occasional smoking was associated with only a nonsignificant 8 percent increase. The interaction effect between smoking and sex was not statistically significant, so fracture risk was affected similarly by cigarette smoking for both females and males.

In our cohort, breastfeeding (risk ratio = 1.04, 95 percent CI: 0.86, 1.23), increasing bone age (per standard deviation increase) (risk ratio = 1.04, 95 percent CI: 0.93, 1.57), or participation in physical activities for more than 1 hour a week (risk ratio = 0.76, 95 percent CI: 0.51, 1.12) was not associated with the risk of fracturing.

Adjusting for socioeconomic status and maternal smoking did not appreciably alter any of the risk ratios for fracture in

relation to height and weight (data not shown). No interaction effect was found between smoking and weight, although adjusting for weight increased the risks of fracture for smokers marginally. The risk ratios were 1.09 (95 percent CI: 0.73, 1.62) for occasional smoking and 1.46 (95 percent CI: 1.07, 1.99) for daily smoking.

Although babies born earlier than 37 weeks of gestation were excluded from the main analyses, all premature babies were compared with babies born at 37 weeks or later to determine whether children or adolescents born prematurely were at greater risk of fracture compared with children born at term. Forty-one study participants were born at less than 37 weeks of gestation, with 20 subjects from this group reporting 32 fractures. However, the relative risk of fracturing compared with those born at term was 1.16 (95 percent CI: 0.75, 1.81).

DISCUSSION

To our knowledge, this study is the first to establish in a birth cohort that children who were born long or were heavy and tall throughout growth had an increased risk of breaking bones during their childhood and adolescence. Risk was also increased for adolescents who were daily smokers. Conversely, birth weight, maternal smoking during pregnancy, breastfeeding, and sports participation did not increase the risk of fracture between birth and 18 years of age.

The fetal-origin-of-disease hypothesis has suggested that modifications to the perinatal environment that result in low birth weight and/or reduced weight early in infancy may negatively contribute to the risk of various diseases in later life (30). Cooper et al. (31) have suggested that osteoporosis may be a disease programmed during early life. It has been shown that birth weight, birth length, and length gain in the first month of life contribute significantly to bone mineral density in children 8 years of age (32). Bone mineral content at the lumbar spine and femoral neck of women aged 21 years is also associated with weight at 1 year of age (9). Similarly, significant relations have been found between birth weight, weight at age 1 year, and bone area and bone mineral content in older adults (33, 34). Furthermore, a low rate of growth between 7 and 15 years of age has also been

shown to be associated with an increased risk of hip fracture late in life (35).

Because bone mineral density and bone mineral content were not measured in our study, we cannot report the effect of birth size or later growth variables on bone mass, size, or density in our cohort. However, to our knowledge, until now no one has ascertained whether perinatal or childhood growth influences the risk of sustaining fractures in childhood and adolescence. Our results show that while birth weight did not have a significant effect on the risk of fracturing during growth, birth length was associated with a 28 percent increase in risk for all prepubertal fractures for every standard deviation unit increase. Although low numbers of prepubertal wrist fractures limited statistical power, birth length tended to be associated with an increase in risk for prepubertal wrist fractures. This finding may reflect a propensity for girls and boys to fracture a wrist before age 9 and before age 11 years, respectively, when height is achieved early.

Greater weight and height, both at age 3 years and from ages 5 to 18 years, were each associated with an increased risk of fracturing. Our results strongly suggest that youngsters who are heavy and/or tall have an increased risk of fracture prepubertally. Children who gain height quickly and are tall early presumably have a higher tempo of growth resulting in a greater rate of bone remodeling, which may increase their bone fragility (36) and susceptibility to fracture. Such children must absorb and retain the nutrients needed to build bone in a shorter time than children who grow more slowly. This requirement may place higher nutritional demands on them (37). In addition, a disassociation between body size and bone development exists such that, by the second year of life, approximately half of final stature has been achieved while only 10 percent of maximal skeletal weight has been attained (9). By age 7 years, 80 percent of predicted adult height has been achieved but bone mineral content is approximately only 40 percent (38). For children who grow tall quickly and early, this discrepancy of bone mass to body mass may be magnified, resulting in a temporary bone density deficit resembling that reported at the pubertal growth spurt (39). Similarly, for children who are heavy and are carrying excess fat mass, this discrepancy may be magnified, conferring a biomechanical disadvantage, which may increase their risk of fracture (14, 40, 41). Children with a high body mass will also fall more heavily than lighter children. Other potential explanations include poor coordination or balance difficulties for larger children, leading to a greater propensity to fall (42). Bigger children may be encouraged to participate in sports with older children of the same size who may be skeletally stronger, which could increase their risk of injury and fracture of their more immature bones.

Our group has previously found, in both boys and girls with recent distal forearm fractures, that more were overweight or obese when compared with fracture-free controls (10, 11) and, for girls, that high body weight is an independent predictor of new fractures at any site over 4 years (14). Others have also found that overweight is associated with forearm fractures in childhood (17). In contrast, Ma and Jones (13, 18) did not find an increase in body weight in chil-

dren with fractures compared with those who did not report a fracture.

Three possible explanations may account for the weak associations seen between height and weight and adolescent fractures. First, overweight children may reduce their participation in sports as they move into adolescence, resulting in fewer fractures. Second, there may be an increase in the number of reported fractures unrelated to body size, such as those of the fingers and toes, because of greater involvement in sports or other risk-taking behavior. However, we found that participation in physical activity did not influence fracture risk, perhaps because physical activity can be both beneficial for bone strength through increased loading (8) while increased exposure to events likely to lead to fracture may elevate fracture risk. Third, bone mineralizes and strengthens rapidly during puberty (38, 43, 44), and, as the skeleton nears its adult strength, the discrepancy between body size and bone strength minimizes so that fewer fractures result from elevated height and high body weight.

Maternal smoking during pregnancy has been associated with reduced bone mineral density in prepubertal Tasmanian children (6), although not with an increased risk of fractures (18). In our larger cohort, we observed a trend toward an increased risk, particularly for prepubertal fractures (38 percent), although the risk did not reach statistical significance. Our information concerning maternal smoking during pregnancy was ascertained retrospectively, when the study participants were aged 9 years. Tomeo et al. (45) found that mothers appear to remember pregnancy-related issues including smoking satisfactorily, and results from other studies suggest that self-reported smoking is reasonably accurate (46). However, some mothers who did smoke during pregnancy may deny doing so, as others have found (47). We recognize that maternal smoking during pregnancy may be a marker of other factors that affect the postnatal or early-life environment of children whose mothers are likely to have smoked during pregnancy.

We found that daily smoking by adolescents (aged 13–18 years) raised their risk of fracturing substantially. Likewise, older adults are at a greater risk of fracture if they smoke, although evidence for an effect of smoking on increasing fracture risk during young adulthood to middle age is more ambiguous (48). Increased fracture risk for older adults who smoke has been attributed to low bone density. Additionally, there may be decreased intestinal calcium absorption because of low serum calcitriol levels (49). Although our evaluation of smoking was simple (daily, occasional, or never) and we did not take into account number of cigarettes smoked daily, we found a strong effect of daily smoking on increasing fracture risk. Our study did not identify the mechanism by which cigarette smoking increases the risk of fracturing in adolescents, although reduced bone density probably plays a role (50). Alternatively, this increased risk of fractures may not be an effect of smoking per se but rather an unidentified behavior that typifies regular adolescent smokers. However, those teenagers who smoked daily in adolescence did not have an increased risk of fractures before age 13 years (data not shown), suggesting that not the characteristics of the smoker but rather the smoking itself raises the risk of fracture. Additionally, in this cohort,

smoking in adolescence did not predict serious injury or traffic crashes in adults aged 21 years (51). Unfortunately, the prevalence of smoking may be increasing in New Zealand teenagers (52), and the long-term effects of smoking as an adolescent on bone health are not known, particularly since many teenage smokers continue smoking into adulthood. Encouragingly, for adult former smokers, the negative skeletal effects of cigarette smoking appear to wane (53, 54).

Low bone mineralization has been reported in children born preterm who tend to be small (55–58). In our study, a trend toward increased fracture risk was observed for children born preterm, although this risk was not statistically significant; it is possible that their smaller body size was protective. Bowden et al. (57) found that preterm children aged 8 years were shorter and lighter and had fewer accidental fractures compared with term controls. Our present analysis lacked statistical power because of the small number of preterm children in our cohort.

A major strength of our study is that participants reported fractures at regular intervals throughout growth, which minimized recall bias. Excellent follow-up rates over the 18 years of the study were also achieved (22). Bone mineral density measurements were not available for cohort members because our study began before the advent of dual energy x-ray absorptiometry. Thus, anthropometric variables or the fracture rate could not be linked to the presence of low or high bone mineral density. We also acknowledge that the present analysis was limited to examining the effects of anthropometry, breastfeeding, sports participation, and smoking on fracture risk. Other potential risk factors for fracture in this age group exist.

To conclude, fractures in children are an important public health problem (59) warranting further study, since more than half of all children experience at least one fracture before the age of 18 years (1). Some evidence exists that the fracture incidence in childhood is increasing in some populations (60, 61) but not all (62). This paper identified high body weight and height as risk factors for fracture, particularly during the prepubertal years, a major concern given the rising rates of obesity at a young age (63, 64). In addition, teenage smoking is a strong risk factor for fracture during the adolescent years, giving further impetus to public health messages to curb teenage smoking. Strategies to reduce the number of fractures in youngsters should include reducing the prevalence of overweight and obesity in children and implementing campaigns to reduce the prevalence of smoking in teenagers. These interventions also have the potential to help prevent the development of many other chronic diseases.

ACKNOWLEDGMENTS

The authors thank the Health Research Council of New Zealand for current grant support. They also thank Dr. Richie Poulton, Director of the Dunedin Multidisciplinary Health and Development Research Unit and the Injury Prevention Unit, for permission to use the data. The Dunedin Multidisciplinary Health and Development Study was initi-

ated by Dr. Phil A. Silva, and the research unit is supported by the Health Research Council of New Zealand. Data collection was partially supported by grant MH-45070 from the US National Institute of Mental Health.

REFERENCES

1. Jones IE, Williams SM, Dow N, et al. How many children remain fracture-free during growth? A longitudinal study of children and adolescents participating in the Dunedin Multidisciplinary Health and Development Study. *Osteoporos Int* 2002; 13:990–5.
2. Kypril K, Chalmers DJ, Langley JD, et al. Child injury morbidity in New Zealand, 1987–1996. *J Paediatr Child Health* 2001; 37:227–34.
3. Fife D. Northeastern Ohio Trauma Study III: incidence of fractures. *Ann Emerg Med* 1985;14:244–8.
4. Donaldson LJ, Cook A, Thomson RG. Incidence of fractures in a geographically defined population. *J Epidemiol Community Health* 1990;44:241–5.
5. Johansen A, Evans RJ, Stone MD, et al. Fracture incidence in England and Wales: a study based on the population of Cardiff. *Injury* 1997;28:655–60.
6. Jones G, Riley M, Dwyer T. Maternal smoking during pregnancy, growth and bone mass in prepubertal children. *J Bone Miner Res* 1999;14:146–51.
7. Jones G, Riley M, Dwyer T. Breastfeeding in early life and bone mass in prepubertal children: a longitudinal study. *Osteoporos Int* 2000;11:146–52.
8. French SA, Fulkerson JA, Story M. Increasing weight-bearing physical activity and calcium intake for bone mass growth in children and adolescents: a review of intervention trials. *Prev Med* 2000;31:722–31.
9. Cooper C, Cawley M, Bhalla A, et al. Childhood growth, physical activity, and peak bone mass in women. *J Bone Miner Res* 1995;10:940–7.
10. Goulding A, Cannan R, Williams SM, et al. Bone mineral density in girls with forearm fractures. *J Bone Miner Res* 1998;13:143–8.
11. Goulding A, Jones IE, Taylor RW, et al. Bone mineral density and body composition in boys with distal forearm fractures: a dual-energy x-ray absorptiometry study. *J Pediatr* 2001;139: 509–15.
12. Marshall D, Johnell O, Wedel H. Meta-analysis of how well measures of bone mineral density predict occurrence of osteoporotic fractures. *BMJ* 1996;312:1254–9.
13. Ma D, Jones G. The association between bone mineral density, metacarpal morphometry, and upper limb fractures in children: a population-based case-control study. *J Clin Endocrinol Metab* 2003;88:1486–91.
14. Goulding A, Jones IE, Taylor RW, et al. More broken bones: a 4-year double cohort study of young girls with and without distal forearm fractures. *J Bone Miner Res* 2000;15:2011–18.
15. Klotzbuecher C, Ross P, Landsman P, et al. Patients with prior fractures have an increased risk of future fractures: a summary of the literature and statistical synthesis. *J Bone Miner Res* 2000;15:721–39.
16. Reid IR. Relationships among body mass, its components, and bone. *Bone* 2002;31:547–55.
17. Skaggs DL, Loro ML, Pitukcheewanont P, et al. Increased body weight and decreased radial cross-sectional dimensions in girls with forearm fractures. *J Bone Miner Res* 2001;16:1337–42.
18. Ma DQ, Jones G. Clinical risk factors but not bone density are

- associated with prevalent fractures in prepubertal children. *J Paediatr Child Health* 2002;38:497–500.
19. Black RE, Williams SM, Jones IE, et al. Children who avoid drinking cow milk have low dietary calcium intakes and poor bone health. *Am J Clin Nutr* 2002;76:675–80.
 20. Wyshak G. Teenaged girls, carbonated beverage consumption, and bone fractures. *Arch Pediatr Adolesc Med* 2000;154:610–13.
 21. Williams SM. Overweight at age 21: the association with body mass index in childhood and adolescence and parents' body mass index. A cohort study of New Zealanders born in 1972–1973. *Int J Obes* 2001;25:158–63.
 22. Silva PA, Stanton WR. From child to adult. The Dunedin Multidisciplinary Health and Development Study. Auckland, New Zealand: Oxford University Press, 1996.
 23. Buckfield P. Perinatal events in the Dunedin City population 1967–1973. *N Z Med J* 1978;88:244–6.
 24. Kuczumski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. *Adv Data* 2000;314:1–27.
 25. Abbassi V. Growth and normal puberty. *Pediatrics* 1998;102(suppl 2):507–11.
 26. Birkbeck J, Herbet C. Skeletal maturity in seven year old Dunedin children. *N Z Med J* 1980;92:312–13.
 27. Stanton WR, Silva PA, Oei TPS. Prevalence of smoking in a Dunedin sample followed from age 9 to 15 years. *N Z Med J* 1989;102:637–9.
 28. Elley J, Irving J. A socioeconomic index for New Zealand based on levels of education and income from 1966 census. *N Z J Educat Studies* 1972;7:153–67.
 29. Huber PJ. Robust statistics. New York, NY: John Wiley & Sons, 1981.
 30. Barker DJ. Fetal and infant origins of adult disease. London, United Kingdom: BMJ Publishing Group, 1992.
 31. Cooper C, Javaid MK, Taylor P, et al. The fetal origins of osteoporotic fracture. *Calcif Tissue Int* 2002;70:391–4.
 32. Jones G, Dwyer T. Birth weight, birth length, and bone density in prepubertal children: evidence for an association that may be mediated by genetic factors. *Calcif Tissue Int* 2000;67:304–8.
 33. Cooper C, Fall C, Egger P, et al. Growth in infancy and bone mass in later life. *Ann Rheum Dis* 1997;56:17–21.
 34. Gale CR, Martyn CN, Kellingray S, et al. Intrauterine programming of adult body composition. *J Clin Endocrinol Metab* 2001;86:267–72.
 35. Cooper C, Eriksson JG, Forsen T, et al. Maternal height, childhood growth and risk of hip fracture in later life: a longitudinal study. *Osteoporos Int* 2001;12:623–9.
 36. Slemenda CW, Peacock M, Hui S, et al. Reduced rates of skeletal remodeling are associated with increased bone mineral density during the development of peak skeletal mass. *J Bone Miner Res* 1997;12:676–82.
 37. Parfitt AM. The two faces of growth: benefits and risks to bone integrity. *Osteoporos Int* 1994;4:382–98.
 38. Bass S, Delmas PD, Pearce G, et al. The differing tempo of growth in bone size, mass, and density in girls is region-specific. *J Clin Invest* 1999;104:795–804.
 39. Fournier PE, Rizzoli R, Slosman DO, et al. Asynchrony between the rates of standing height gain and bone mass accumulation during puberty. *Osteoporos Int* 1997;7:525–32.
 40. Goulding A, Taylor RW, Jones IE, et al. Overweight and obese children have low bone mass and area for their weight. *Int J Obes* 2000;24:627–32.
 41. Davidson PL, Goulding A, Chalmers DJ. Biomechanical analysis of arm fracture in obese boys. *J Paediatr Child Health* 2003;39:657–64.
 42. Goulding A, Jones IE, Taylor RW, et al. Dynamic and static tests of balance and postural sway in boys: effects of previous wrist bone fractures and high adiposity. *Gait Posture* 2003;17:136–41.
 43. Bonjour JP, Theintz G, Buchs B, et al. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. *J Clin Endocrinol Metab* 1991;73:555–63.
 44. Heaney RP, Abrams S, Dawson-Hughes B, et al. Peak bone mass. *Osteoporos Int* 2000;11:985–1009.
 45. Tomeo CA, Rich-Edwards JW, Michels KB, et al. Reproducibility and validity of maternal recall of pregnancy-related events. *Epidemiology* 1999;10:774–7.
 46. Patrick DL, Cheadle A, Thompson DC, et al. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health* 1994;84:1086–93.
 47. von Kries R, Toschke AM, Koletzko B, et al. Maternal smoking during pregnancy and childhood obesity. *Am J Epidemiol* 2002;156:954–61.
 48. Jesudason D, Need AG. Effects of smoking on bone and mineral metabolism. *Endocrinologist* 2002;12:199–209.
 49. Brot C, Jorgensen NR, Sorensen OH. The influence of smoking on vitamin D status and calcium metabolism. *Eur J Clin Nutr* 1999;53:920–6.
 50. Valimaki MJ, Karkkainen M, Lamberg-Allardt C, et al. Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. *Cardiovascular Risk in Young Finns Study Group. BMJ* 1994;309:230–5.
 51. Begg DJ, Langley JD, Williams SM. A longitudinal study of lifestyle factors as predictors of injuries and crashes among young adults. *Accid Anal Prev* 1999;31:1–11.
 52. Reeder AI, Williams S, McGee R, et al. Tobacco smoking among fourth form school students in Wellington, New Zealand, 1991–97. *Aust N Z J Public Health* 1999;23:494–500.
 53. Ward KD, Klesges RC. A meta-analysis of the effects of cigarette smoking on bone mineral density. *Calcif Tissue Int* 2001;68:259–70.
 54. Gerdhem P, Obrant KJ. Effects of cigarette-smoking on bone mass as assessed by dual-energy x-ray absorptiometry and ultrasound. *Osteoporos Int* 2002;13:932–6.
 55. Zamora SA, Belli DC, Rizzoli R, et al. Lower femoral neck bone mineral density in prepubertal former preterm girls. *Bone* 2001;29:424–7.
 56. Specker BL, Johannsen N, Binkley T, et al. Total body bone mineral content and tibial cortical bone measures in preschool children. *J Bone Miner Res* 2001;16:2298–305.
 57. Bowden LS, Jones CJ, Ryan SW. Bone mineralisation in ex-preterm infants aged 8 years. *Eur J Pediatr* 1999;158:658–61.
 58. Fewtrell MS, Prentice A, Jones SC, et al. Bone mineralization and turnover in preterm infants at 8–12 years of age: the effect of early diet. *J Bone Miner Res* 1999;14:810–20.
 59. Jones G, Cooley HM. Symptomatic fracture incidence in those under 50 years of age in southern Tasmania. *J Paediatr Child Health* 2002;38:278–83.
 60. Hagino H, Yamamoto K, Ohshiro H, et al. Increasing incidence of distal radius fractures in Japanese children and adolescents. *J Orthop Sci* 2000;5:356–60.
 61. Khosla S, Melton LJ, Dekutoski MB, et al. Incidence of childhood distal forearm fractures over 30 years. A population-based study. *JAMA* 2003;290:1479–85.
 62. Tiderius CJ, Landin LA, Duppe H. Decreasing incidence of fractures in children—an epidemiological analysis of 1,673 fractures in Malmo, Sweden, 1993–1994. *Acta Orthop Scand* 1999;70:622–6.
 63. Deckelbaum RJ, Williams CL. Childhood obesity: the health issue. *Obes Res* 2001;9(suppl 4):239–43.
 64. Booth ML, Chey T, Wake M, et al. Change in the prevalence of overweight and obesity among young Australians, 1969–1997. *Am J Clin Nutr* 2003;77:29–36.