# YOUTH

## *A Health Plan–Based Lifestyle Intervention Increases Bone Mineral Density in Adolescent Girls*

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**Objective:** To test the efficacy of a health plan–based lifestyle intervention to increase bone mineral density in adolescent girls.

**Design:** Two-year randomized, controlled trial.

**Setting:** Large health maintenance organization.

**Participants:** Girls 14 to 16 years old with body mass index below the national median.

**Intervention:** Behavioral intervention (bimonthly group meetings, quarterly coaching telephone calls, and weekly self-monitoring) designed to improve diet and increase physical activity.

**Main Outcome Measures:** Total bone mineral density was measured by dual-energy x-ray absorptiometry. Behavioral outcomes included intake of calcium, vitamin D, soda, and fruits and vegetables; high-impact and strength-training physical activity; measures of strength and fitness; and biomarkers (osteocalcin and naltrexone).

**Results:** Compared with control subjects, girls in the intervention group had significantly higher bone mineral density in the spine and trochanter regions during the first study year, which was maintained during the second study year. The naltrexone biomarker demonstrated a greater relative decrease in the intervention group compared with the control group, with nonsignificant changes in osteocalcin consistent with more bone building in the intervention group. Participants in the intervention group reported significantly greater consumption of calcium in both study years, vitamin D in the first year, and fruits and vegetables in both years. We found no effect on soda consumption or target exercise rates.

**Conclusions:** A comprehensive health care–based lifestyle intervention can effectively improve dietary intake and increase bone mineral gains in adolescent girls. To our knowledge, this study is the first to significantly improve bone mass in adolescent girls in a non–school-based intervention.

**Trial Registration:** Clinicaltrials.gov Identifier: NCT00067600.

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THE PRIMARY PREVENTION OF osteoporosis is an important public health target. Almost half of all women in the United States older than 50 years demonstrate low bone density (osteopenia).[1] An estimated 1.3 million osteoporosis-related fractures occur each year in the United States, with annual costs of approximately $13.8 billion.[2] One determinant of lifetime osteopenia and osteoporosis risk is low bone mineral density (BMD). Because 90% of peak bone mass is acquired by age 18 years,[3-5] interventions to maximize BMD in youth may decrease the incidence of osteopenia later in life. For this reason, the National Institute of Child Health and Health Development recently requested applications (RFA: HD-97-006) for the prevention of adult osteoporosis by targeting BMD in youth.

Although a substantial component of osteoporosis risk is genetic,[6,7] both diet and physical activity are important modifiers of bone accrual.[3] Several controlled trials have found that increasing calcium intake increases BMD in youth.[8-10] Other dietary factors also may maximize the retention of calcium in bones, but few randomized trials have examined these factors in adolescents. Studies suggest that greater fruit and vegetable intake is important for bone health[11,12] and is associated with higher BMD.[13,14] In addition, studies[15,16] suggest that achievement of peak bone mass in adolescent girls is contingent on adequate vitamin D intake. Further, consuming caffeinated beverages, particularly colas, increases risk of bone fracture.[17,18] Finally, many studies have suggested that increasing weight-bearing activity increases BMD in children and adolescents.[19-24]

**Table 1. Study Intervention and Adherence Components**

| Component | Enhanced Fitness (Intervention) | Total Health (Comparison) |
|---|---|---|
| **Intervention** | | |
| Re-treat, to create group cohesion among study participants and orient to study* | 1 Time at study beginning | 1 Time at study beginning |
| Annual individual visits to provide individualized feedback and motivation* | Annually | Annually |
| Coaching calls to address individual adherence issues | 4 Times per year | NA |
| Team meetings: information and group support | Bimonthly | NA |
| Self-monitoring postcards with behavioral targets shifting weekly | Weekly | NA |
| **Adherence and retention** | | |
| Study Web site: information and monitoring own point accumulation* | Ongoing | Ongoing |
| Team meetings: social interaction and parental involvement | Quarterly | Quarterly |
| Study incentives/points | Ongoing | Ongoing |
| Youth and parent newsletter* | Twice yearly | Twice yearly |
| Participant membership in fitness center | Ongoing | Ongoing |

Abbreviation: NA, not available.
*Content differs for intervention and comparison conditions; intervention focused on diet and exercise, whereas comparison focused on other health issues.

Although much of the research on building healthy bones in youth has targeted younger children,[19,21,23,25,26] adolescents may be an equally important target population. Eating and exercise patterns established in adolescence may be more likely to be sustained into adulthood than similar efforts aimed at younger children.[27,28] Gains in bone mass are most rapid during adolescence, with as much as 51% of peak bone mass accumulated during pubertal growth.[29,30] Interventions to prevent osteoporosis are particularly important in adolescent girls, because they are at a higher risk of developing osteoporosis in adulthood than males.[31] Recent reports suggest that vigorous exercise declines in adolescents,[32] which makes this time key for intervention.

Preventive interventions conducted in youth generally have involved calcium supplementation, controlled feeding trials, or prescribed exercise in a controlled setting; that is, they have not emphasized sustainable behavioral practices and, thus, not represented community trials. Further, existing youth interventions are mainly school based,[33-36] largely overlooking the opportunities in other settings such as health care. Inasmuch as most children and adolescents (about 80%) visit a medical provider at least annually (76 million annual contacts with physicians[37]), such visits are a largely untapped setting in which to offer primary prevention programs. Pediatric patients are influenced by physician advice and are receptive to health behavior recommendations.[38] Thus, adolescents may comply with targeted lifestyle interventions offered through health care settings more than with those offered in schools.

## METHODS

### OVERVIEW

This randomized controlled trial (YOUTH) tested the efficacy of a lifestyle intervention for increasing BMD in adolescent girls initially 14 to 16 years old. The goal of the intervention was to improve diet and increase physical activity. The 3 dietary targets were increasing dairy consumption, eating 8 servings of fruits and vegetables daily, and decreasing soft drink intake. The 2 primary physical activity targets were high-impact exercise and strength training.

### SETTING

Kaiser Permanente Northwest is a nonprofit, group-model health maintenance organization (HMO) in the Portland, Ore, metropolitan region that provides comprehensive medical care to more than 440 000 members, including 15 768 female adolescents between 14 and 16 years of age. The research center is located within the HMO but conducts independent, public domain research. The HMO Human Subjects Protection Committee monitored and approved all study procedures.

### STUDY POPULATION AND RECRUITMENT, SCREENING, AND RANDOMIZATION

We selected adolescent girls with body mass index below the national median to enrich our sample with girls at risk of low peak BMD.[39,40] We also targeted potential participants by selecting for characteristics we expected would enhance adherence to the study (ie, younger girls [freshmen and sophomores], parent or guardian willing to participate in the study, and no indication of psychiatric or psychosocial disorders). We excluded potential participants with any apparent contraindication to the dietary or exercise portions of the intervention, including current or past disordered eating behavior. Potential participants were identified through the HMO's electronic medical record. Health plan member contracts with the HMO provide consent for use of their data in research. Members who met the selection criteria were mailed study invitations, followed by telephone calls from research staff. An informational meeting for interested families meeting study criteria preceded randomization. Eligible adolescent girls were randomized by a computer program developed by one of us (M.A.) into either the lifestyle intervention group or an attentional control group after baseline data collection (between September 1, 2000, and August 31, 2001). The project manager informed participants of group assignment to keep assessors blinded. Treatment group assignment was made by a design-adaptive randomization to minimize group imbalance on physical activity, calcium intake, age, and other factors.[41,42] Design-adaptive randomization sequentially assigned girls to the control or intervention groups to achieve, at each step, the maximum balance of factors predictive of bone measurements, such as menarcheal age and participation in organized sports. To conceal allocation, the project biostatistician (M.A.) made allocations in response to project staff requests.

### INTERVENTION

The YOUTH intervention emphasized adolescents actively developing strategies for healthy dietary and exercise practices that they could maintain in adulthood. Participants attended group

and individual meetings, participated in activities, and received coaching telephone calls (**Table 1**). They also received psychoeducational information, recorded their diet and exercise goals and achievements, and kept in touch with their cohort through a Web-based study site. We combined elements especially for adolescents (peer-oriented, community-building activities) with those widely recognized as important in lifestyle interventions (individual tailoring). Further, our intervention drew heavily from both mentoring models and motivational interviewing or enhancement techniques.[43] Because this trial involved a 2-year intervention and follow-up, we incorporated several adherence and retention components (Table 1). The intervention has been extensively described elsewhere.[39]

## ASSESSMENT

Staff who performed clinical and dietary and physical activity assessments were masked to the experimental condition of the participants. These assessors had no additional contact with participants.

## BONE MINERAL DENSITY

Bone mineral density was measured using dual-energy x-ray absorptiometry (DEXA; QDR 2000, Hologic Inc, Waltham, Mass) at baseline and at 1- and 2-year follow-up. We assessed BMD and bone mineral content (BMC) for the total body and at specific sites: lumbar spine (L2 through L4), trochanter, femoral neck, and total hip. Independently determined in vivo precision (coefficient of variation) for total hip and lumbar spine in our laboratory were 1.4% and 1.7%, respectively. Phantom scans performed daily during the observation period revealed no change in DEXA machine performance. Our adolescent girls had mostly completed linear growth, and we anticipated little change in bone dimensions during follow-up. Thus, BMD was the primary outcome measure. Nevertheless, we also measured BMC, bone mineral apparent density (BMAD), and bone area. Because volumetric density (BMAD) is difficult to estimate using DEXA, we limited BMAD results to L2 through L4, for which there are established reference ranges for teenagers.[44]

## BODY COMPOSITION AND PHYSICAL DEVELOPMENT

Certified technicians measured weight and height using a standardized protocol.[45] Body mass index was calculated as weight in kilograms divided by height in meters squared. The total-body scan (DEXA) was used to measure lean and fat masses. We used years since menarche at baseline as our measure of sexual maturation because[46] our minimum age requirement (14 years) meant that most subjects (97%) had reached menarche. Month and year of menarche was updated at every diet recall for those who had not reached menarche at baseline.

## BIOMARKERS

We collected blood samples from participants at the beginning and end of the study to examine biochemical markers of bone formation (osteocalcin; Diagnostic Products Corp, Los Angeles, Calif) and bone resorption (*N*-terminal telopeptides; Ostex International, Inc, Seattle, Wash). Blood samples were drawn in the morning after overnight fasting and handled and assayed according to the manufacturer's specifications.

## DIETARY INTAKE

Certified dietary interviewers used unannounced 24-hour telephone diet recalls to obtain data on all foods consumed, preparation method, and portion sizes. Participants were trained to estimate portion size using real food and food models at the screening visit, and received visual aids for estimating portion size of various foods. At baseline, we obtained data from 3 unannounced diet recalls for a 2-week period. Postrandomization, 1 recall was obtained every other month, targeting 4 weekdays and 2 weekend days per year to cover seasonal effects. The 6 dietary recalls in each year were averaged for analysis. Data were directly entered into the ESHA database (ESHA Food Processor, version 8.1, 2003; ESHA Research Inc, Salem, Ore). We limited the nutrient variables to the food group–based nutrition categories potentially relevant for bone mineral accrual: total calcium intake, in milligrams per day; total vitamin D, in international units per day; and fruits and vegetables, in servings per day. In addition, we adapted the ESHA program to output soda intake, in ounces per day, and vitamin supplementation.

## WEIGHT-BEARING PHYSICAL ACTIVITY, STRENGTH, AND FITNESS

We used both laboratory and self-reported measures to assess weight-bearing physical activity, strength, and fitness. To determine weight-bearing physical activity, we adapted a 72-hour physical activity recall from the Previous Day Physical Activity Recall form[47,48] and administered it like the dietary recalls. Because we examined activity most relevant for bone mineral accrual (high impact, spinal motion, and weight-loading activities), physical activity recall focused on exercise rather than usual daily activities or sedentary behaviors. We defined "high impact" as movement in which both feet were simultaneously off the ground (eg, jumping or running) and "strength training" as any activity that provided muscular resistance (eg, weight training and resistance band use).

Strength and fitness were assessed at the 3 annual clinic visits using standardized protocols and trained assessors. Assessments included hand grip (overall strength), Roman chair and sit-ups (lower back strength), and vertical jump (hip and upper thigh strength). We used sit-ups and vertical jump as representative strength measures.

## OTHER STUDY MEASURES

At baseline and follow-up visits, girls completed questionnaires about potential moderators and mediators of outcomes. Only the demographic characteristics and the participant's osteoporosis risk are included here. We defined "adult osteoporosis risk" as the proportion of first- and second-degree relatives of the participant's parents (eg, their parents, aunts, and uncles) whom the parents identified as having hip fractures or osteoporosis. Teen participants also reported their self-perceived risk of osteoporosis later in life.

## ANALYSIS

Statistical analyses were conducted using SAS Release 8.2 (SAS Institute Inc, Cary, NC) and STATA version 6.0 (StataCorp, College Station, Tex). Bone mineral density was our primary dependent variable, and the intervention effect was estimated as the adjusted (for baseline values) mean difference between the intervention and control conditions after years 1 and 2. We used a conditional change model and the Zellner seemingly unrelated regression models.[49-51] This approach uses joint estimates of several regression models. Baseline and change equations were estimated simultaneously because we expected that the 2 equations were not independent. Adjusting for the correlated errors generally leads to more efficient estimates of the

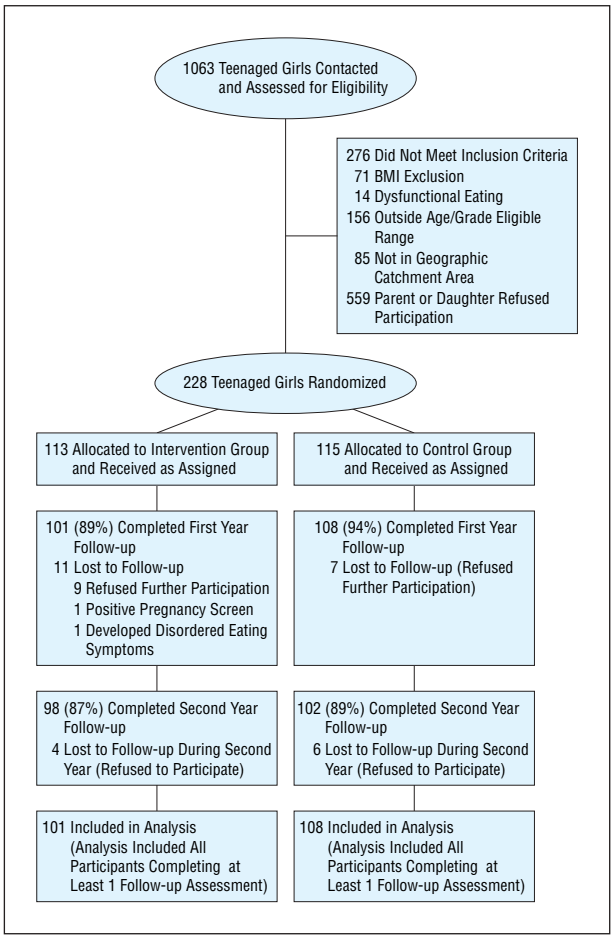


**Figure 1.** Consolidated Standards of Reporting Trials (CONSORT) diagram. Participant flow through the clinical trial. BMI, body mass index (calculated as weight in kilograms divided by height in meters squared).

coefficients and reduced standard errors in both equations than would result from the use of separate equation estimations. Treatment condition was the primary independent variable. All analyses were adjusted for baseline age, years since menarche, risk of adult osteoporosis, height, body mass index, and the respective bone mass variable. We analyzed the intervention's effects on bone mineral over the initial year of the intervention and across the entire 2-year period. All significance tests were 2-sided.

We used the same regression approach for secondary outcomes: changes in diet and physical activity. In addition, we examined behavioral (overall energy intake and overall physical activity) and anthropometric factors (weight, height, body mass index, and lean and fat masses) that were not targeted for behavioral change.

Of the 1063 girls originally contacted, 228 met the inclusion criteria, agreed to participate, and were randomized to either the intervention or the control group (**Figure 1**). Of those randomized, 210 (92%) underwent at least 1 bone mineral follow-up test. For those with missing values for the first-year follow-up DEXA measurement (n=8), the data were imputed by averaging baseline and second-year DEXA values. One girl was excluded after a positive pregnancy screening, bringing the 1-year outcome analysis sample to 209. Two hundred girls had DEXA data at the 2-year follow-up; the sustainability analysis was limited to these girls. Blood was drawn in all girls for biomarker analyses. In a laboratory error, a box of samples was lost; all remaining paired samples (n=130) were analyzed. We repeated all nonblood analyses with this biomarker subsample; patterns (direction and significance of results) were comparable to those of the entire sample (data not shown).

## RESULTS

### DESCRIPTIVE CHARACTERISTICS

Baseline values for study participants (**Table 2**) showed they were mainly white (81%) and from middle- to upper-middle-income working homes. The participants were in the lower half of the body mass index distribution (20.6) per the selection criteria, and 97% had reached menarche at enrollment. At baseline, average consumption included 986 mg/d of calcium, 161 IU of vitamin D, 3.6 servings of fruits and vegetables, and less than 6 oz per day of soda. At baseline, total physical activity was 61.9 min/d (including 13.9 min/d of high-impact activity and 6.9 min/d of strength training), with 68.9% of participants reporting participation in organized team sports. No statistically significant differences were found for these variables between the intervention and control groups at baseline.

### INTERVENTION EFFECTS ON MAIN DIET AND EXERCISE TARGETS

The intervention had a substantial effect on the 3 main dietary targets but not on exercise (Table 2). Participants in the intervention group reported significantly higher consumption compared with those in the control group for calcium in both study years (adjusted mean difference [AMD], 216.6 and 241.3 mg, respectively; $P<.001$), vitamin D in the first year of the study (AMD, 34.3 IU; $P=.02$), and fruit and vegetable servings in both study years (AMD, 0.74 and 0.79 servings, respectively; $P\leq.01$). We found no effect of the intervention on soda consumption or significant differences between the conditions in target exercise rates during either year.

### INTERVENTION EFFECTS ON BONE MINERAL VARIABLES AND MARKERS OF BONE TURNOVER

Significantly higher BMD was found in the intervention group compared with the control group in the spine (AMD, 0.01; $P<.001$) and trochanter region (AMD, 0.007; $P=.05$) and a trend toward higher density in the total hip (AMD, 0.006; $P=.08$) after 1 year of intervention (**Table 3** and **Figure 2**). We found no significant differences between the groups for BMD for the total body or the femoral neck region or for bone area or BMC for any of the bone regions. The 2 groups differed in spinal BMAD at the year 1 follow-up (AMD, 0.01; $P=.001$).

Data in Table 3 and Figure 2 suggest that the intervention effects on BMD in the spine (AMD, 0.01; $P=.007$) and trochanter region (AMD, 0.01; $P=.03$) were maintained during the second study year. During the second year, we observed no differences in bone areas in the 2 groups but significantly higher levels of BMC for the total body (AMD, 19.78; $P=.43$) and spine (AMD, 7.09; $P=.03$) in the intervention group compared with the control group. Also, the 2 groups differed in spinal BMAD at the year 2 follow-up (AMD, 0.01; $P=.02$). In addition, the

**Table 2. Baseline to Year 1 and Year 2 Intervention Outcomes: Behavioral and Other Intermediary Factors***

| Factor | Control Group (n = 108) | | | Intervention Group (n = 101) | | | *P* Value | |
|---|---|---|---|---|---|---|---|---|
| | Baseline Value | Adjusted Change to Year 1† | Adjusted Change to Year 2† | Baseline Value | Adjusted Change to Year 1† | Adjusted Change to Year 2† | Baseline to Year 1† | Baseline to Year 2† |
| Diet | | | | | | | | |
| Energy, kcal | 1 724.35 (41.39) | −83.86 (36.65) | −22.60 (31.34) | 1733.09 (43.91) | 42.29 (37.90) | 91.57 (33.00) | .02 | 0.01 |
| Total calcium level, mg/d | 977.43 (38.78) | −64.41 (38.41) | −13.72 (39.27) | 994.37 (46.70) | 152.21 (39.72) | 227.56 (41.35) | <.001 | <.001 |
| Vitamin D level, IU/d | 157.01 (11.28) | 2.95 (10.33) | 54.03 (13.76) | 166.42 (14.13) | 37.27 (10.68) | 86.13 (14.49) | .02 | 0.11 |
| Fruits and vegetables, servings/d | 3.53 (0.21) | 0.01 (0.18) | 0.16 (0.21) | 3.68 (0.24) | 0.74 (0.19) | 0.95 (0.22) | .005 | .01 |
| Soda, oz/d | 5.20 (0.69) | −0.05 (0.47) | −0.98 (0.38) | 5.13 (0.69) | −0.95 (0.48) | −1.24 (0.40) | .18 | .65 |
| Strength and physical activity | | | | | | | | |
| High-impact activity, min/d | 14.44 (1.77) | −0.14 (1.19) | −2.98 (0.96) | 13.23 (1.44) | −2.63 (1.23) | −4.07 (1.01) | .15 | .43 |
| Strength training, min/d | 8.05 (2.58) | −3.36 (0.97) | −0.94 (0.66) | 5.74 (1.05) | −0.83 (1.00) | −0.70 (0.70) | .07 | .80 |
| Total activity, min/d | 61.43 (5.32) | −7.98 (3.22) | −10.01 (3.21) | 62.45 (4.29) | −11.24 (3.33) | −14.51 (3.38) | .48 | .34 |
| Sit-ups, No./min‡ | 34.21 (0.92) | 0.64 (0.61) | 1.57 (0.67) | 32.91 (0.82) | 0.96 (0.63) | 2.57 (0.69) | .71 | .30 |
| Vertical jump, in‡ | 12.62 (0.28) | 0.92 (0.23) | 0.84 (0.23) | 12.67 (0.23) | 0.86 (0.23) | 0.65 (0.23) | .86 | .55 |
| Anthropometry | | | | | | | | |
| Height, in‡ | 64.69 (0.25) | 0.20 (0.07) | 0.37 (0.07) | 64.34 (0.29) | 0.30 (0.07) | 0.48 (0.07) | .29 | .29 |
| Weight, lb‡ | 123.79 (1.52) | 3.00 (0.54) | 4.73 (0.78) | 120.83 (1.40) | 1.71 (0.56) | 4.38 (0.80) | .10 | .75 |
| BMI‡ | 20.75 (0.19) | 0.38 (0.09) | 0.55 (0.13) | 20.51 (0.19) | 0.10 (0.09) | 0.42 (0.13) | .03 | .46 |

Abbreviation: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared).

SI conversion factors: To convert calcium to millimoles per liter, multiply by 0.25; ounces to grams, multiply by 28; inches to centimeters, multiply by 2.54; and pounds to kilograms, multiply by 0.45.

*Data are given as mean (standard error), unless otherwise indicated.

†Adjusted for baseline values.

‡Missing values for both control and intervention groups were imputed by matching the participant with missing data with a similar participant in the control group based on menarcheal age, baseline BMI, and baseline height. Missing data were as follows: height, weight, and BMI in 7 girls in the control group and 1 girl in the intervention group; vertical jump, 7 girls in the control group and 3 girls in the intervention group; and sit-ups, 8 girls in the control group and 3 girls in the intervention group.

*N*-terminal telopeptides biomarker demonstrated a larger relative decrease in the intervention group compared with the control group (AMD, 2.05; $P=.02$), with nonsignificant changes in osteocalcin. This combination is consistent with more net bone formation in participants in the intervention group.

## COMMENT

The YOUTH health care–based lifestyle intervention increased BMD gains and improved dietary intake during a 2-year period. The intervention resulted in significant increases in BMD in the spine and femoral trochanter and increases in dietary calcium, vitamin D, and fruit and vegetable consumption. As expected, in adolescents who had essentially finished growing, we observed no changes in bone size, and the greater increase in BMD seemed to come from a greater accrual of BMC in the intervention group. Further, the biomarkers collected in the baseline and second-year follow-up visits were consistent with the observed bone mineral changes. Changes achieved in BMD and dietary behavior were achieved largely during the first year of the intervention. In the second year, the difference between groups was maintained and BMD and dietary behavior were not further improved in the intervention group. Finally, our retention rate for participants was 88% for the 2 years of the study.

Although we did not directly examine the cellular basis for the BMD changes, the maintenance of serum osteocalcin levels (a marker of osteoblastic function) with a relative reduction in *N*-terminal telopeptides levels (a marker of osteoclast activity) in the intervention group suggests that the intervention reduced bone resorption while allowing bone formation to continue. We would expect increases in fruit and vegetable intake to reduce dietary acid load, and fruits and vegetables have been associated with reduced bone resorption, maintained bone formation, and higher BMD.[13] Similarly, increased calcium and vitamin D intake has been shown to reduce bone resorption. These findings suggest that the skeletal changes induced by the intervention are biologically credible and are likely to enhance bone strength.

The significant increase in BMD in the intervention group was associated with targeted dietary behaviors. This increase in BMD is especially significant because the dietary changes occurred in a community setting. The researchers had no control over the physical environment, and the individually targeted intervention did not affect the girls' peer groups. Other studies with calcium-related bone mineral changes have relied on supplementation rather than influence of adolescents' dietary behavior.[52,53] One recent study targeting dietary calcium

**Table 3. Baseline to Year 1 and Year 2 Bone Marker and Body Composition Outcomes***

| Outcome | Control Group (n = 108) | | | Intervention Group (n = 101) | | | *P* Value | |
|---|---|---|---|---|---|---|---|---|
| | Baseline Value | Adjusted Change to Year 1† | Adjusted Change to Year 2† | Baseline Value | Adjusted Change to Year 1† | Adjusted Change to Year 2† | Baseline Year 1† | Baseline Year 2† |
| Bone mineral density | | | | | | | | |
| Total body, g/cm$^2$ | 1.114 (0.008) | 0.024 (0.002) | 0.025 (0.002) | 1.114 (0.008) | 0.027 (0.002) | 0.029 (0.003) | .27 | .19 |
| Spine, g/cm$^2$ | 0.973 (0.009) | 0.017 (0.002) | 0.033 (0.003) | 0.962 (0.010) | 0.029 (0.002) | 0.045 (0.003) | <.001 | 0.007 |
| Trochanter, cm$^2$ | 0.775 (0.010) | 0.003 (0.002) | 0.003 (0.003) | 0.763 (0.011) | 0.010 (0.002) | 0.013 (0.003) | .03 | .03 |
| Femoral neck, cm$^2$ | 0.894 (0.010) | 0.009 (0.003) | 0.015 (0.004) | 0.891 (0.012) | 0.010 (0.003) | 0.020 (0.004) | .70 | .44 |
| Total hip, cm$^2$ | 0.974 (0.010) | 0.009 (0.002) | 0.017 (0.003) | 0.966 (0.011) | 0.015 (0.002) | 0.021 (0.003) | .06 | .36 |
| Bone mineral apparent density‡ | | | | | | | | |
| Spine | 0.148 (0.001) | 0.002 (0.000) | 0.005 (0.000) | 0.148 (0.001) | 0.004 (0.000) | 0.006 (0.000) | 0.003 | 0.02 |
| Bone mineral content | | | | | | | | |
| Total body, g | 2087.951 (28.515) | 74.499 (4.943) | 91.847 (6.408) | 2059.446 (26.311) | 85.134 (5.113) | 110.629 (6.561) | .14 | .04 |
| Spine, g | 54.602 (1.101) | 1.369 (0.314) | 2.554 (0.229) | 53.643 (0.998) | 2.001 (0.324) | 3.263 (0.235) | .17 | .03 |
| Trochanter, g | 7.561 (0.185) | 0.124 (0.061) | 0.169 (0.066) | 7.274 (0.141) | 0.189 (0.063) | 0.334 (0.067) | .47 | .08 |
| Femoral neck, g | 4.152 (0.058) | 0.103 (0.016) | 0.113 (0.022) | 4.117 (0.059) | 0.078 (0.017) | 0.095 (0.023) | .30 | .59 |
| Total hip, g | 30.648 (0.489) | 0.505 (0.121) | 1.077 (0.120) | 29.818 (0.438) | 0.667 (0.125) | 1.203 (1.203) | .36 | .46 |
| Bone area | | | | | | | | |
| Total body, cm$^2$ | 1867.373 (15.757) | 25.966 (3.224) | 41.651 (3.468) | 1844.583 (15.056) | 30.560 (3.335) | 49.348 (3.551) | .33 | .12 |
| Spine, cm$^2$ | 55.787 (0.842) | 0.530 (0.280) | 0.769 (0.099) | 55.546 (0.680) | 0.403 (0.289) | 0.770 (0.102) | .75 | .997 |
| Trochanter, cm$^2$ | 9.696 (0.160) | 0.119 (0.062) | 0.178 (0.064) | 9.532 (0.126) | 0.096 (0.064) | 0.269 (0.066) | .80 | .33 |
| Femoral neck, cm$^2$ | 4.642 (0.037) | 0.069 (0.016) | 0.050 (0.019) | 4.629 (0.034) | 0.037 (0.016) | 0.008 (0.019) | .16 | .13 |
| Total hip, cm$^2$ | 31.412 (0.323) | 0.247 (0.084) | 0.574 (0.072) | 30.888 (0.277) | 0.182 (0.087) | 0.541 (0.073) | .60 | .75 |
| Body composition | | | | | | | | |
| Fat mass, % of weight | 24.353 (0.512) | 0.582 (0.204) | 0.673 (0.275) | 24.106 (0.443) | 0.285 (0.211) | 0.482 (0.282) | .32 | .63 |
| Lean mass, % of weight | 67.147 (.488) | –0.002 (0.194) | –0.188 (0.266) | 67.446 (0.428) | –0.079 (0.200) | –0.223 (0.272) | .72 | .93 |
| Biomarkers | | | | | | | | |
| Osteocalcin, ng/mL§ | 17.932 (0.715) | | –5.597 (0.438) | 19.214 (1.006) | | –5.557 (0.420) | | .95 |
| Naltrexone, nM BCE‖ | 36.462 (2.024) | | –11.163 (0.618) | 37.024 (1.825) | | –13.211 (0.610) | | .02 |

Abbreviation: BCE, bone collagen equivalents.
*Data are given as mean (standard error), unless otherwise indicated.
†Adjusted for baseline values age, menarche, risk of osteoporosis, height, and BMI.
‡Intervention group, n = 90; control group, n = 94.
§Intervention group, n = 69; control group, n = 65.
‖Intervention group, n = 66; control group, n = 66.

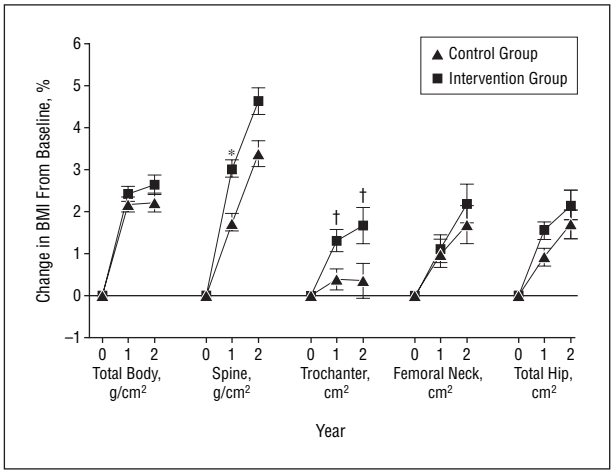


**Figure 2.** Percent changes in bone mineral density during 2 years. *$P$ <.01; †$P$ <.05.

showed significant increases in dietary calcium but not associated bone mineral changes.[54] In addition, studies targeting bone mineral changes have not emphasized other dietary factors that may contribute to BMD.[11-13] In this study, baseline calcium intake was already close to recommendations, whereas vitamin D and fruit and vegetable intake was below recommendations; this suggests the importance of vitamin D and fruits and vegetables in the outcomes. The improvements achieved in the intervention group in fruit and vegetable consumption (about 20% increase in year 1 and 26% overall increase by year 2; from 3.68 servings at baseline to 4.42 and 4.62 for follow-up years 1 and 2, respectively) exceeded changes achieved in school-based adolescent studies that have specifically examined fruit and vegetable consumption.[55,56] The intervention did not significantly affect soda consumption; however, study participants reported drinking little soda.

Despite significant improvements in BMD and dietary targets, reported levels of physical activity and physiologic strength measures did not differ between the intervention and comparison groups. Although levels in individual girls varied substantially, overall trends suggested that physical activity declined in both study conditions. This finding mirrors reports of overall decline

in physical activity during adolescence[32,57,58] and a recent community trial that attempted to increase weight-bearing physical activity to promote bone mass gains in younger girls.[54] Studies that have positively affected adolescent girls' physical activity were school-based interventions that enrolled girls in structured physical education classes[53,59-61] rather than relying on self-directed changes. In addition to this study's component of self-directed change, our study population reported an initially high level of physical activity: 69% of the girls participated in team sports. Since they were already active, this group may have been a particularly difficult population in which to increase or even shift physical activity. Finally, despite the decline in physical activity, we did not observe a commensurate decline in physical strength or fitness measures.

Although this study uniquely contributes to the previous research, this medical setting has some limitations. Our population was largely white, from middle- to upper-middle-income working families, and had relatively high levels of reported calcium consumption and physical activity at baseline. Therefore, the intervention might need adjustments in different populations. Further, some intervention elements, such as events for participant motivation and retention, may not be easily replicated in all medical settings. Another limitation is that health plans might have less participant contact than schools do. We addressed this limitation by providing a wide range of intervention components with both in-person and remote study contact to maximize participant exposure to the intervention. The effect on participants' dietary habits was more substantial than that achieved in most school-based interventions targeting these factors, although possible differences in participant socioeconomic status may have influenced the ease of achieving the dietary targets. Our results suggest that the dietary intervention designed to empower high school–aged girls to take charge of their health was reasonably successful. Conversely, we had more difficulty in achieving our physical activity targets than school-based interventions do. Health care settings may be best suited to helping adolescents achieve change in domains in which individual tailoring is important and the behavior is more individual; conversely, substantially increasing physical activity may be maximized with the built-in community and structure that school interventions provide.

In summary, the YOUTH project is one of very few preventive research interventions in adolescents conducted in a health plan setting. Information available to medical providers may provide ways of targeting such interventions (eg, a family history of hip fracture or osteoporosis). Our results suggest that a comprehensive health care–based lifestyle intervention can effectively increase bone mineral gains and improve dietary intake. Future research should examine what this magnitude of BMD gain means for adult osteoporosis risk. To our knowledge, this study is the first to significantly improve bone mass in adolescents in a non–school-based intervention emphasizing self-directed behavioral change.

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**Author Contributions:** Dr DeBar had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* DeBar, Ritenbaugh, Aicken, Orwoll, Elliot, Vuckovic, Stevens, and Moe. *Intervention design*: Stevens. *Acquisition of data:* DeBar, Ritenbaugh, and Dickerson. *Analysis and interpretation of data:* Ritenbaugh, Aicken, Orwoll, Dickerson, Vuckovic, and Stevens. *Drafting of the manuscript:* DeBar, Ritenbaugh, and Aicken. *Critical revision of the manuscript for important intellectual content*: Ritenbaugh, Aicken, Orwoll, Elliot, Dickerson, Vuckovic, Stevens, and Moe. *Statistical analysis:* Aicken. *Obtained funding:* Ritenbaugh, Aicken, and Stevens. *Administrative, technical, and material support:* DeBar, Ritenbaugh, Orwoll, Elliot, Dickerson, Stevens, and Moe. *Study supervision:* DeBar and Ritenbaugh. *Qualitative research*: Vuckovic.
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**Dedication:** We dedicate this article to the memory of Dr Irving, who was an essential part of the conceptualization and realization of the project.

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gest that it would be prudent for health care professionals to consider screening persons 14 years or older who have a history of an NICU admission before July 1992.

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**Author Contributions:** Mr Cagle had full access to all program data and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Williams and McMahon. *Acquisition of data:* Cagle, Jacob, Homan, and Christensen. *Analysis and interpretation of data:* Cagle, Jacob, Homan, and McMahon. *Drafting of the manuscript:* Cagle, Williams, Christensen, and McMahon. *Critical revision of the manuscript for important intellectual content*: Cagle, Jacob, Homan, and McMahon. *Statistical analysis:* Cagle. *Obtained funding:* Williams and McMahon. *Administrative, technical, and material support:* Cagle, Jacob, Homan, and Williams. *Study supervision:* Cagle, Williams, Christensen, and McMahon.
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## *Correction*

**Incorrect Trial Registration Identifier Number.** In the article titled "YOUTH: A Health Plan–Based Lifestyle Intervention Increases Bone Mineral Density in Adolescent Girls," by DeBar et al published in the December issue of the ARCHIVES (2006;160:1269-1276) the trial registration identifier number on page 1269 should have been NCT00067600.