Dose-response of physical activity and low back pain, osteoarthritis, and osteoporosis

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ABSTRACT

VUORI, I. M. Dose-response of physical activity and low back pain, osteoarthritis, and osteoporosis. *Med. Sci. Sports Exerc.*, Vol. 33, No. 6, Suppl., 2001, pp. S551–S586. **Purpose:** The purpose of this study was to examine the evidence for causal relationships between physical activity (PA) and low back pain (LBP), osteoarthritis (OA), and osteoporosis (OP), and for dose-response relations involved. **Methods:** Computer database searches and personal retrieval systems were used to locate relevant literature. **Results:** PA can be effective in preventing LBP (Category A) but prolonged, heavy loading can lead to LBP (Category C). Specific exercises have not been found effective in treatment of acute LBP (Category A), but PA can be effective in chronic LBP (Category B), especially for diminishing the effects of deconditioning. No evidence indicates that PA directly prevents OA. Large amounts of intensive PA involving high impacts or torsional loadings or causing injuries increases risk of OA (Category C). Light or moderate PA does not increase the risk of OA (Category C). PA can be effective in the treatment and rehabilitation of OA (Category B). High-intensity loading is osteogenic and possibly useful in prevention of OP (Category A) at the loaded site, but low to moderate loading is not osteogenic (Category D). Static efforts and slow movements are ineffective or less effective than fast application of force (Category B). The types of PA to attain the effects mentioned above are known except in the case of prevention of LBP, but dose-response relationships are poorly known; at best, semiquantitatively on the basis of just a few studies. **Conclusion:** Given the shown primary and/or secondary preventative effectiveness of PA regarding LBP, OA, and OP, research to elucidate the inadequately known dose-response relations should be given high priority. **Key Words:** PHYSICAL ACTIVITY, EXERCISE, SPORT, LOW BACK PAIN, OSTEOARTHRITIS, OSTEOPOROSIS, PREVENTION, TREATMENT, DOSE-RESPONSE, REVIEW

Low back pain (LBP), osteoarthritis (OA), and osteoporosis (OP) are prevalent and increasing musculoskeletal disorders that cause a great amount of suffering, loss of productivity and independence, as well as costs to individuals and societies. The prevalence of all these conditions is increasing, partly because of aging of populations and partly because of widespread adverse changes in lifestyles and environments. There is increasing evidence that physical activity is related to the development and course of these conditions, but the relationships of causality, directions, strength, and modifying factors are only partially known. However, in order to advise people and to engineer work, leisure, domestic chores, and environments regarding physical activity for the best of health, thorough knowledge of the effects on health of various forms of physical activity and of the dose-response relationships involved is needed. In this article, the published literature on the relationships between physical activity (PA) and LBP, OA, and OP is reviewed focusing primarily on the question, How much and what kind of PA (dose, exposure) cause development, prevent development, or prevent worsening of LBP, OA, and OP? The primary interest is on the effects of leisure time physical activity (LTPA) in apparently healthy individuals.

The analysis is conducted in the following successive steps: examination of the evidence for 1) the existence of one or more of the possible relationships; 2) causality of the relationships; 3) mechanisms of the effects; 4) characteristics of PA causing a given health outcome; 5) possibilities to assess and quantitate the dose or exposure; 6) analysis of published studies regarding dose-response relationships; 7) descriptive and quantitative conclusions; and 8) recommendations for further research.

The analysis is derived mainly from human studies. Information from experimental studies on animals is used especially to obtain firm evidence of the existence of a relationship, its causality and mechanism, and the characteristics of the activity causing the outcome.

METHODS

The material was collected by a computerized literature search of the MEDLINE/PubMed, and DataStar, SPORT, and the Cochrane database from 1990 to July 2000 to identify human studies on relationships between PA and LBP, OA, and OP. The key words were exercise, exercise therapy, physical education and training, physical activity, physical fitness, and sport connected with dose-response, level, volume, quantity, amount, dose, effect, and impact or influence of PA/exercise. In addition, bibliographies of major reviews published in peer-reviewed journals and major textbooks as well as in part of the original publications were

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cross-referenced and the author's own archives were used especially to identify studies published before 1990. Special emphasis was paid to identify randomized (RCT) and other controlled (CT) trials. Relevant articles derived from title and abstract were copied for review.

The computerized searches yielded the following results. The DataStar search found 330 articles on LBP, 327 on OA, and 524 on OP. The PubMed search revealed 29 reviews, 45 controlled trials, and 78 other studies on LBP. The corresponding figures for OA were 18, 20, and 45, and for OP 50, 18, and 70. The search of the Sport database revealed 14 articles on LBP, 30 on OA, and 72 on OP. A substantial part of the publications identified by the three searches were the same. The search of the Cochrane Library on LBP revealed 180 studies in the controlled trials registers and 30 completed systematic reviews. The corresponding figures for OA were 76 and 16, and for OP, 77 and 16.

RESULTS

LBP

Background. LBP is pain, muscle tension, or stiffness localized below the costal margin and above the inferior gluteal folds, with or without leg pain (sciatica). It may be acute (aLBP) or chronic (persisting 12 wk or more) (cLBP). Nonspecific LBP is that not attributed to recognizable pathology, such as infection, inflammation, rheumatoid arthritis, fracture, tumor, or OP. LBP is nonspecific in about 85% of people. The origin and mechanisms of the pain are not definitely known.

Risk factors for LBP are poorly understood: the most frequently reported are heavy physical work; frequent bending, twisting, lifting, pulling, and pushing; repetitive work; static postures; and vibrations. Psychosocial risk factors include anxiety, depression, job dissatisfaction, and mental stress at work. Several risk factors related to inactivity or immobilization have been proposed: reduced muscle strength in back, abdominal, and thigh muscles; reduced endurance in back muscles; hypermobility in lumbar column; and hypomobility of hip joints (96). Genetics is likely to play a significant role in the development of the processes leading to LBP (9).

PA might relate to LBP as a provoking, preventing, worsening, or improving factor. The theoretical rationale for these relations includes the following ideas, derived from the summary of Suni (234): 1) PA can induce acute and repetitive subclinical or more severe injuries in the back structures; 2) higher strength of the muscles of the back and trunk could protect the back from injury or minimize the effects of injurious events; 3) higher endurance of the trunk muscles helps to maintain motor control because of less fatigue in various tasks thus decreasing the risk of high loading of spine structures or occurrence of malfunctions and consequently development of injury; 4) better flexibility may decrease the risk of injury especially during lifting and bending activities; 5) good motor skills decrease the risk of injury in various tasks; and 6) good general or aerobic endurance helps to counteract fatigue and development of injury. Additional suggested mechanisms include improved circulation to the back structures and improved mood, which would influence favorably sensitivity to pain (233).

In addition, PA may influence the development or the course of an LBP episode in indirect, unspecific ways, e.g., through influences on body mass, mood, perceptions, and motivation, and by decreasing or abolishing the effects of physiological deconditioning because of inactivity or hypoactivity. Current knowledge of the risk factors, genesis, and mechanisms of action of PA in relation to LBP provide only a weak theoretical basis to search for dose-response relationships between PA and prevention, causation, treatment, and secondary prevention of LBP.

Review of research evidence. In the following review, the examination of evidence for associations between PA and LBP is mainly limited to LTPA and to low back symptoms and clinical findings. Thus, occupational activities, results of various fitness tests, and consequences of LBP such as absence from work are not thoroughly considered.

LTPA and incidence of LBP. Physical activity may have dual roles as positive and negative influences on the back (252). One case-control study (174) did not find increased risk of lumbar herniation attributable to sports and weight lifting, and a population-based prospective cohort study with 1-yr follow-up on 2715 adults (41) did not find increased or decreased risk of LBP associated with most types of LTPA, although home-improvement work in men and regular sport in women increased the risk. Increased risk of developing low back problems and pathology associated with long-term heavy physical loading is supported by studies on workers (205,233,251). A recent cross-sectional study on a large population sample revealed that activities characterized by an awkward posture, by maintaining the posture for a long time, or by often bending or rotating the trunk increased the risk of LBP, with odds ratios between 1.1 and 1.6. More than 13% of the 1-yr prevalence of LBP could be attributed to these activities (196). Three prospective longitudinal population-based studies did not find any relationship between LTPA and development of LBP (5,78,130), but among industrial workers, low levels of LTPA were predictive of development of LBP during 5-yr follow-up (143). A recent systematic review (233) of RCTs as well as earlier reviews on the topic concluded that there is convincing evidence that exercise training can be effective and is currently the only effective tested modality for prevention of LBP. One recent RCT (93) not included in the review cited above found no differences in LBP episodes at 6, 12, or 24 months in previously asymptomatic subjects with weak abdominal muscle strength after participation in either back education or back education plus abdominal exercise instruction group. Summary of the reviewed RCTs is presented in Table 1.

In conclusion, there is strong evidence (Category A) that LTPA can have a primary preventive effect on LBP. Most forms of LTPA as commonly practiced do not seem to increase the risk of LBP. Prolonged heavy occupa-

TABLE 1. RCTs on prevention of LBP by PA.

	Subjects						
Reference	*N*	Age	Sex	Population	Physical Activity	Results	Comment
Donchin et al., 1990 (50)	CAL = 46	45	Men: 37%	Hospital employees, at least 3 annual episodes of LBP	CAL: calisthenics to strengthen abdominal muscles to expand spinal forward flexion and rectifying general posture; supervised sessions 45 min, 2×/wk, 3 mo	Monthly surveillance over 1 yr showed significantly less "painful months" in CAL (4.5) as compared with BS (7.3) and C (7.4). Significantly larger increase in trunk forward flexion and abdominal muscle strength in CAL as compared with BS and C.	
	BS = 46	48	Men: 35%		BS: back school (instruction, advice), 5 sessions 12 mo, 90 min		
	C = 50	45	Men: 30%		C: control group, no measures		
Gundevall et al., 1993 (76)	T = 28	38	Men: 1 (of all subjects)	Personnel at a geriatric hospital, with or without back symptoms	T: individual exercise sessions 5 times by physiotherapist, exercises to increase dynamic endurance, isometric strength and functional coordination of trunk muscles performed during working hours, in average 6×/mo, 20 min, during 13 mo	Absence because of LBP: 28 day/1 prs in T, 155 days/12 prs in C ($P < 0.004$). Days with complaints: 54 in T, 94 in C ($P < 0.02$). Significant increase of back muscle strength in T vs C.	
	C = 32	37			C: no measures	Significant increase of back muscle strength in T vs C.	
Helewa et al., 1999 (40)	T+BE = 203	38	Men: 46%	City residents, employees, and students of academic and medical institutions, asymptomatic and with weak abdominal muscle strength	T+BE: T: physiotherapist instructed individually simple exercises to be performed daily at home	No significant differences between the groups in LBP episodes at 6, 12, or 24 mo. No improvement of abdominal muscle strength in T+BE.	Poor compliance: loss to follow-up 36% in T+BE, 17% in BE
	BE = 199	38	Men: 47%		BE: one 90-min class given by a physiotherapist at baseline and at 1 and 2 yr.		
Kellet et al., 1991 (110)	T = 58	41 (for 37)	Men: 70%	Employees of one large industrial firm, with or without current LBP, willing for training	T: duration 1½ years; supervised session 1×/wk during working hours, 30–35 min exercises for flexibility, strength, endurance, and coordination and in one third of the classes back education for 10 min; in addition, at least 30 min endurance activity 1×/wk	In T, 50% and 51% decrease of episodes of LBP and sick days because of LBP during 1½-yr intervention period compared with respective data during the 1½ years prior to the intervention. In C, the corresponding percentage changes were 58% increase in LBP episodes and 65% increase in sick days because of LBP. The differences between the groups are significant.	36% of the subjects in T lost from follow-up
	C = 53	42 (for 48)	Men: 70%		C: control group, no measures		
Linton et al., 1989 (147)	T = 36	20–59	Men: 0	Nursing personnel with a history of intermittent LBP during the 2 yr prior to the study	T: 5-wk program at a rehabilitation clinic, 4 h of aerobic activities and 4 h of back education daily	At 6 mo, subjects in group T reported greater improvements than C subjects in pain, fatigue, and activities of daily living. At 18 mo, sick days for T had increased from baseline.	Individual contribution of each intervention to the positive results is unclear
	C = 30				C: control group, no measures		
Linton et al., 1996 (148)	T = 36	42	Men: 31%	Employees of tobacco and distribution companies, LBP during preceeding year, otherwise healthy, sedentary	T: individually planned training	No difference in LBP between the groups.	
	C = 32	42	Men: 28%		C: training instructions, free admission to gym		

tional and sports activities seem to increase the risk of LBP, but the role of subclinical or more severe injuries cannot be completely excluded. At the other end of the PA spectrum, a systematic, critical review of the current literature did not find support for the popular opinion that sitting while at work is associated with increased risk of LBP (84). Concerning dose-response issues, the available evidence suggests that most commonly practiced LTPA activities are safe in terms of LBP within a broad range of volume and intensity. For primary prevention of LBP the possibly effective characteristics of LTPA have not been verified, and therefore dose-response relationships are not known either.

LTPA in secondary prevention. Several reviews have examined the effects of exercise training and LTPA in the treatment and secondary prevention of aLBP and cLBP (16,26,28,61,96,121,131,249,259). The most recent review of RCTs (until February 2000, $N = 39$) (250) presents the current evidence using a rigorous protocol of data collection and analysis. The conclusions are as follows:

For aLBP. Strong evidence (Category A) indicates that exercise therapy is not more effective than inactive treatments or other active treatments. Flexion and extension exercises are not effective. Sound evidence is lacking to confirm the claim that exercises in aLBP may prevent future recurrences or chronicity. Low stress activities such as walking, biking, or swimming can be started during the first 2 wk.

For cLBP. 1) Exercise versus other active treatment: strong evidence (Category A) indicates that exercise therapy and conventional physiotherapy are equally effective and that exercise therapy is more effective than usual care by a general practitioner. Limited evidence (Category C) suggests that exercise provides better outcomes than back school. 2) Exercise therapy versus inactive or "placebo" treatment: the evidence is conflicting (Category C) regarding the effectiveness of exercise therapy for cLBP. 3) There is conflicting evidence (Category C) about which type of exercises, extension or flexion, are more effective. 4) Strong evidence (Category A) indicates that strengthening exercises are more effective than other types of exercise. The evidence is conflicting that strengthening exercises are more effective than inactive treatment (Category C).

The reviewers conclude that specific exercises are not recommended for patients with aLBP or cLBP. However, exercises may be useful within an active rehabilitation program if they facilitate and precipitate increasing ordinary activity and returning to work. Pertinent data of the reviewed studies are presented in Table 2.

Another recent systematic literature review of 16 RCTs (all included in van Tulder et al. (250)) (233) essentially agrees with the above-cited conclusions by stating that in aLBP various forms of exercise training are ineffective treatment modalities (Category A), and in cLBP exercise training is effective in decreasing pain (Category A).

Some studies from 1999–2000 not included in the cited reviews are worth reviewing. One RCT found that endurance training of trunk extensor muscles for 6 wk in subjects with subacute LBP decreased symptoms and improved function at 3 wk but not at 6 wk in the training group as compared with the control group (34). This study thus suggests that endurance training of the trunk muscles expedites the recovery process for patients with an acute episode of LBP.

Another RCT on subjects with cLBP found that an individually adjusted exercise program specially designed to train trunk muscle function (endurance) and coordination decreased back pain intensity and self-reported functional disability in the treated group more than in the placebo-treated subjects at 6- and 12-month follow-up (105).

Mannion et al. (160) compared the efficacy of modern active physiotherapy, muscle reconditioning on training devices, and low-impact aerobics for cLBP in a randomized trial. All three treatments proved to be equally efficacious in reducing symptoms and disability in tasks of daily living immediately after the therapies and during the following 6 months. The equal results in the three groups suggest that the effects were not reduced through specific physiologic adaptations but rather through some "central" psychological mechanism(s). Essential information of the three studies cited above is included in Table 2.

Dose-response relationship could be directly examined in one study only (158). In this CT, intensive as compared with light intensity back strengthening program resulted in significantly better improvement in a combined pain, disability, and impairment index at 3- and 9-month follow-up. In another CT (159), the "dose" in two exercise programs differed in the degree of flexion in dynamic exercises and in including or excluding hyperextension. LBP rating improved more in the group that used wider flexion and hyperextension in the exercises at 3 but not at 12 months. No subgroup analyses were reported in the published studies on dose-response issues. Comparison of different studies regarding dose-response relationships is not warranted or even possible because of wide differences in a number of factors that influence the results.

Concluding remarks. The current evidence of the role of PA in causation and primary and secondary prevention of LPB can be summarized as follows.

1. Prolonged, repetitive, heavy physical activity at work or in sports can cause LBP in susceptible individuals (Category C), but the role of injury is not known. No dose-response relationships have been established and, because of the large variation of individual susceptibility and to the multiple physical, physiological, psychological, and social factors involved, it is likely that only crude, categorical dose-response relationships can be established.
2. Strong evidence (Category A) indicates that PA can have a preventive effect on LBP. The characteristics of effective activity have not been established. However, it is worth noting that trunk extensor endurance was systematically associated with LBP in cross-sectional studies (234), and it had predictive value for first-time LBP in two population studies (16,153), and endurance training of trunk muscles showed positive results in two RCTs on secondary prevention of LBP (34,105).
3. Strong evidence (Category A) shows that specific exercises are not effective in the treatment of acute LBP, but continuing ordinary activities and starting low-stress aerobic activities during the first 2 wk is indicated in order to improve recovery and lessen disability.
4. The role of PA in the treatment and secondary prevention of cLBP should be examined from at least two points of view: first, symptoms and psychosocial consequences; and second, biological effects of prolonged deconditioning. Some evidence (Category B) suggests that PA, especially as structured exercises, alleviates symptoms of cLBP and may improve function. These

TABLE 2. RCTs on the effectiveness of exercise training as treatment/rehabilitation of LBP.

	Subjects						
Reference	***N***	**Age**	**Sex**	**Group**	**Exercise Training**	**Results**	**Comment**
Bentsen et al., 1997 (13)	T1 = 41 T2 = 33	57	F	Chronic nonspecific LBP	T1: home, back and abdominal strengthening exercises, 10×/d, 12 mo + fitness center, dynamic exercises, 2×/wk, 30 min, 3 mo T2: home only, as above	T1 more improved in functional status after 12 mo.	Data poorly presented.
Bronfort et al., 1996 (23)	T1 = 71 T2 = 71 R = 71	20–60	F, M	Chronic nonspecific LBP	T1: strengthening exercises for trunk, legs, 20 reps each, 20 sessions, 11 weeks + manipulative therapy T2: stretching + manipulative therapy, 20 1-h sessions, 11 wk R: strengthening exercises as in T, + NSAIDs	No significant differences in pain, functional status, or generic health after 5 and 11 wk between the groups.	
Buswell, 1982 (25)	T1 = 25 T2 = 25	16–59	F, M	Chronic recurrent LBP	T1: flexion exercises, 8–14 times T2: extension exercises, 8–14 times	No difference in change of pain between the groups.	Data incompletely presented.
Cherkin et al., 1998 (32)	T = 133 R = 122 R2 = 66	20–64	F, M	Acute LBP	T: McKenzie exercises up to 9 times, 1 mo R1: chiropractic manipulation up to 9 times R2: educational booklet	No significant differences in global improvement or functional status between T and R1, or T and R2.	
Chok et al., 1999 (34)	T = 30 R = 24	21–54	F, M	Subacute LBP	T: trunk muscle endurance exercises, up to 5 series of 10 reps for 6 cycles, 30–45 min, 3×/wk, 6 wk at physiotherapy department R: hot pack, advice, booklet	At 3 wk, reduced pain and improved function in T vs C, no differences at 6 wk.	
Coxhead et al., 1981 (40)	T = 150 R = 142	42	F, M	Acute and chronic LBP	T: range-of-motion exercises, first week daily, next 3 wk decreasing frequency R: traction or manipulation or corset, 4 wk	Comparable number of patients improved after 4 wk and 4 mo and in comparable degree on pain scale in both groups.	
Davies et al., 1979 (43)	T1 = 14 T2 = 14 R = 15	15–45	F, M	LBP between 3 wk and 6 mo duration	T1: extension exercises and diathermy, 4 wk T2: isometric flexion, strengthening trunk and abdominal muscles, diathermy, 4 wk R: diathermy, 4 wk	No significant difference between the groups in the number of patients showing improvement after 2 and 4 wk.	
Delitto et al., 1993 (47)	T1 = 14 T2 = 10	14–50	F, M	Acute or subacute LBP	T1: McKenzie extension and mobilization, supervised, 3×/wk, advice to train at home T2: Williams flexion exercises, supervised 3×/wk, advice to train at home	Functional status improved significantly more in T1 than T2 after 3 and 5 days.	
Deyo et al., 1990 (49)	T1 = 34 T2 = 29 R1 = 31 R2 = 31	18–70	F, M	Chronic LBP	T1: relaxation and stretching exercises (12), 2–3×/d, and TENS T2: exercises as above, and sham TENS R1: TENS (transcutaneous nervous stimulation) R2: sham TENS	More improvement of pain in T1+T2 than in R1+R2 after 4 wk. No significant differences in pain intensity or in functional status after 4 and 12 wk between the groups.	
Elnaggar et al., 1991 (56)	T1 = 28 T2 = 28 R = 98	20–50	F, M	Chronic LBP	T1: McKenzie extension exercises, 6 exercises, 10 repetitions, 30 min·d^{-1}, 7×/wk, 2 wk T2: Williams flexion exercises, 6 types, 10 reps, 30 min·d^{-1}, 7×/wk, 2 wk R: usual primary care management	No significant difference between the groups regarding decrease of pain.	
Faas et al., 1993 (60)	T = 156 R2 = 162 R1 = 155	16–65	F, M	Acute LBP	T: stretching, flexion, isometric abdominal exercises, individually by physiotherapist, 20 min, 2×/wk, 5 wk R1: usual care R2: placebo ultrasound	No significant differences between the groups in change of pain, recurrences of pain, or functional status except NHP energy more improved in T than in R1 during first 3 mo.	
Farrell and Twomey, 1982 (62)	T = 24 R = 24	20–65	F, M	Acute LBP	T: isometric flexion exercises for abdominal muscles and diathermy, 3×/wk, 3 wk, advice to exercise 3–4×/d at home R: passive manipulation	R significantly more effective than T regarding the number of symptom-free patients within 2 wk. No difference in pain score after 3 wk between the groups.	
Frost et al., 1995, 1998 (68,69)	T = 36 R = 35	18–55	F, M	Chronic LBP	T: fitness program, 8 1-h sessions during 4 wk and back school R: back school, advice on exercise	Functional status and pain improved significantly more after 4 wk in T than R. After 6 mo functional status more improved in T than R. After 2 yr reduction in functional status significantly less in T than R.	
Hansen et al., 1993 (82)	T = 60 R1 = 59 R2 = 61	21–64	F, M	(Sub)chronic LBP	T: intensive dynamic back muscle training, 5 series, 10 reps, total 300 contractions, 1 h, 2×/wk, 4 wk R1: physical therapy including slowly progressive back and abdominal muscle exercise, 1 h, 2×/wk, 4 wk R2: placebo	No significant differences in pain level between groups after treatment and after 1, 6, and 12 mo. Overall treatment effect was significantly better in T and R1 than in R2 at all evaluations.	
Hemmilä et al., 1997 (95)	T = 35 R1 = 45 R2 = 34	17–64	F, M	Chronic LBP	T: a variety of bending and rotation exercises 10×/d, stretching maximum 10×/h, 6 wk R1: gentle mobilization, maximum 10×/h, 6 wk R2: various, largely passive physiotherapy modalities, 10×/h, 6 wk	R1 had improved more than T at 6 mo, no other differences between groups in pain measures.	

TABLE 2. *Continued*

Reference	Subjects				Exercise Training	Results	Comment
	N	Age	Sex	Group			
Johansen et al., 1995 (102)	T1 = 13 T2 = 14	18–65	F, M	Chronic LBP	T1: dynamic back, neck, and abdominal endurance exercises, stretching, maximum 100 reps, 1 h 2×/wk, supervised, 3 mo T2: coordination and balance exercises, 2×/wk, supervised, 3 mo	No significant differences between the groups in changes of pain or function.	
Kankaanpää et al., 1999 (105)	T = 30 R = 24	40 ± 8	F, M	Chronic LBP	T: supervised progressive exercises for trunk muscle function and coordination with specific equipment 24 sessions, 1.5 h, 12 wk, instructions for home exercise R: passive treatments considered to have placebo effect	Back pain intensity and functional disability decreased and lumbar endurance improved significantly more in T than in R at 1-yr follow-up.	
Kendall and Jenkins, 1968 (115)	T1 = 14 T2 = 14 T3 = 14	Not reported	Not reported	Chronic LBP	T1: isometric strengthening exercises for abdominal and trunk muscles, 12 reps, 3×/d, postural advice T2: mobilization and strengthening exercises, 6 types, 6–12 reps, 2×/d, postural advice T3: strengthening extension exercises for back extensors, 5 types, 6–12 reps, 2×/d, postural advice	Significantly more improved patients in T1 than in T2 after 1 and 3 mo.	
Klaber Moffett et al., 1999 (120)	T = 89	18–60	F, M	LBP between 6 wk–6 mo duration	T: supervised strengthening, stretching and relaxation exercises, 8 sessions, 1 h, 4 wk, brief back education	During 1-yr follow-up, T compared with R showed significantly greater improvement in disability score and back pain scale, reported less days off work (378 vs 607), and used fewer health care resources.	
Lidström and Zachrison, 1970 (144)	T1 = 21 R = 21 T2 = 20	21–61	F, M	Chronic LBP	T1: strengthening exercises for back and abdominal muscles, mobilizing exercises, 10 treatments by physiotherapist, 1 mo T2: isometric strengthening of abdominal and hip extensor muscles, 6 times each, pelvic traction, 10 treatments by physiotherapist, 1 mo R: hot packs, rest	T2 patients improved significantly more in 4 wk and needed less analgesics after treatment than T1 and R patients.	
Lindström et al., 1992 (145) and 1992 (146)	T = 51 R = 52	41 ± 11	F, M	Chronic LBP	T: back school, ergonomics, individual submaximal strength and endurance training by various exercise modes, 3×/wk until return to work R: usual care	Significantly more T patients than R patients returned to work in 6 and 12 wk, and duration of absence from work significantly shorter in T than R during the 2nd follow-up year. No intergroup difference in functional status after 1 yr.	
Ljunggren et al., 1997 (149)	T = 64 R = 62	18–65	F, M	Chronic LBP	T: physiotherapy exercises, back muscle strengthening, stretching, 3 series, 10 reps, 3×/wk, 12 mo R: specific exercises with "TerapiMaster," strengthening exercises for back, thigh, buttocks, abdominal and chest muscles, 12 mo	No differences between groups in days of absence from work at 12 mo and 24 mo. No differences between groups in days of absence from work at 12 mo and 24 mo.	
Malmivaara et al., 1995 (156)	T = 52 R1 = 67 R2 = 67	41	F, M	Acute LBP	T: back extension and lateral bending exercises at home until pain subsided, individual instructions from physiotherapist R1: bed rest and advice to continue ordinary activities as tolerated R2: advice to avoid bed rest and to continue ordinary activities as tolerated	Significantly better outcome on functional status at 3 wk in R2 than in T.	
Manniche et al., 1988 (158)	T1 = 27 T3 = 31 T2 = 32	20–70	F, M	Chronic or recurrent LBP	T1: intensive supervised back extensor strengthening exercise 2 times 50 reps each exercise, 30 sessions, 1.5 h, 3 mo T2: mild isometric exercises, 10 reps, 8 sessions, 1 h, 1 mo, massage T3: mild back extensor strengthening exercises, identical to T1 but 20 reps each exercise in 45 min	Significantly more improvement in T1 than in T2 and T3 in physical impairment index after 3 and 9 mo.	
Manniche et al., 1993 (159)	T1 = 31 T2 = 31	18–74	F, M	Chronic LBP	T1: intensive dynamic exercises plus hyperextension, 2 sessions, 1–1.5 $\mathrm{h \cdot wk^{-1}}$, 24 sessions in 3 mo T2: intensive dynamic exercises as in T1 but no hyperextension	Overall improvement not significantly different between the groups, but significantly more improvement of pain in T1 than in T2 at 3 mo.	
Mannion et al., 1999 (160)	T1 = 49 T2 = 50 T3 = 49	45 ± 10	F, M	Chronic LBP	T1: individually prescribed active physiotherapy including exercises, 2×/wk, instructions for home training, 3 mo T2: progressive strengthening of trunk muscles by specific equipment, 2×/wk, 1 h, 3 mo T3: progressive low-impact, aerobic exercises and stretching, in classes, 2×/wk, 3 mo	All three treatments were equally efficacious in reducing pain intensity, pain frequency, and disability in daily tasks immediately after treatment. These effects were well maintained over the following 6 mo with the exception of disability regressing toward pretreatment value in the physiotherapy group.	
Martin et al., 1980 (164)	T1 = 12 T2 = 12 R = 12	20–58	F, M	Chronic LBP	T1: supervised strengthening and mobilizing abdominal and back muscle exercises, 20 min, 3×/wk, 3 wk, advice to exercise at home T2: supervised isometric exercises to strengthen abdominal and pelvic floor exercises, 20 min, 3×/wk, 3 wk, advice to train at home R: detuned ultrasound and diathermy, 20 min, 3×/wk, 3 wk	T1 and R improved significantly more on pain intensity at 5 weeks than T2.	

TABLE 2. *Continued*

Reference	Subjects				Exercise Training	Results	Comment
	N	**Age**	**Sex**	**Group**			
Nwuga, 1982 (185)	T = 25 R = 26	20–40	F	Acute LBP	T: isometric flexion exercises of back and abdominal muscles, 10 contractions, 3×/wk until no pain, diathermy, education R: spinal manipulation, 3×/wk until no pain, education	Spinal flexion and SLR improved significantly more in R than T. No data given on pain, global improvement, or functional status.	
Nwuga and Nwuga, 1985 (186)	T1 = 31 T2 = 31	20–40	F	Acute LBP	T1: McKenzie extension exercises prescribed by physiotherapist T2: Williams flexion exercises prescribed by physiotherapist	Significantly more improvement of pain in T1 than in T2.	
O'Sullivan et al., 1997 (189)	T = 22 R = 22	16–49	F, M	Chronic LBP	T: training of deep abdominal muscles at home, 10–15 min·d^{-1}, 10 wk R: usual care including various exercises	Significantly more improvement of pain and functional status in T than in R after treatment and up to 30 mo.	
Risch et al., 1993 (206)	T = 31 R = 23	22–70	F, M	Chronic LBP	T: dynamic extension exercises 2×/wk, 4 wk, then 1×/wk, 6 wk R: waiting list controls	Pain score and physical disability score improved significantly more in T than in R from before to after treatment.	
Sachs et al., 1994 (214)	T = 14 R = 16	35 ± 8	F, M	Chronic LBP	T: work tolerance rehabilitation: stretching, strengthening, and cardiovascular training, 4–6 h plus strength training on B-200 Isostation R: work tolerance rehabilitation only	No significant difference in range of motion after 3 wk.	
Seferlis et al., 1998 (220)	T = 60 R1 = 60 R2 = 60	19–64	F, M	Acute LBP	T: intensive strength, endurance, and coordination training in groups, 3×/wk, 8 wk	No significant intergroup differences in pain or functional status at 1, 3, and 12 mo or in the number of days off work because of back pain after 1 yr.	
Snook et al., 1998 (226)	T = 43 R = 42	30–60	F, M	Chronic or recurrent LBP	T: 45-min video instruction for a variety of exercises, training at home R: advice to avoid lumbar flexion for the first 2 h of every day	Baseline differences between groups. No direct comparison of effects between groups, but R improved more.	
Stankovic and Johnell, 1990, 1995 (231,232)	T = 50 R = 50	34 ± 10	F, M	Acute LBP	T: McKenzie extension exercises R: mini-back school	Significantly less pain and better spinal mobility in T than in R after 3 wk and 1 yr.	
Torstensen et al., 1998 (240)	T1 = 71 T2 = 70 R = 67	20–65	F, M	Chronic LBP	T1: mobilizing and stabilizing exercises in groups, total about 1000 reps/session, 3×/wk, 12 wk T2: self-organized walking, 1 h, 3×/wk, 12 wk R: conventional physiotherapy, 1 h, 3×/wk, 12 wk	Significantly greater decrease of low back pain in T1 and R than in T2 after 1 yr, and in leg pain in T1 and R than in T2 after treatment. Significantly better functional status in T1 and R than in T2 after treatment and after 1 yr.	
Turner et al., 1990 (242)	T1 = 24 T2 = 24 R1 = 25 R2 = 23	20–65	F, M	Chronic LBP	T1: aerobic exercises, 20 min, 5×/wk, 60–70% HRmax, 8 wk T2: identical to T1 plus behavioral therapy, spouses participated, 8 sessions·wk^{-1}, 2 h R1: behavioral program identical to T2; no aerobic training R2: waiting list control	T2 improved significantly more than T1 and R2 after treatment. No significant intergroup differences after 6 and 12 mo.	
Underwood and Morgan, 1998 (244)	T = 35 R = 40	16–70	F, M	Acute LBP	T: teaching of McKenzie principles, other advice, home exercises R: usual care, general advice	No significant intergroup differences in functional status and pain intensity after 4, 12, and 52 weeks, but back pain was reported to be no problem after 1 yr in 50% of T and 14% in R.	
Waterworth and Hunter, 1985 (261)	T = 34 R1 = 36 R2 = 38	18–50	F, M	Acute LBP	T: flexion and extension exercise plus passive physiotherapy 5×/wk, 10–12 d R1: NSAIDs, 10 d R2: spinal manipulation and mechanical therapy according to McKenzie, 5×/wk, 10–12 d	No significant differences between groups in pain, mobility, or overall improvement.	
White, 1996a (263)	T1 = 76 T2 = 72		M	Chronic LBP, received workmen's compensation	T1: mild static trunk exercises, back exercise classes, calisthenics in pool, physiotherapy until improvement or deterioration T2: vigorous flexion and extension exercises, heavy occupational therapy until improvement or deterioration	No significant difference between the groups in the number of patients showing improvement.	
White, 1996b (263)	T = 99 R = 95	19–60	M	Chronic LBP, received workmen's compensation for the condition	T: hospital bed rest, progressive activities until heavy, 6 wk unless fit for work earlier R: usual care	During 3 mo after discharge from study higher percentage of T patients (42%) than R patients (15%) showed satisfactory result at work.	
Zylbergold and Piper, 1981 (269)	T1 = 10 T2 = 10 R = 8	25–65		Patients from waiting list for physiotherapy	T1: instruction to perform pelvic tilt flexion exercises at home, 2×/wk, 1 mo T2: instructions for back care and pelvic tilt exercises at home R: manual therapy, 2×/wk, 1 mo	No intergroup differences in pain, functional status, or mobility after 1 mo.	

T, training; R, reference group; F, female; M, males.

effects may be mainly because of other than physiological conditioning effects of training. If so, then the type and dose of exercise could not be logically determined using physiological training principles, but effective activity would depend mainly on individual preferences and perceptions, and circumstantial factors. On the other hand, musculoskeletal and cardiorespiratory deconditioning effects as a consequence of cLBP follow known biological principles, and strong evidence (Category A) shows that those kinds of effects can be diminished or abolished by appropriate PA. It is likely that the known dose-response relationships of PA would apply in those kinds of programs. Return to work is not a recommendable outcome measure in studying the effects of physical activity on LBP patients, because return to work depends strongly on many factors unrelated to the effects of PA.

Recommendations for research. Given the great public health significance of LBP and the evidence of effectiveness of PA (as one of few promising modalities) in its primary and secondary prevention, this area of research should be given high priority. Development of and wide international agreement on standardized methodology to assess and rate LBP is required in order to improve the quality and comparability of studies on this field.

Incomplete knowledge of the causes and mechanisms of the symptoms in the LBP syndrome hampers advancement of research targeted at their prevention and alleviation. Thorough research on this area is needed.

The possible preventive and provoking roles of various types and intensities of PA in the occurrence of LBP should be searched by large-scale, long-term, prospective population studies using close monitoring of PA and other living habits, injuries, and symptoms as well as assessments of health-related fitness.

Given the evidence of effectiveness of exercise regimens in secondary prevention of cLBP, sufficiently large-scale, carefully planned, conducted, and documented clinical, preferably RCTs, should be conducted comparing different exercises alone or as parts of a common basic program. A clear distinction in the aims and design of the studies should be made whether the measures are targeted to influence the primary features of the syndrome or the deconditioning effects resulting from prolonged inactivity or hypoactivity.

OA

Background. OA is a chronic degenerative joint disease characterized primarily by progressive loss of articular cartilage. Loss of cartilage leads to narrowing of the joint space. The clinical syndrome of OA is diagnosed when, in addition to the radiological findings, narrowing of joint space, and osteophytes, the patient presents also symptoms and signs such as joint pain, restriction of motion, crepitus with motion, joint effusions, and deformity (24).

Articular cartilage is highly resistant to stress caused by physical loading. However, sudden single or repetitive impact or torsional loadings can damage articular cartilage and the calcified subchondral bone region. Acute disruption of normal articular cartilage requires contact stresses between the adjacent surfaces of 25 MPa (megapascals, newtons per square meter) or more. The peak articular contact stresses in, for example, running, jumping, and throwing are in the range of 4–9 MPa. These kinds of activities are unlikely to cause damage in healthy joints as evidenced by extensive animal experiments (24,94). However, chronic or repetitive stresses less than 25 MPa may cause articular surface damage or degeneration. Several studies show strong association between chronic or repetitive increased mechanical loading especially in occupational work and development of OA (190). Slowly applied loads allow the cartilage to deform and muscle contraction to absorb much of the energy and stabilize the joint. Therefore, the joints tolerate slowly applied loads much better than sudden impacts or torsional loading. Aging leads to alterations that may increase the probability of cartilage degeneration and OA (24).

The above considerations suggest that in searching for dose-response relationships between PA and development of OA, the focus in dose should be primarily on the size of impact loadings on the joints, the rate of the force application, the number of repetitions of high-impact loading, and the total time during which these loadings occur. One dimension of the dose is also the degree of torsion of the movement causing the loading. In ambulatory persons, the forces on joint surfaces caused by locomotion cannot be measured. Ground reaction forces of, for example, walking, running, and jumping can be measured, but the forces transmitted to the joint surfaces vary greatly, depending especially on the damping caused by muscles and their contraction at the time of the impact (24). Thus, dose-response relations of PA and development of OA in human studies have to be examined in descriptive, in best case semiquantitative terms.

Animal studies have shown that moderate PA causes beneficial structural and functional alterations in joints (94,213). However, it has not been shown that PA could prevent development of OA in ambulatory subjects through the physiological benefits. Indirectly, high PA may have a preventive effect by counteracting development of obesity, a strong risk factor for OA. Evidence from RCTs indicates that patients with OA can benefit from PA as a secondary preventive measure. Thus, examination of dose-response relationships between PA and OA is indicated also in the context of secondary prevention.

Review of research evidence. The following review is limited to examination of the role and dose-response relationships of PA and development and secondary prevention of OA to OA of hip and knee joints because these are the most relevant conditions in terms of the scope of this review.

PA and development of OA. Several studies on workers in different occupations have found increased prevalence of OA in loaded joints (190,253). Corresponding observations have been made on athletes in a number of sport disciplines (24,74,128,190,218). Because of a number of factors it is not possible to accurately define dose-response relationships between loading and OA in sports. These factors include differences in individual susceptibility to OA; strong self-selection to begin; and continuation/discontinuation of a certain sport because of such issues as differences in experiencing musculoskeletal symptoms, differences in exercise patterns, and differences in the number of subclinical and overt injuries. However, by combining the findings of incidence and prevalence studies and the current understanding of the harmful effects of impact and torsional loading on joints, it is possible to group sports and other activities into risk categories on the basis of estimated differences in intensity; frequency; and rate of joint injury, impact, and torsional loading (24). In this classification, recreational aerobic activities (especially in water) belong in the low loading category, and most ball games as well as competitive running in the high loading sports. The role of injury in the genesis of OA is strongly substantiated by several previous (210,218) and by a newly published 36-yr follow-up study on 1321 former medical students who gave information of living habits, injuries, and health in annually repeated self-administered surveys. In those reporting injury on the corresponding joint at entry to the cohort or during the follow-up, the risk of developing OA of the knee was 5.17-fold and that of hip OA 3.5-fold that of those not reporting injury (70).

Several studies indicate that among the participants of the same sport, OA of knee or hip is more common in elite compared with recreational athletes (88). The risk of OA has been found to increase with the exposure time (hours per week and duration of activity in years) to sport and recreational PA (230,254) (only in a group of active controls, $N = 138$,216,255) (Table 3). Occupational loading added to sport loading increases the risk of OA in the loaded joint (216,254,255). Running offers the most favorable opportunities for examination of dose-response relationships between sport and risk of OA, although self-selection causes difficulties in the interpretation of the findings. Eight of the 14 studies reviewed by Panush and Inzinna (190) and Gross and Marti (74) did not find significant differences in OA risk between runners and various control groups. In two of the included studies (168,175) the higher frequency of OA in knee or hip in runners was ascribed to anatomic abnormalities. In a 9-yr follow-up of previously reported runners (135,136) showing no difference in OA between runners and controls, Lane et al. (137) did not find significant differences in knee or hip OA or their progression between runners and controls (Table 4).

Moderate levels of PA have not been found to be associated with increased risk of OA in several population-based studies (31,64,81,99,138,166,216,254,255), but the risk was significantly increased in the most active subjects in several of these studies (31,64,81,99,138,166,216,254,255). Three of these studies (64,81,166) are follow-up investigations of the same cohort (Table 5). The dose associated with significantly increased risk of OA expressed in easily understandable terms was at least 4 $h \cdot d^{-1}$ of heavy PA for radiographic and at least 3 $h \cdot d^{-1}$ for symptomatic OA of the knee in one study (166) and running at least 20 $miles \cdot wk^{-1}$ for hip or knee OA in another study (31).

In conclusion, no studies have shown evidence for direct preventive effects of PA against development of OA of the weight-bearing joints in ambulatory subjects. Light and moderate activities even in large amounts have not been shown to increase the risk (Category C), but several epidemiological studies of various designs in general and selected populations show evidence (Category C) that a large amount of heavy PA for a long period of time increases the risk of OA of the loaded joints. Limited information suggests that the amount of heavy PA associated with increased risk is several hours a day for many years, probably decades. Sports that cause a large amount of high impacts and torsional loading of joints involve especially high relative risk. These observations agree well with the results from RCTs in animals. Dose-response relationships between PA and OA are obscured by several intervening factors, e.g., differences in individual susceptibility, performance technique, and equipment used.

PA and secondary prevention of OA. A recent systematic review of RCTs on this topic (248) summarizes most of the current evidence (until September 1997). Thirteen publications on 11 trials were assessed for effectiveness of exercise therapy in patients with OA of the hip or knee (Table 6). Six trials satisfied at least 50% of the validity criteria. Effect sizes indicated small to moderate beneficial effects of exercise therapy on pain. Small beneficial effects were found on self-reported and observed disability and walking, and moderate to great beneficial effects on patients' global assessment of effect. Thus, the reviewed studies show modest beneficial short-term effects of exercise therapy in patients with OA of the knee and to a lesser extent in patients with hip OA (only one trial). Hardly any information is available on long-term effects of exercise therapy. Results of the four trials comparing effects of different exercise programs remained inconclusive.

Two additional randomized trials are worth reviewing. One study found that low-intensity cycling (40% of heart rate reserve (HRR)) for 10 wk was as effective as high-intensity cycling (70% of HRR) in improving function and gait, decreasing pain, and increasing aerobic capacity in older subjects (157). Cycling did not increase acute pain in either group. Deyle et al. (48,187) evaluated the effectiveness of a program combining manual therapy and range-of-motion, strengthening, stretching, and aerobic (stationary bicycle and walking) exercises for 4 wk on 83 patients with knee OA randomized in active and placebo treatment groups. At 1 yr, the treated patients showed clinically and statistically significant gains over baseline scores on knee function, pain and stiffness, as well as on walking distance. Twenty percent of the patients in the placebo group and 5% of the patients in the treatment group had undergone knee arthroplasty. Pertinent information of these two studies is included in Table 6.

In conclusion, there is evidence that PA, especially in the form of supervised exercises, is effective in the treatment and rehabilitation of patients with knee OA (Category B).

TABLE 3. Association of OA and level or exposure time of PA in sports and recreational activities.[a]

Author	Type of Study	Population	Osteoarthritis	Physical Activity	Association	Comment
Roos et al., 1994 (210)	Case-control	71 elite and 71 nonelite ex-soccer players, 142 age-matched controls, age $\bar{x}$ 61–63 yr.	Radiographically confirmed knee OA	Soccer playing	Prevalence of OA: 15.5% in ex-elite, 4.2% in ex-nonelite players, and 2.8% in controls. Exclusion of subjects with knee injuries: higher rate among ex-elite players but no difference between ex-nonelite players and controls.	The increased prevalence of knee OA in ex-elite soccer players without diagnosed knee injuries suggests that the sport itself at this level may result in OA.
Sandmark and Vingård, 1999 (216)	Case-referent	Study population: all men born between 1921 and 1938 and living in 14 counties during the period 1991–1995. Cases: subjects who had knee prosthetic surgery because of primary OA in 1991–1993, 325 men and 300 women. Referents: randomly selected from the study population, 264 men and 284 women. Subjects with trauma, surgery or disease affecting the knee region were excluded.	Primary OA of the knee requiring prosthetic surgery	The same procedures as in the two studies above	Relative risk of developing severe knee OA was 2.9 [1.3–6.5] in men aged 55–65 yr who were highly exposed to all kinds of sports. Cross-country skiing, soccer, ice hockey and bandy, and track and field increased the risk in men but jogging did not. Occupational loading added to the risk attributable to sports. No associations were found in women.	Moderate daily generally was not associated with risk of knee OA.
Spector et al., 1996 (230)	Retrospective cohort study	81 female ex-athletes, 40–65 yr, and 977 age-matched female controls invited to clinical and radiological examination.	Radiographically confirmed hip or knee OA	Detailed information of current and past participation in sports and other physical activities by nurse- or self-administered questionnaire	Age, height, and weight adjusted OR for OA of patella-femoral joint was 2.97 [1.15–7.67] and OR for OA of hip was 1.60 [0.73–3.48] in ex-athletes. Within the control group, a subgroup reporting long-term vigorous exercise had risks of OA similar to those of ex-athletes.	No clear risk factors were seen in the ex-athletes.
Vingård et al., 1993 (254)	Case-control	Study population: all 50 to 70-yr-old men in the referral area of four hospitals. Cases: men who received a total hip replacement because of primary OA during 4 yr, $N = 247$. Controls: randomly from the population, $N = 322$. Subjects with history of injury excluded.	Primary OA of hip requiring prosthetic surgery	Detailed information of participation in various sports by interview, each exposure (in hours) was aggregated to the age of 49 and also in two periods: to 29 and 30–49 yr of age	Risk of developing hip OA increased with medium exposure (total number of hours) (RR, 1.3–2.6) and high exposure (RR, 2.8–4.5) but not with years of low exposure. Medium and high occupational loading added to the risk because of sport loading (both high; RR, 8.5 [4.0–17.9]).	Most hazardous sports seemed to be track and field sports (RR, 3.7) and racket sports (RR, 3.3).
Vingård et al., 1998 (255)	Case-control	Study population: all 50 to 70-yr-old women in five counties. Cases: women who had received total hip replacement because of primary OA during 4 yr, $N = 230$. Controls: randomly from the population, age-matched, $N = 273$.	Primary OA of hip requiring prosthetic surgery	As in the study above, but aggregation of exposure to age 50	Relative risk of developing hip OA was 2.3 [1.5–3.7] for those with high and 1.5 [0.9–2.5] for those with medium sport exposure. Medium and high occupational load added to the risk because of sport loading (both high; RR, 4.3 [1.7–11.0]).	Physical load from sporting activities seems to be a moderate risk factor for women for development of severe OA of the hip.

RR, relative risk; OR, odds ratio.

[a] 95% confidence intervals are given in brackets. $\bar{x}$ denotes mean.

Only limited evidence is available regarding hip OA. The evidence comparing effectiveness of different exercise regimens is inconclusive. Dose-response relationships have been reported in only one RCT and it suggests that low-intensity stationary bicycling is as effective as higher intensity stationary biking (Category B).

Recommendations for research. Intensive PA, particularly sports that include rapid movements, high impacts,

TABLE 4. Studies of running and risk of developing OA of the hips or knees.[a]

Reference	Subjects	Mean Age (yr)	Training		Site	Diagnostic Method	Risk and Comments
			Yr	Amount			
Puranen et al., 1975 (201)	74 champion runners	56	21		Hip	Clinical, radiographic	No more hip OA than in nonrunners
McDermott and Freyne, 1983 (168)	20	35	13	48 miles·wk^{-1}	Knee	Radiographic, clinical	High incidence (30%) of knee OA, attributed to previous injuries and anatomic abnormalities
Sohn and Micheli, 1985 (229)	504 ex-runners	57	9–15	18–29 miles·wk^{-1}	Hip, knee	Clinical	No association between moderate long distance running and development of OA
Lane et al., 1986 (133)	41 distance runners	58	9	4.5 h·wk^{-1}	Hip, knee	Radiographic, clinical	No difference to controls in clinical OA among male or female runners
Panush et al., 1986 (191)	17 ex–long-distance runners	53	12	28 miles·wk^{-1}	Hip, knee	Radiographic	Comparable low prevalence of OA in runners and matched controls
Lane et al., 1987 (134)	498 distance runners	59	12	27 miles·wk^{-1}	Hip, knee	Clinical	No difference to controls in conditions thought to predispose to OA
Marti et al., 1989 (163)	27 ex–long-distance runners	42	20	50–100 km·wk^{-1}	Hip	Clinical, radiographic	Incidence of moderate to severe OA 16% in runners, 0% in controls; best predictors of OA: age, distance run, speed
Konradsen et al., 1990 (124)	27 ex–long-distance runners	58	40	20–40 km·wk^{-1}	Hip, knee	Clinical, radiographic	No significant differences in the signs of OA between runners and matched controls
Kujala et al., 1994 (128)	199 ex–elite long-distance runners	50 at entry	Not given	Not given	Hip, knee, ankle	Hospital admissions because of OA	Age-adjusted OR for hospitalization 1.84 (0.93–3.61) in ex-runners compared to controls; mean age at first admission 70.3 yr, in controls 61.2 yr
Kujala et al., 1995 (129)	28 ex-runners	60	30	Total 9500 h	Knee		Risk of developing OA in runners not increased (RR, 1.06)
Lane et al., 1995 (132)	35 nonelite long-distance runners	65	10–13	3 h·wk^{-1}	Hip, knee	Clinical, radiographic	Same incidence of OA in runners and matched controls
Lane et al., 1998 (137)	28 nonelite long-distance runners	66	17	2 h, 18 miles·wk^{-1}	Hip, knee	Clinical, radiographic	Radiographic hip OA and progression of radiographic knee OA similar for runners and matched controls

[a] See also Spector et al. 1996 (230); Sandmark and Vingård, 1999 (216) in Table 3; and Cheng et al., 2000 (31), in Table 5.

torsional loads, and injuries on joints are shown to increase substantially the risk of development of OA. An important task for research is to develop measures to decrease the size of impacts and torsional loadings on joints in PA, especially in running and ball games, as well as to develop measures to decrease the risk of joint injuries.

Short-term effectiveness of PA in secondary prevention of knee OA is shown. Long-term effectiveness of PA should be tested and comparison of effectiveness of different regimens is needed. Especially important is comparison of low-intensity and higher intensity activities on various aspects of OA. These aspects should be studied also in patients with OA of the hip.

OP

Background. OP is a disease characterized by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk. These changes can be assessed indirectly through noninvasive measurement of areal bone mineral density (BMD), measured in grams per square centimeter. Bone density accounts for 75–85% of the variance in ultimate strength of bone tissue. Low bone mass by itself causes few, if any, symptoms except in severe OP. Thus, OP is defined most commonly by an intermediate outcome (BMD), not by a health outcome (e.g., fracture) (177).

BMD is considered normal if it is no lower than 1 SD below the mean for young adult women. BMD between 1.0 and 2.5 SD below the mean indicates osteopenia or low bone mass and BMD 2.5 SD or more below the young adult means indicates osteoporosis (268). Fracture risk increases 1.5- to 3-fold for each SD fall in BMD (162). Age-adjusted risk of hip fracture is increased 2.4- to 3-fold for each 1 SD decline in BMD (42). One SD is around 10% of BMD. Prevention of OP can be defined as preventing BMD from dropping lower than 2.5 SD below the mean for young adult women. Two factors determine the amount of bone later in

TABLE 5. Association of OA and PA in various populations.[a]

Author	Type of Study	Population	Osteoarthritis	Physical Activity	Association	Comment
Imeokparia et al., 1994 (99)	Case-control	Cases: 85 men, 154 women Controls: randomly from the same community sample, 85 men, 154 women. Age in all groups: mean 66–67 yr	Knee radiographically confirmed	Retrospectively by interview, occupational, leisure-sport, home-based, four levels in each by estimated energy expenditure, collapsed to high and low	In high PA women age-adjusted OR 1.66 [1.0–2.72], persisted when controlled for confounders. No significant association in men. History of knee injury did not alter the risk estimations.	In women, risk increased slightly with increased BMI.
Lane et al., 1999 (138)	Cross-sectional	5818 women over 65 yr, age $\bar{x}$ 72 yr, recruited from populations of four areas	Hip, radiographically confirmed and symptomatic	Detailed recreational PA retrospectively by questionnaire	For women in the highest quartile of PA (1–5×/wk vs lowest quartile) OR significantly increased [1.4–1.7 depending on age at the time of exposure]. Corresponding findings regarding symptomatic OA.	Moderate participation in recreational PA was associated with OA of hip.
Framingham Study						
Hannan et al., 1993 (81)	Longitudinal cohort study	1404 subjects (584 men, 820 women), mean age 73 yr	Knee, radiographically confirmed	Amount and intensity questioned by interview at examinations 29 and 12 yr prior to the diagnostic procedures	No significant association between habitual PA and OA of knee in men or women.	Habitual physical activity was not found to be a risk factor for knee OA.
Felson et al., 1997 (64)	Longitudinal cohort study, follow-up time 9 yr	598 subjects without knee OA at baseline, mean age 70.5 yr, 63.7% women; part of the cohort of the previous study	Knee, radiographically confirmed 8–9 yr after the baseline examination	Amount and intensity questioned by interview at examinations 29 and 12 yr prior to the baseline assessment and at 4 yr during the follow-up	OR for OA increased significantly with increasing habitual PA being 3.3 [1.4–7.5] in the highest quartile. Risk increased in subjects both with and without knee symptoms at baseline or during follow-up.	High BMI at baseline and increase of weight during follow-up increased the risk of knee OA especially in women.
McAlindon et al., 1999 (166)	Longitudinal cohort study	470 subjects (177 men, 293 women) without knee OA at baseline, mean age 70.1 yr	Knee, radiographically confirmed and symptom-based 8–9 yr after the baseline examination	Detailed inquiry of the type, amount, and intensity of PA about in the middle of the follow-up	Increasing amount of heavy PA was associated with increased risk, e.g., OR for $\geq$4 h·d^{-1} compared with no heavy PA = 7.0 [2.4–20]. No association with light or moderate PA and knee OA.	Increase of BMI increased the risk of knee OA.
Cheng et al., 2000 (31)	Prospective, mean follow-up 10 yr	16,961 subjects, 20–87 yr examined at Cooper Clinic	Physician-diagnosed knee or hip; OA reported by the subject in follow-up survey	Amount of walking/jogging or other regular PA at baseline as reported by the subject	Hazard ratio 2.4 [1.5–3.9] (controlled for other potential risk factors) in men under age 50 reporting high levels of PA (running 20 or more miles·wk^{-1}). No association among older men or women.	Increasing BMI increased the risk especially in women; moderate-intensity PA levels recommended by recent public health guidelines are not likely to increase risk of hip or knee OA.

[a] 95% confidence intervals are given in brackets. $\bar{x}$ denotes mean.

life: the bone mass accumulated during youth (peak bone mass) and the subsequent rate of bone loss.

PA can influence bone mass by causing compressive or bending loads on bone. These can cause a temporary deformation, strain, in bone leading to primary and secondary responses in bone that stimulate bone formation. These responses have been shown to be proportional to the load in cell and organ cultures and in animal models (225). The purpose of these responses can be thought of as parts of a homeostatic mechanism that aims to keep deformation of bone because or mechanical loading within narrow limits. High loads lead to increased bone formation and decreased bone loss and low loading leads to decreased bone formation and increased resorption (67). Findings from animal studies support the concept of bone as a "mechanostat" (243). Also, human studies ranging from the effects of immobilization and inactivity through sedentariness to active lifestyle, recreational exercise, and intensive sports practice show decrease, maintenance, and increase of BMD corresponding to this model. However, the stress-strain relationships regarding bone's adaptation are complex. Current knowledge derived especially from animal experiments suggests that bone's adaptation to loading is determined by three basic rules (243): 1) dynamic rather than static loading is effective; 2) only short duration of mechanical loading is necessary to initiate an adaptive response and the capacity of bone tissue to respond to the stimulus at one time is saturated by few loading cycles; and 3) bone cells accommodate to a customary loading, making them less responsive to routine loading signals.

Application of these basic rules to practical exercise regimens means that each session should include movements causing high loads at high rates, i.e., they should be forceful and fast, and they should load the bone from variable directions, but the number of movements need not be great. The loading can be caused by gravitational forces or by muscular contractions. The forces have to focus specifically on the targeted bone area in order to be

TABLE 6. RCTs on the effectiveness of exercise training in patients with OA of the hip or knee.

Reference	N	Subjects	Exercise Training: Mode and Intensity	Frequency	Duration	Results
Minor et al., 1989 (171)	80	64 yr, males and females, OA of hip, knee	T1: walking, 60–80% HRmax T2: aerobic aquatics, R: range of motion (control)	3×/wk	12 wk	The walking and aquatics exercise groups showed significant improvement over the control group in aerobic capacity, 50-foot walking time, depression, anxiety, and PA.
Kovar et al., 1992 (126)	102	70 yr, males and females, OA of knee	T: walking + patient education R: usual care (control)	3×/wk, 30 min	8 wk	Significant 18% improvements in walking distance, 39% improvement in functional status, 27% decrease in pain in the walking group, significantly different from the changes in the control group.
Börjeson et al., 1996 (19)	68	64 yr, OA of knee	T: exercises to increase strength and ROM R: no treatment	3×/wk, recommended to be done also at home	5 wk	Significant improvement of perceived knee status and of descending steps in T, not in R. Only small objective improvements in functions.
Schilke et al., 1996 (219)	29	65 yr, most females, OA of knee	T: muscle strengthening R: nonexercising control group	3×/wk, 6 sets of 5 maximum contractions	8 wk	Significant subjective improvements in pain, stiffness, mobility, and arthritis activity, more improvement in strength measures in the experimental as compared to the control group.
Bautch et al., 1997 (10)	30	69 yr, OA of knee	T: ROM exercises, individualized low-intensity walking, education R: education	3×/wk, 1 h 1×/wk, 1 h	12 wk 12 wk	Significant decrease in pain in T, no change in R.
Ettinger et al., 1997 (59)	439	70 yr, males and females, OA of knee	T1: walking, 50–70% HRR T2: calisthenics R: health education	3×/wk, 40 min	3 mo facility + 15 mo home	Modest but consistent improvements in self-reported pain and disability and better scores on performance measures of function compared with those participating in health education programs. Greater improvements in those completing more exercise.
van Baar et al., 1998 (247)	201	40–85 yr, males and females, OA of hip or knee	T: individualized exercises for muscle strength and length, mobility, coordination, movement abilities, and locomotion administered by physiotherapist	12 wk		Significantly larger decrease of pain (medium effect size) and observed disability (small effect size) in T than in R.
Mangione et al., 1999 (157)	39	71 yr, males and females, OA of knee	Stationary cycling T1: 70% HRR T2: 40% HRR	3×/wk, 25 min	10 wk	Both T1 and T2 improved significantly and similarly in several objectively measured functions and in the amount of overall pain. Cycling did not increase acute pain in either group.
O'Reilly et al., 1999 (187)	191	62 yr, males and females with knee pain	T: strengthening exercises R: no intervention	Daily at home	6 mo	Pain scores and physical function scores improved more in T than in R.
Deyle et al., 2000 (48)	83	60 (T), 62 (R) yr, males and females, OA of knee	T: manual therapy and supervised knee exercise program (stretching, ROM, and strengthening exercises) R: placebo	2×/wk and at home	4 wk	In T, significant improvement in walking performance and standardized OA assessment score over baseline at 4 and 8 wk and 1 yr, not in R. By 1 yr, patients in R had significantly more knee surgeries than patients in T.

T, training; R, reference group; ROM, range of motion.

effective. Loading has to repeat frequently and continuously in order to maintain BMD, and loading has to increase periodically if the aim is to stimulate continuing increase of bone mass.

Review of research evidence. Peak bone mass accumulated in youth and the subsequent rate of bone loss are thought to be equally important in determining bone mass at age 70. Numerous cross-sectional studies in athletes (258) (Tables 7–9) and in population samples (37,54,97,104,183, 239,245,246,262) (Table 10), some longitudinal studies (4,12,36,39,142,161,173,202) (Tables 11 and 12), and at least seven RCTs (17,20,66,77,172,227,265) (Table 13) and a

TABLE 7. Bone mineral mass in female athletes taking part in high-magnitude loading sports (results selected from cross-sectional studies).

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
Flodgren et al., 1999 (65)	Flatwater sprint kayakers	10 (6 M, 4 F) (6 M, 4 F)	19	Total body		DXA
				Head		
	Matched controls	20	19	Ribs	6.4*	
				Humerus (L/R)	10.4*/11.7*	
				Legs L/R		
				Femur neck L/R		
				Femur Ward's L/R		
				Femur trochanter L/R		
				Spine	10.9*	
				Lumbar spine		
				Pelvis	5.1*	
				Arms (BMC) L/R	15.7*/10.6*	
Davee et al., 1990 (44)	Muscle-building	9	20–30	L2–L4	+10*	DPA
	Controls	9				
Heinonen et al., 1993 (87)	Weight-lifters	18	25 ± 5	L2–L4	+15*	DXA
				Femoral neck	+10*	
				Distal femur	+19*	
				Patella	+22*	
				Proximal tibia	+12*	
				Calcaneus	+4*	
				Distal radius	+29*	
	Controls	25	23 ± 3			
Heinrich et al., 1990 (92)	Body-builders	11	26 ± 5	L2–L4	+12*	DPA
				Femoral neck	+15*	
				Ward's triangle	+23*	
				Greater trochanter	+12*	
				Proximal radius	+9*	SPA
				Distal radius	+16*	
	Controls	18	25 ± 4			

M, males; F, females; L, left; R, right; DPA, dual photon absorptiometry; DXA, dual-energy x-ray absorptiometry; SPA, single photon absorptiometry.
* $P < 0.05$.

nonrandomized controlled trial (73) show evidence (Category A) that physical activity in youth can contribute to increased peak bone mass (33,52,140,257). The cited studies do not allow quantitative dose-response analysis, although their findings generally agree with the results of animal experiments regarding the characteristics of effective loading stimulus described above and allow categorization of physical activities according to their characteristics and potential to influence bone mass (258). Thus, weight-bearing activities producing high-magnitude (Table 7) and high-rate (impact) (Table 9) loadings stimulate effectively bone formation, and non–weight-bearing activities without these characteristics such as swimming even in large amounts has not been shown in any study to increase BMD at any site in female subjects (3,29,38,51,57,63, 141,188,236,238).

Unfortunately, it is not known whether and in what degree and by what kind of physical activity the increased peak bone mass attained by exercise training can be preserved for decades until old age. Limited information suggests that much or most of it may be lost during adult years (107–109), but diminution of peak bone mass can be deterred by substantially less PA than what was practiced while gaining it (6,91,118,125).

Clinically relevant studies to analyze possible dose-response relationships between PA and prevention of OP are RCTs and other controlled trials in middle-aged and elderly women with bone mass (density) as the primary outcome. Most of these studies have been analyzed in several recent thorough reviews (14,58,111,112,113,140,212,260,266). The newest analyses by Wolff et al. (266) and Wallace and Cumming (260) cover the non-RCTs to the end of 1996 and RCTs to the beginning of 1998. Pertinent information of most randomized and nonrandomized exercise training trials on bone mass in pre- and postmenopausal women is summarized in Table 14. The reviews agree well on quantitative results and on most views and can be summarized as follows. Quantitative analyses show strong evidence (Category A) that PA can be effective in maintaining bone mass in premenopausal and decreasing substantially loss of bone in postmenopausal women. This effect was 1) about $1\%\cdot yr^{-1}$ in favor of the exercising subjects, 2) mainly because of decreased rate of loss of bone in the exercisers, 3) seen both in lumbar spine and femoral neck, and 4) seen in both pre- and postmenopausal women. Both endurance or aerobic and strength or progressive resistance training as well as impact and nonimpact exercises can be effective, and no definite differences between the types of training were seen. However, only a few studies were available to analyze the effect of nonimpact exercise or strength training on the hip in premenopausal women. The results of studies using high-intensity strength training were more consistent and showed positive effects compared with those using low-intensity resistance training (140). The size of the effect of exercise training on bone mass was found to be comparable to that of calcium supplementation.

The reviews pointed out several shortcomings of the studies such as heterogeneity of subjects, training programs, and measurements; small sample sizes; and short duration and inadequate description and quantification of the training programs. In general, the training program characteristics,

TABLE 8. Bone mineral mass in female athletes taking part in repetitive loading (weight bearing) sports (results selected from cross-sectional studies).

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
Dook et al., 1997 (51)	Running and field hockey	20	46 ± 3	Whole body	+7*	DXA
				Regional leg	+7*	
				Regional arm	+3	
	Sedentary controls	20	46 ± 2			
Harber et al., 1991 (83)	Eumenorrheic runners	17	27 ± 5	Calcaneus	–2	CST
	Amenorrheic runners	11	26 ± 6	Calcaneus	0	
	Normoactive controls	14	27 ± 7			
Heinonen et al., 1993 (87)	Orienteers	30	23 ± 3	L2–L4	0	DXA
				Femoral neck	+2	
				Distal femur	+5*	
				Patella	+3*	
				Proximal tibia	+4*	
				Calcaneus	+4	
				Distal radius	+1	
	Cross-country skiers	28	21 ± 3	L2–L4	0	
				Femoral neck	+5	
				Distal femur	+5	
				Patella	+2	
				Proximal tibia	+3	
				Calcaneus	+3	
				Distal radius	–1	
	Active controls	25	23 ± 3			
Heinonen et al., 1995 (88)	Orienteers	30	23 ± 3	L2–L4	+8	DXA
				Femoral neck	+3	
				Distal femur	+6*	
				Patella	+4	
				Proximal tibia	+7*	
				Calcaneus	+11	
				Distal radius	–4	
	Speed skaters	14	21 ± 9	L2–L4	+6	
				Femoral neck	+4	
				Distal femur	+7*	
				Patella	+5	
				Proximal tibia	+6	
				Calcaneus	+3	
				Distal radius	–6	
	Cross-country skiers	28	21 ± 3	L2–L4	+3	
				Femoral neck	+5	
				Distal femur	+5	
				Patella	+2	
				Proximal tibia	+5*	
				Calcaneus	+9*	
				Distal radius	–7*	
Kirk et al., 1989 (119)	Long-distance runners	10	25–35	T12–L3	+1	OCT
	Sedentary controls	10				
	Long-distance runners	9	55–65	T12–L3	–7	
	Sedentary controls	9		Midradius	+1	
Lane et al., 1986 (133)	Long-distance runners	6	56	L1	+35*	OCT
	Matched controls	6	56			
Myerson et al., 1992 (176)	Eumenorrheic runners	13	30 ± 1	Total body	+10*	DPA
	Amenorrheic runners	13	30 ± 1	Total body	–7	
	Controls	12	27 ± 1			
Petterson et al., 2000 (194)	Cross-country skiers	16	16 ± 0.3	Total body	+1.8	DXA
	Normoactive controls	16	16 ± 0.7	Head	+0.9	
				Humerus right	+6.9*	
				Humerus left	+9.2*	
				Humerus diaphysis right	+5.1	
				Humerus diaphysis left	+8.1*	
				Spine	–0.9	
				Femoral neck	+8.9*	
				Femoral diaphysis	+7.6*	
				Distal femur	+5.0	
				Greater trochanter	+9.3*	
				Proximal tibia	+6.6	
				Tibia diaphysis	+2.1	
Suominen et al., 1992 (235)	Long-distance runners, skiers	18	66 –85	Calcaneus	+12 and +6	SPA
	Population sample	42	70–81			
Wolman et al., 1991 (267)	Runners	21	25–28	Femoral shaft	+8*	DPA
	Rowers	36	24–26	Femoral shaft	+2	
	Sedentary controls	13	27–30			

CST, Compton scattering technique; DPA, dual photon absorptiometry; DXA, dual-energy x-ray absorptiometry; QCT, quantitative computed tomography; SPA, single photon absorptiometry.

* $P < 0.05$.

TABLE 9. Bone mineral mass in female athletes taking part in sports producing strains on bones at a high rate (e.g., impacts) and usually from many directions (results selected from cross-sectional studies).

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
Alfredson et al., 1997 (1)	Volleyball	13	21 ± 4	Whole body	+6.1*	DXA
				Head	–1.8	
	Matched normoactive controls	13	25 ± 2	Lumbar spine	+13.2*	
				Femoral neck	+15.8*	
				Ward's triangle	+17.9*	
				Trochanter	+18.8*	
				Whole femur dominant/nondominant	+5.8/+8.2*	
				Humerus dominant/nondominant	+9.5*/10.0*	
Courteix et al., 1998 (38)	Gymnasts	18	10 ± 1	Whole body	+14.4*	DXA
				Midradius	+15.5*	
	Controls	13	11 ± 1	Distal radius	+33	
				L2–L4	+11*	
				Femoral neck	+15*	
				Trochanter	+11	
				Ward's triangle	+15*	
Dook et al., 1997 (51)	Basketball and netball	20	46 ± 3	Whole body	+8*	DXA
				Regional leg	+8*	
				Regional arm	+4	
	Sedentary controls	20	46 ± 2			
Düppe et al., 1996 (53)	Active football players	96	18 ± 4	Total body	+4*	DXA
				Lumbar spine	+5	
				Femoral neck	+11*	
				Trochanter	+11*	
				Ward's triangle	+11*	
	Controls	90	20 ± 5			
	Former football players	25	40 ± 5	Total body	+4*	
				Lumbar spine	–2	
				Femoral neck	+7*	
				Trochanter	+11*	
				Ward's triangle	+9*	
	Controls	57	37 ± 4			
Fehling et al., 1995 (63)	Volleyball players	8	20 ± 1	L2–L4	+11*	
				Femoral neck	+15*	
				Ward's triangle	+17*	
				Total body	+13*	
				Left arm	+5*	
				Right arm	+7*	
				Left leg	+15*	
				Right leg	+12*	
				Right pelvis	+19*	
	Gymnasts	7	20 ± 1	L2–L4	+14*	
				Femoral neck	+15*	
				Ward's triangle	+17*	
				Total body	+11*	
				Left arm	+15*	
				Right arm	+16*	
				Left leg	+10*	
				Right leg	+10*	
				Right pelvis	+15*	
	Controls	13	20 ± 1			
Haapasalo et al., 1994 (79)	Squash players	19	18–32	Proximal humerus	+9*	DXA
				Humeral shaft	+10*	
				Radial shaft	+1*	
				Ulnar shaft	–1*	
				Distal radius	+10*	
				Distal ulna	+24*	
				Calcaneus	+12*	
	Controls	19	19–33			
Heinonen et al., 1995 (88)	Squash players	18	25 ± 4	L2–L4	+14*	DXA
				Femoral neck	+17*	
				Distal femur	+11*	
				Patella	+7*	
				Proximal tibia	+13*	
				Calcaneus	+19*	
				Distal radius	+11*	
	Aerobic dancers	27	28 ± 4	L2–L4	+3	
				Femoral neck	+9*	
				Distal femur	+3	
				Patella	+2	
				Proximal tibia	+6*	
				Calcaneus	+14*	
				Distal radius	–8*	
	Controls	25	24 ± 5			

TABLE 9. *Continued*

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
Jacobson et al., 1984 (101)	Tennis players	11	18–22	Spine	+11*	DPA
				Distal radius	+17*	SPA
				Midradius	+12*	
				Metatarsal	+23*	
	Age-matched controls	11				
Kannus et al., 1995 (106)	Tennis and squash players	105	16–50	Proximal humerus	+10*	DXA
				Humeral shaft	+10*	
				Radial shaft	+3	
				Distal radius	+8*	
				Calcaneus	+11*	
	Controls	50	16–48			
Kirchner et al., 1995 (117)	Gymnasts	26	20 ± 0.2	Lumbar spine	+18*	DXA
				Total proximal femur	+21*	
				Femoral neck		
				Ward's triangle		
				Whole body	+22*	
					+25*	
					+10*	
	Controls	26	20 ± 0.2			
Kirchner et al., 1996 (118)	Former gymnasts	18	36 ± 1	Lumbar spine	+16*	DXA
				Femoral neck	+18*	
				Ward's triangle	+22*	
				Whole body	+9*	
	Controls	15	37 ± 1			
Lee et al., 1995 (141)	Volleyball players	11	19 ± 1	Total body	+17	DXA
				Lumbar spine	+18	
				Femoral neck	+11	
				Trochanter	+17	
				Ward's triangle	+4	
				Spine	+6*	
				Pelvis	+10*	
				Left arm	+12*	
				Right arm	+13*	
				Left leg	+17*	
				Right leg	+15*	
	Basketball players	7	20 ± 2	Total body	+9*	
				Lumbar spine	+14*	
				Femoral neck	+20*	
				Trochanter	+24*	
				Ward's triangle	+18*	
				Spine	+5*	
				Pelvis	+11*	
				Left arm	+12*	
				Right arm	+17*	
				Left leg	+15*	
				Right leg	+17*	
	Soccer players	9	19 ± 1	Total body	+4	
				Lumbar spine	+6*	
				Femoral neck	+10*	
				Trochanter	+16*	
				Ward's triangle	+12	
				Spine	+5	
				Pelvis	+7*	
				Left arm	+1	
				Right arm	+3	
				Left leg	+11*	
				Right leg	+11*	
	Sedentary controls	11	22 ± 1			
Nichols et al., 1994 (180)	Gymnasts	11	19 ± 1	Preseason:		DXA
				Lumbar spine	+8*	
				Femoral neck	+11*	
	Controls	11	21 ± 2			
Nichols et al., 1995 (181)	Basketball players	14	19 ± 1	L2–L4	+10*	DXA
				Femoral neck	+14*	
				Total body	+10*	
				Leg	+15*	
				Arm	+8*	
	Gymnasts	15	19 ± 1	L2–L4	+9*	
				Femoral neck	+11*	
				Total body	+5*	
				Leg	+7*	
				Arm	+12*	

TABLE 9. *Continued*

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
	Tennis players	6	23 ± 4	L2–L4	+4*	
				Femoral neck	+3*	
				Total body	+6*	
				Leg	+9*	
				Arm	+6*	
	Volleyball players	13	19 ± 1	L2–L4	+13*	
				Femoral neck	+14*	
				Total body	+10*	
				Leg	+15*	
				Arm	+8*	
	Nonathletes	12	21 ± 2			
Nichols-Richardson et al., 2000 (182)	Gymnasts	16	10 ± 2	Whole body		DXA
				Femur		
	Matched controls	16	10 ± 1	L1–L4		
Pearce et al., 1996 (192)	Ballet dancers: <40 mo oligomenorrhea	17	14 ± 0.2	Lumbar spine	–2	DXA
				Femoral neck	+9*	
				Ward's triangle	+10*	
				Trochanter	+9*	
				Arms	–4	
	>40 mo oligomenorrhea	24	18 ± 0.2	Lumbar spine	–4	
				Femoral neck	+4	
				Ward's triangle	+4	
				Trochanter	+2	
				Arms	–8	
	Controls	46	18 ± 0.2			
Pettersson et al., 2000 (195)	Rope-skipping	10	18 ± 1	Whole body	+7*	DXA
				Humerus, right	+10*	
	Normoactive controls	25	18 ± 1	Lumbar spine	+14*	
				Femoral neck	+10*	
				Trochanter	+14*	
				Total femur	+4*	
				Femur diaphysis	+8*	
				Tibia diaphysis	+20*	
				Radius UD	+33*	
				Radius proximal	6	
Rissner et al., 1990 (207)	Volleyball players	12	20 ± 2	Lumbar spine	+15*	DPA
				Calcaneus	+26*	SPA
	Basketball players	9	20 ± 1	Lumbar spine	+12*	
				Calcaneus	+36*	
	Nonathletes	13	20 ± 2			
Robinson et al., 1995 (208)	Gymnasts	21	22 ± 3	Whole body	+2	DXA
				Lumbar spine	+6	
				Femoral neck	+12*	
	Controls	19	19 ± 2			
Sandström et al., 2000 (217)	Ice hockey	14	22 ± 4	Whole body	+7*	DXA
				Head	–3	
	Normoactive controls	14	22 ± 4	Lumbar spine	+9*	
				Femoral neck	+18*	
				Ward's triangle	+20*	
				Trochanter	+22*	
Slemenda and Johnston, 1993 (223)	Figure skaters	22	10 – 23	Total body	+7*	DXA
				Spine	+6	
				Trunk	+8*	
				Legs	+10*	
				Pelvis	+14*	
				Arm	+4	
	Controls	22	10 – 23			
Söderman et al., 2000 (228)	Soccer	41	16 ± 0.3	Whole body	+3*	DXA
				Head	–2	
	Normoactive matched controls	41	16 ± 1	L2–L4	+6*	
				Femoral neck, dominant	+10*	
				Ward's triangle	+10*	
				Trochanter	+17*	
Taaffe et al., 1995 (236)	Gymnasts	13	19 ± 1	Whole body	+3	DXA
				Lumbar spine	+8	
				Femoral neck	+15*	
				Trochanter	+15*	
	Controls	19	19 ± 2			
Taaffe et al., 1997 (237)	Gymnasts, 8-mo cohort	26	20 ± 1	Whole body	+1	DXA

TABLE 9. *Continued*

Reference	Athlete Group	*N*	Age (yr)	Bone Site	Bone Mineral Mass: Difference Between Athletes and Controls (%)	Technique
				Lumbar spine	+6	
				Femoral neck	+14	
	Controls	14	19 ± 2			
	Gymnasts, 12-mo cohort	8	19 ± 1	Whole body	+4	
				Lumbar spine	+4	
				Femoral neck	+20*	
	Controls	11	20 ± 2			

DPA, dual photon absorptiometry; DXA, dual-energy x-ray absorptiometry; SPA, single photon absorptiometry.
* $P < 0.05$.

such as its duration and the background characteristics of the subjects (i.e., hormone replacement, smoking, or use of alcohol), did not influence the results substantially in the primary and subgroup analyses. However, low compliance was considered to explain poor results in some studies. The design of the study seemed to influence the results substantially, because in the review of Wolff et al. (266), the effect of training was almost twice as high in non-RCTs compared with RCTs.

Since the completion of the above-cited reviews, new RCTs (8,46,80,90,91,155,197,215), randomized trials without a control group (98,123), and nonrandomized, controlled studies (100,264) on middle-aged and older women and men have been published. The aims, subjects, exercise programs, and measurements are also heterogeneous in these studies. Regarding dose-response issues, the studies in general support the notion that high-intensity exercise can increase or maintain bone mass in premenopausal women (8,90,264) and elderly men (155), but that high-intensity resistance training was not effective in postmenopausal women not on hormone replacement therapy (166), and the same high-impact exercise program that was effective in premenopausal was not effective in postmenopausal women (8). Heinonen et al. (90) found that a multiexercise intensive (about 70% $\dot{V}O_{2max}$) endurance program consisting of walking, stair climbing, ergometer cycling, and jogging for 18 months in perimenopausal women resulted in maintenance of prestudy femoral neck BMD, and the trend of change was significantly different from that in the control group. A calisthenics (dynamic strength-endurance exercises for trunk, pelvis, hip, and lower limbs) program was not effective regarding maintenance of bone mass at the trained sites. The proposed explanation for the findings is that the strength-endurance exercises did but the calisthenics exercise did not fulfil the conditions for effective osteogenic stimulus in terms of the magnitude, rate, and distribution of the stress applied on bones. An extension study of an RCT showing positive effect of high-impact exercise on femoral neck BMD in premenopausal women supports the idea that the results on bone can be maintained with less-demanding training than that used in the original program (91).

Humphries et al. (98) did not find changes in lumbar spine BMD in older women taking or not taking hormone replacement therapy in a high-intensity strength training program despite substantial increase in muscle strength, but the duration of the program was only 24 wk. Kohrt et al. (123) compared supervised high-intensity resistance endurance and physiotherapy program for 9 mo in elderly women and men with physical frailty and found significant increase or trend of increase on hip and spine BMD in the high-intensity group and decreasing trend in the home exercise group in changes of whole body and Ward's triangle BMD. The differences between the group changes were statistically significant. None of the studies cited above showed a moderate- to low-intensity exercise program to influence bone mass in pre- or postmenopausal women, in frail elderly persons (46), or in patients with rheumatoid arthritis (80), except for one study. Iwamaoto et al. (100) found that, in postmenopausal osteoporotic women on calcium and vitamin D_3 supplementation, daily outdoor walking (documented by step counters) and gymnastics for 12 mo increased lumbar spine BMD significantly in relation to a nonexercising control group.

PA is related not only to increased but also to decreased bone mass, particularly in women. A large volume of intensive training for prolonged periods such as seen especially in competitive runners can lead to osteopenia. The mediating mechanisms are complicated and not completely known, but one mechanism is disruption of normal ovarian function that leads to inhibition of the production of gonadotropin-releasing hormone by the hypothalamus and to decreased estrogen content in blood. These athletes have oligorrhea or amenorrhea and gradually bone mass decreases, or in young athletes peak bone mass remains low. As a consequence, the risk of stress fractures increases, but it is not definitively known if the risk of osteoporosis in later life is also increased (11,71). In addition to hormonal disturbances, low body mass and, frequently, eating disorders can contribute to athletes' osteopenia. No quantitative dose-response relationship regarding this condition has been defined.

Concluding remarks. The available information from published studies does not allow definition of any quantitative dose-response relationships between PA and bone mass, a surrogate indicator of OP. Basic bone research indicates the existence of dose-response relationships but also that they are complicated because of the fact that several factors in the dose, especially the magnitude, rate, and directions of the loads, determine the response and these relationships change with the adaptation of the bone to a

TABLE 10. Studies on association of bone mass and PA in various population samples.

Reference	Type of Study	Population	*N*	Age (yr)	PA	Bone Site, Method	Findings	Comments
Cooper et al., 1995 (37)	Cross-sectional (for PA)	Women born in a city during 2 yr	153	21	Several indices of past and current PA by interview	Lumbar spine, femoral neck, DXA	Dose-related association between lumbar (5% difference between least and most active, $P = 0.07$) and femoral neck (12% difference $P < 0.01$) BMD and amount (min·d^{-1}) of outdoor leisure walking.	PA was the strongest lifestyle determinant of BMD.
Düppe et al., 1997 (54)	Cross-sectional	Random sample of the 15 to 42-yr-old population of a city	395	15–42	Current leisure time, occupational, and transport PA by validated questionnaire	Spine, proximal femur, DXA	No association between BMD and PA in 15 to 16-yr-old subjects, but in 21 to 42-yr-old men significantly (9%) higher femoral BMD in highly active (vs least active) subjects. In women similar but nonsignificant trend.	Occupational activity showed similar but weaker association to femoral BMD.
Ho et al., 1997 (97)	Cross-sectional	Invited sample of women clients of a medical clinic	273	21–40	Minnesota Leisure Time PA questionnaire, previous 12 mo, activity in METs	L2–L4 proximal femur, DXA	In highly active 21 to 30-yr-old women, spine and femur BMD significantly (6–7%) higher than in less active women. No influence of PA on BMD in the older women was seen.	
Jones and Dwyer, 1998 (103)	Cross-sectional	A cohort of 8-yr-old children	330, 115 girls, 215 boys	8	Sports participation and other PA by questionnaire, aerobic capacity and strength measurements	Lumbar spine, femoral neck, total body, DXA	In boys, sports participation was associated with 4% higher hip and spine BMD. In girls, PWC_{170} was associated positively with hip and spine BMD.	
Kanders et al., 1988 (104)	Cross-sectional	Convenience sample	60 women	25–34	Minnesota Leisure Time PA questionnaire	Lumbar spine (DXA), radius (SPA)	No relation between radius BMD and PA. Vertebral BMD correlated significantly to energy expenditure of PA, $r = 0.41$. About 8% difference in vertebral BMD among less and more active women (cut point, 970 kcal·d^{-1} in PA)	Additive effect of Ca and PA.
Nordström et al., 1997 (183)	Cross-sectional	A convenience sample of men with low or moderate level of PA	33	25	Detailed history of participation in exercise and sports by interview	Total body, L2–L4, femoral neck, Ward's triangle, trochanter, humerus and head, DXA	The amount of PA (hours per week) at the time of peak bone mass attainment was significant predictor of BMD of the total body and the sites at proximal femur in these young men with low to moderate level of PA.	
Ruiz et al., 1995 (211)	Cross-sectional	A convenience sample of young girls and boys	151	7–15	Detailed data of participation in sports and exercise by interview	L1–L4, femoral head and neck, trochanter, upper third of femoral diaphysis	Weekly duration of sports activity significantly influenced vertebral and femoral sites, especially in girls and during puberty.	
Teegarden et al., 1996 (239)	Cross-sectional	Minimally active young women	204	18–31	Detailed self-reported data of PA during leisure and in occupation 5 yr before enrollment as well as sport participation in high school and college	Total body, femoral neck and spine by DXA, and radius by SPA	PA at high school or during 5 previous yr (expressed as estimated energy expenditure) were significant predictors of BMD or BMC at most measured sites.	
Valdimarsson et al., 1999 (245)	Cross-sectional	Random sample of women from one city	254	16, 18, and 20	Detailed data of sport and exercise participation during the last 3 mo by standardized questionnaire	Total body, lumbar spine, hip, and distal forearm	Especially weight-bearing activity correlated significantly with BMC and BMD of the measured sites in uni- and multivariate analyses. Positive relationship between training hours per week and total body BMD in all age groups; most marked from 0 to 3 h·wk^{-1} and among the 16-yr-olds. Thus, training >7 h·wk^{-1} associated with 4–6% greater BMD vs training <0.5 h·wk^{-1}.	

DXA, dual-energy x-ray absorptiometry; SPA, single photon absorptiometry.

TABLE 11. Longitudinal observational studies with repeated PA assessment and bone measurements only at the end of the follow-up period.

Reference	Type of Study	Population	N	Age (yr)	PA	Bone Site, Method	Findings
Kemper et al., 2000 (114)	Longitudinal cohort study, longitudinal data of PA	A cohort of school children formed in 1977	182, males and females	28	Metabolic and mechanic PA during the last 3 mo by interview, neuromotor and cardiorespiratory fitness. All variables also from previous assessments since age 13.	Lumbar, femoral neck, and radius BMD by DXA at the end of follow-up period	Both PA indicators and neuromotor (but not cardiorespiratory) fitness significantly positively related to lumbar and femoral but not to radius BMD.
Lloyd et al., 2000 (150)	Longitudinal cohort study, longitudinal data of PA	A cohort representing white adolescents attending public school in one region	81	18	Detailed sport and exercise participation between ages 12–18 by standardized questionnaire	Total body, proximal femur by DXA at the end of the follow-up period	Cumulative sport-exercise scores between ages 12 and 18 yr associated with hip BMD at age 18 yr ($r = 0.42$). The amount of PA that distinguishes a sedentary from active (nearly daily basis) teenager is related to significant (~5%) increase in peak hip BMD.
Välimäki et al., 1994 (246)	Prospective cohort study, longitudinal data of PA	Representative sample from five regions	264 (153 females, 111 males)	20–29	Summary score derived from question on weekly frequency of PA >30 min asked thrice in 12 yr prior to the bone measurement	L2–L4 and femoral neck by DXA at the end of the follow-up period	In most active quartile compared with least active after significant differences in BMD (adjusted for age and weight) were found: in men, 11% higher in femoral neck and 8% higher at lumbar spine; in women, 8% higher in femoral neck.

DXA, dual-energy x-ray absorptiometry.

given load. In accordance with these principles, there is strong evidence (Category A) that high-intensity loading in relation to the strength of the bone causes an osteogenic response specifically at the loaded bone site if the internal milieu is adequate. The evidence in also strong (Category A) showing that bone's responsiveness to external loading is highly dependent on the influence of female and male sex hormones. Evidence suggesting that low- to moderate-intensity exercise (in relation to the bone strength) causes an increased osteogenic response is weak (Category D). The characteristics of a minimum effective dose of an osteogenic stimulus are not known. Quantitative relationships regarding the rate of application of the force are not known, but static efforts and slow movements have been found to be ineffective or less effective than fast application of force (Category B). Some evidence suggests that a borderline between noneffective and effective loading in aerobic activities is around 70% $\dot{V}O_{2max}$, corresponding to fast or brisk walking or walking at or above anaerobic threshold (Category C). High-impact activities are likely to be effective (Category A). Data are not sufficient to make firm conclusions regarding the required number of loadings in one session or the interval between sessions. However, findings of studies on animals indicate a need for rather few loadings per session and rather frequent loadings, several times per week. Also, the duration of an exercise program necessary to achieve the full effect is not known, but duration of program did not influence the results of some meta-analyses. On the basis of bone biology, it is likely, however, that loading should be continued more than a half year so that its effects on BMD could be seen. Decreased loading leads to decrease of bone mass, but the dose-response relations between change of loading and change of bone mass are not known.

Recommendations for research. Elucidation of dose-response relationships between PA and bone mass requires development of detailed descriptions of the PA exposure in epidemiological studies and quantitative measures of the exercise regimens in clinical trials. Animal experiments provide a firm basis to test the effectiveness of PA on bone mass, and in agreement with the animal studies, high-impact and high-intensity activities have been shown to be effective in clinical trials. A major task for research is to develop methods to apply effective loading on the targeted (finally clinically relevant) areas. This requires innovative ideas and rational application of basic bone biological and biomechanical principles and techniques. An important research task is to increase understanding of the muscle-bone interaction and to manipulate it in such a way that the damping effect of muscles could be decreased in controlled ways, thus allowing the loading to influence the bone more directly and more effectively and more safely.

Additional adequately designed and conducted RCTs, especially on subjects with increased risk of OP, should be conducted to compare the effectiveness of different exercise regimens (doses) on BMD and other parameters related to bone strength.

Long-term (several years) RCTs should be conducted to see continuation or discontinuation of the effectiveness on bone mass of appropriate (progressive and nonprogressive) exercise regimens.

In contrast to other modalities influencing BMD (drugs, calcium), the effects of PA on bone are specific to the loaded sites. These effects on bone geometry, mass distribution, internal architecture, etc., may not be seen as significant changes in BMD, but they might have substantial effect on the breaking strength of bone. These possibilities should be tested.

The amount and other characteristics of loading needed to preserve the increased bone mass and other effects on bone

TABLE 12. Longitudinal observational studies with repeated PA assessments and bone measurements.

Reference	Type of Study	Population	*N*	Age (yr)	PA	Bone Site, Method	Findings	Comments
Bailey et al., 1999 (4)	6-yr longitudinal cohort study	A sample of school children in one city	113 (60 boys, 53 girls)	8–14 at the beginning	Detailed data at least twice a year by questionnaire	Total body, L1–L4, femoral neck annually by DXA	One yr after peak BMC velocity the most active quartile of boys exhibited 9% and girls 16% higher total body BMC compared with the respective values in the least active quartiles. Corresponding differences at the femoral neck BMC were 7% and 11%.	First study to demonstrate that growing skeleton responds to everyday PA by increased bone mineral accrual.
Bennel et al., 1997 (12)	12-mo longitudinal cohort study	A cohort of elite and subelite athletes and nonathlete controls	166 (85 men, 81 women)	17–26	Detailed information of historical PA by 5-yr periods between ages 5 and 19 by questionnaire, and current PA by questionnaire at baseline and at the conclusion of the study	Total body, upper limb, L1–L4, proximal femur, tibia/fibula, and foot by DXA	Over the 12 mo both athletes and controls showed modest increases in total body BMC and femur BMD ($P < 0.001$). Changes in bone density were independent of exercise status except at lumbar spine, were power athletes gained significantly more bone density than the other groups.	The findings provide further support for the concept that bone response to mechanical loading depends on the bone site and mode of exercise.
Cohen et al., 1995 (36)	7-mo longitudinal study	A group of novice rowers and age-matched controls	25 men	19	Detailed data of training and other PA by interviews and logs	L1–L4, femoral neck, trochanter, Ward's triangle by DXA	In rowers, BMD of L1–L4 increased by 3% ($P < 0.001$) and mean BMC by 4% ($P < 0.001$), no significant change in controls. No significant changes of BMD or BMC at femoral sites.	
Courteix et al., 1999 (39)	12-mo longitudinal study	A group of gymnasts and nonexercising children, swimmers as controls	35 women	12	Detailed data of training and other PA by interviews and logs	Whole body, L2–L4, femoral neck, intertrochanteric region, Ward's triangle, nondominant radius	Percentage changes in BMD before compared with after investigation tended to be greater in gymnasts.	
Leichter et al., 1989 (142)	14-wk longitudinal study	A sample of army recruits	223 men	18–21	Highly intensive military training at least 8 $h\cdot d^{-1}$, 6 $d\cdot wk^{-1}$	Absolute bone density (all mass constituents of bone per unit volume) of distal tibia by Compton scattering technique	Mean bone density in the right and left distal tibia increased significantly by 8%.	Because of the strenuousness of the training 45% of the subjects experienced stress fractures.
Margulies et al., 1986 (161)	14-wk longitudinal study	A sample of army recruits	268 men	18–21	Highly intensive military training at least 8 $h\cdot d^{-1}$, 6 $d\cdot wk^{-1}$	BMC of distal tibia by single-beam photon absorptiometry	Mean BMC of the left distal tibia increased by 11% and that of right tibia by 5%.	Participation in the very hard training was interrupted in 41% of the subjects, mainly because of stress fractures.
Morris et al., 1999 (173)	18-mo longitudinal study	A group of rowers and matched controls	24 women	14–15	Top-level school based rowing training in rowers, no formalized exercise outside school in controls	Total body, proximal femur, femoral neck, L2–L4 by DXA	L2–L4 bone accrual of ovulatory rowers significantly greater (BMC 8%, BMD 6%) than that of the anovulatory rowers (BMC 1%, BMD 4%) and ovulatory controls (BMC 0.5%, BMD 1%). No differences in total body or femoral bone measurements among groups.	Osteogenic benefits of rowing were less when training was associated with low estrogen and progesterone metabolite excretion.
Recker et al., 1992 (202)	Longitudinal prospective study of up to 5 yr, average 3.4 yr	A sample of students from two universities	156 women	Average 21 at entry	By accelerometer (Caltrac) for 4 d prior to each 6-mo visit	L2–L4 and forearm every 6 mo, total body twice (early in study and at last visit)	Significant bone gain at each site in these women in their third decade. The rate of spinal bone gain correlated positively with the amount of PA. Estimated effect of activity ranges from 0.3% to 8.4% between least and most active women, none of the subjects exercising heavily.	

DXA, dual-energy x-ray absorptiometry.

TABLE 13. Nonrandomized (NRCT) and RCT exercise training trials studying the effect of PA on bone mass.

Reference	Type of Study	Population	*N*	Age (yr)	PA	Bone Site, Method	Findings	Comments
Blimkie et al., 1996 (17)	6-mo RCT	A group of postmenarcheal female students in one school, randomized to exercise and control group	36 females	14–18	Resistance exercises with machines, 3×/wk Controls: usual activity	Total body, lumbar spine by DXA	No significant differences in bone measurements between exercise and control groups.	
Bradney et al., 1998 (20)	8-mo RCT (randomized schools)	A group of prepubertal male students	40 boys	8–11	Variety of weight-bearing activities Controls: regular physical education classes	Total body, lumbar spine, pelvis, leg, head, arm by DXA	Significant intervention effect in total body and lumbar spine but not in hip.	
Friedlander et al., 1995 (66)	2-yr RCT	A group of recruited healthy women randomized to exercise training and stretching control group	127 women, 63 complete the study	20–35	Supervised aerobics and weight training, stretching for controls, Ca supplementation to both groups, 3×/wk, 1 h	Lumbar spine, femoral neck, and trochanter by DXA, L1–L3 by QCT, calcaneus by SPA	Significant positive differences in BMD between exercise and stretching groups for spinal trabecular, femoral neck and trochanteric, and calcaneal measurements, and exercise group demonstrated significant gain in BMD for spinal integral (1.3%), femoral trochanteric (2.6%), and calcaneal (5.6%) measurements.	Combined aerobics and weight training has beneficial effects on BMD in young women.
Gleeson et al., 1990 (73)	12-mo NRCT, training group and matched control group	Recruited premenopausal eumenorrheic sedentary to very active subjects	68 women	Average 33 ± 6	Upper and lower extremity weight training, with machines, 3×/wk, 30 min, 60% 1 RM	Calcaneus by SPA, lumbar spine by DXA, three times	No significant changes in calcaneus. Lumbar BMD increased 0.8% (NS) in the training and decreased by 0.5% (NS) in the control group, but between matched pairs the difference was significant.	The intensity of the program was moderate and the exercises did not directly load the vertebrae.
Gutin et al., 1999 (77)	8-mo randomized, modified crossover study (PA–no PA or no PA–PA)	A group of obese children	79 obese boys and girls	7–11	Exercises with machines and games, 5×/wk, 40 min	Total body	Significant intervention effect in total body BMD.	
Morris et al., 1997 (172)	10-mo NRCT	Students of two schools assigned to training and students of two matched schools assigned to control subjects	71 premenarcheal girls	9–10	A variety of vigorous, high-impact aerobic exercises 3×/wk, 30 min per session for 10 mo	L2–L4, proximal femur, femoral neck by DXA in the beginning and at the end of the study period	The exercise group showed significantly greater gain of BMD compared with the controls: total body, 3.5% vs 1.2%; L2–L4, 4.8% vs 1.2%; proximal femur, 4.5% vs 1.3%; and femoral neck, 12.0% vs 1.7%.	High-impact, strength-building exercise was effective for bone mineral acquisition.
Snow-Harter et al., 1992 (227)	8-mo RCT	A group of recruited women students, randomly assigned to running, weight training, or control group	52 women, 31 complete the study	20	Running: HR 70–80%, HRmax, progressive, from 4 to 10 miles·wk^{-1}, 3×/wk Weight training: supervised, circuit training, 3×/wk, progressive intensity from 25–70% to 85% of the 1-RM at the time. Controls: maintenance of recreational activity	L2–L4, proximal femur by DXA	L2–L4 increased similarly and significantly in runners (1.3%) and weight trainers (1.2%) and significantly more than in controls. No significant changes in the femoral bone measurements.	Progressive running and weight training increased lumbar spine bone mineral in young women.
Witzke and Snow, 2000 (265)	9-mo NRCT	A group of postmenarcheal girls recruited specially for the exercise group from two schools, controls from the same schools matched for age and menarche	56	13–15	Variety of progressive exercises increasing strength of and causing moderate to high impacts (plyometric jump training) on especially lower extremities, 3×/wk, 30–45 min	Whole body, L2–L4, femoral neck, trochanter, (3.1% vs 1.9%)	Plyometric training may increase bone mass during adolescent growth.	

DXA, dual-energy x-ray absorptiometry; QCT, quantitative computed tomography; SPA, single photon absorptiometry; 1-RM, one-repetition maximum; HR, heart rate.

TABLE 14. Pertinent data of non-RCTs and RCTs studying the effect of PA on bone mass in pre- and postmenopausal women.

Type of Regimen	Reference	Subjects		Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
		N	Age (yr)				
Premenopausal (and perimenopausal) women							
Strength/resistance training regimens, NRCTs	Rockwell et al., 1990 (209)	T = 10 C = 7	36–40	9 mo, 8-station circuit, 2×/wk, 12 reps × 2 sets, 70% 1 RM	LS, FM by DXA	BMD decreased in LS in T, no change in C.	Differences between the groups at baseline.
	Vuori et al., 1994 (256)	T = 12 C = 12	21–22	12 mo, unilateral leg press, 4×/wk, 10 reps × 5 sets, 80% 1 RM	LS, FN, distal femur, patella, proximal tibia, calcaneus, ΣBMC by DXA	No significant training effects in BMD (except increase in patella) but positive trend.	
Strength/resistance training regimens, RCTs	Lohman et al., 1995 (151)	T = 22 C = 34	28–29	18 mo, 3×/wk, 8–12 reps × 3 sets, 12 exercises, 75–80% 1 RM	Total body, LS, FN, arm, leg by DXA	BMD increased in LS and FN as compared with C.	
Aerobic, endurance, or impact training regimens, RCTs (and RT without C)	Bassey et al., 1998 (8)	Tpre = 30 Cpre = 25 Tpost = 69 Cpost = 54	38 36 55 54	6 mo in premenopausal and 12 mo in postmenopausal women, 50 vertical jumps on 6 d·wk^{-1} of mean height 8.5 cm	LS, FN by DXA	BMD FN increased significantly (2.8%) in premenopausal T and significantly more than in premenopausal C. In postmenopausal T and C no difference.	Same regimen effective in pre- but not in postmenopausal women.
	Heinonen et al., 1998 (90)	Tend = 34 Tcalist = 36 C = 35	52–53	18 mo, endurance 2–3×/wk, 50 min, 55–75% $\dot{V}O_{2\max}$ Calisthenics 2–6×/wk, 50 min, strength-endurance exercises Controls: light stretching 1×/wk	LS, FN, calcaneus, distal radius by DXA, repeated 6 times	Significant training effect (maintenance) in FN of Tend, no training effects in LS or by calisthenics.	The movement in calisthenics training may have been too slow and pliant to result in effective loading stimulus.
	Heinonen et al., 1996 (89)	Timp = 49 C = 49	35–45	18 mo, 3×/wk, 60 min, jumping, calisthenics, progressive, in class Controls: maintenance of previous activities	LS, FN, trochanter, distal femur, patella, proximal tibia, calcaneus, dominant radius by DXA	Significantly greater increase of BMD of FN in Timp (1.6%) than in C (0.6%), no intergroup differences between the changes of BMD at the non–weight-bearing bone sites.	
	Humphries et al., 2000 (98)	Tweight = 35 Twalk = 29	45–65	24 wk, Twalk 2×/wk, 50 min, walking Tweight 2×/wk, 50 min, dynamic concentric and eccentric contractions Increased gradually to 90% 1 RM, 2–4 reps	LS by DXA	No significant group differences in LS BMD.	Training period only 24 wk.

T, training group; C, control group; DXA, dual-energy x-ray absorptiometry; RM, repetition maximum; HR, heart rate; SPA, single photon absorptiometry; DPA, dual photon absorptiometry; QCT, quantitative computed tomography.

TABLE 14. *Continued*

Type of Regimen	Reference	Subjects		Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
		N	**Age (yr)**				
	Salamone et al., 1999 (215)	T = 115 C = 126	47	18 mo, moderate-intensity aerobic activities and lowering dietary fat intake to lose weight	LS and FN by DXA	Annualized rate of FN BMD loss was twofold higher ($P < 0.015$) in the T (lifestyle intervention) (0.8%) than in the C (weight stable) (0.4%) group.	Large increases in PA attenuated LS BMD loss, no effect on FN BMD loss.
	Snow-Harter et al., 1992 (227)	TW = 12 Taer = 10 C = 8	20	8 mo TW: weight training 3×/wk, 3 sets, 8–12 reps, 85% 1 RM, 14 exercises; Taer: 3×/wk, running 70–80% HRmax, mileage increased	LS, proximal femur by DXA	BMD, LS increased significantly in TW ($1.8\%\cdot yr^{-1}$) and Taer ($2.0\%\cdot yr^{-1}$) compared with C ($-1.2\%\cdot yr^{-1}$).	
Postmenopausal women							
Strength/resistance training regimens, NRCTs	Beverly et al., 1989 (15)	T = 69 C = no, nontrained arm as control	62	1½ mo, 6×/wk, 30 sec, (mostly) unilateral squeeze tennis ball, 3 times consecutively, morning and night	Wrist BMC by SPA	Significant 3.4% BMC gain in trained wrist, nonsignificant 1.9% increase in nontrained wrist, no intergroup comparison reported.	
	Braith et al., 1996 (21)	T = 8 C = 8	56–62	6 mo, 2×/wk, 1 set 10–15 reps, 15 RM, 8 exercises	Total body, LS, FN by DXA	Significant increases in total body, LS and FN in T, no change in C.	Heart transplant patients.
	Menkes et al., 1993 (170)	T = 9 C = 7	50–70, males	16 wk, 3×/wk, 1–2 sets, 15 reps, 5 RM, 13 exercises	Total body, LS, FN by DXA	Increased FN (+6.5%) in T vs C (–1.3%).	
	Pruitt et al., 1992 (199)	T = 17 C = 10	54–56	9 mo, 3×/wk, 1 set, 1 rep, 10 RM, 11 exercises	Radius and ulna by SPA, LS and FN by DPA	Increased BMD in LS in T, decreased in C.	
	Peterson et al., 1991 (193)	Taer = 17 Taer + strength = 18 C = 19	36–37	1 yr; aerobic dancing (class): 3×/wk, 50 min Strength training (home): 3×/wk, 2 sets, 8–12 reps, 6 exercises	Radius, ulna and humerus by SPA, LS, proximal femur by DPA	Nonsignificant increase in humerus and proximal femur BMD in Taer + strength and Taer vs C	
	Simkin et al., 1987 (221)	T = 14 C = 26	53–74	5 mo, 3×/wk, 15 min, 4 forearm exercises	Distal radius by Compton scattering and by SPA	Increased BMD (3.8%) in radius in T vs decreased (–1.9%) in C (by Compton scattering).	
Strength/resistance training regimens, RCTs	Chow et al., 1987 (35)	Taer = 17 Taer + strength = 16 C = 15	50–62	1 yr; aerobic training: 3×/wk, 40 min, 80% HRmax Strength training 3×/wk, 10 reps, 10 RM, upper and lower body	Trunk, thighs calcium bone index by neutron activation analysis	Increased calcium bone index in Taer and Taer + strength vs C. No difference between Taer and Taer + strength.	
	Heikkinen et al., 1997 (96)	T = 13 C = 13	53	36 mo, 1×/wk exercises in fitness center + advice to exercise 2 $h\cdot wk^{-1}$	LS, FN, Ward's triangle, trochanter	Increased BMD in Ward's triangle and trochanter in T, decrease in C, intergroup differences significant.	The given results refer to those in exercising and nonexercising placebo group of a drug trial.

TABLE 14. *Continued*

Type of Regimen	Reference	Subjects		Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
		N	Age (yr)				
	Kerr et al., 1996 (116)	Tstr = 23 Tedn = 19	56 58	12 mo 3×/wk, 20–30 min, unilateral training of upper and lower limb, other side as control Strength = 3 × 8 RM; endurance: 3 × 20 RM	Radius (ultradistal, shaft), FN, Ward's triangle, trochanter, and intertrochanteric site by DXA	BMD increased significantly more in trained as compared with control site in the Tstr at Ward's triangle, trochanter, and intertrochanteric site.	Peak load was more important than the number of loading cycles in increasing bone mass. However, the volume of training was different in Tstr and Tend.
	Lord et al., 1996 (152)	T = 90 C = 89	72 69	10.5 mo, 2×/wk, strengthening, coordination, balance, and weight-bearing exercise	LS, FN, trochanter by DXA	No significant differences between groups in changes of BMD.	
	Lynch and Judge, 1992 (154)			15 mo resistance exercise and brisk walking	FN, trochanter by DXA	BMD at both sites decreased more in the exercising than in the control group.	
	Maddalozzo and Snow, 2000 (results only for women given here) (155)	Thigh = 12 Tmod = 13	53	24 wk, 3×/wk, 75 min, all muscle groups, supervised High intensity (final): 3 sets, 2–4 reps, 90+% 1 RM Moderate intensity: 3 sets, 10–13 reps, 40–60% 1 RM	Whole body, LS, FN, Ward's triangle, trochanter by DXA	No significant intergroup differences between changes of BMD.	In men training resulted in increased BMD at LS and trochanter.
	McCartney et al., 1995 (167)	T = 76 C = 66	60–80	42 wk, 2×/wk, 7 exercises, 3 sets, 10–12 reps, 50–80% 1 RM	Total body BMD and BMC, LS by DPA	No significant changes.	
	Nelson et al., 1994 (179)	T = 20 C = 19	50–70	1 yr, 2×/wk, 5 exercises, 3 sets, 8 reps, 80% 1 RM	Total body, LS, FN	BMD increased in LS (0.9%) and FN (1.0%) in T and decreased in LS (–2.5%) and FN (–1.8%) in C, difference of change significant.	
	Nichols et al., 1995 (181)	T = 17 C = 17	60–84	1 yr, 3×/wk, 8 exercises, 3 sets, 10–12 reps, 80% 1 RM	Total body, LS, FN by DXA	No significant changes of BMD.	
	Notelovitz et al., 1991 (184)	T+estr = 19 C+estr = 11	43 46	1 yr, 3×/wk, 6 exercises, 1 set, 8 reps, 8 RM	Radius by SPA, total body, spine by DPA	BMD of LS and radial midshaft increased significantly and significantly more in T+estr (+8.3% and +4.1%, respectively) than in C+estr (+1.5% and –0.3%, respectively).	
	Pruitt et al., 1995 (200)	Thigh = 8 Tlow = 7 C = 11	65–79	1 yr; high-intensity training 80% RM, low-intensity training 40% RM, both 3 sets, 14 reps, 10 exercises	LS, FN by DXA	Similar but nonsignificant pattern for the loss of LS BMD.	

TABLE 14. *Continued*

Type of Regimen	Reference	Subjects		Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
		N	Age (yr)				
	Revel et al., 1993 (203)	T = 23 C = 26	51–71	1 yr, 2–3×/d, psoas training 60 reps/hip, 5 kg weight	Lumbar trabecular BMD by QCT	Nonsignificant decrease in BMD of C vs T.	
	Rhodes et al., 2000 (204)	T = 22 C = 22	65–75	1 yr, 3×/wk, 1 h, supervised circuit training, 3 sets, 8 reps, 75% 1 RM	LS, FN, Ward's triangle, trochanter by DXA	Nonsignificant increase of BMD in T and nonsignificant decrease in C.	
	Sinaki et al., 1989 (222)	T = 34 C = 31	49–65	24 mo, once a day, 5×/wk, weight-bearing exercises for the back	LS by DPA	BMD of LS decreased by 1.4% in T and 1.2% in C.	
	Smidt et al., 1992 (224)	T = 22 C = 27	57 55	10 mo, 3–4×/wk, 3 resistance exercises for trunk muscles, 3 sets, 10 reps, 70% of maximum strength	LS, FN, Ward's triangle, trochanter by DPA	No significant intergroup differences in changes of BMD but trend in favor of T except in Ward's triangle.	
Aerobic training regimens, NRCTs	Aloia et al., 1978 (2)	T = 9 C = 9	53 52	12 mo, 3×/wk, 60 min, conditioning exercises	Distal radius by SPA Total body Ca by neutron activation analysis	No difference in change of radius BMC between groups; total body Ca increased in T, decreased in C, difference significant	
	Bloomfield et al., 1993 (18)	T = 7 C = 7	62 59	8 mo, 3×/wk, 50 min, non–weight-bearing aerobic exercise at 60–80% HRmax, calisthenics	LS, femur by DPA	LS BMD increased by 5.3%·yr^{-1} in T and decreased by 3.7%·yr^{-1} in C, difference significant. In femur, no significant difference between changes in T and C.	
	Caplan et al., 1993 (27)	T = 19 C = 11	66 65	24 mo, 2×/wk, 60 min, weight-bearing aerobic exercise, advice to exercise ≥1×/wk extra	LS, FN by DPA	BMD of LS decreased by 0.4%·yr^{-1} in T and 1.6%·yr^{-1} in C. No significant differences of BMD changes of FN between T and C.	
	Cavanaugh and Cann, 1988 (30)	T = 8 C = 9	55 57	1 yr, 3×/wk, 15–40 min walking, 60–85% $\dot{V}O_{2max}$, progressive	LS by QCT	BMD of LS decreased in both groups at similar rate, –5.6% in T, –4.0% in C.	
	Dalsky et al., 1988 (43)	17 18	62 63	9 mo, 3×/wk, 50–60 min, walking and jogging, stairclimbing 70–90% $\dot{V}O_{2max}$	LS by DPA	BMC of LS increased by 5.2% in T, decreased by 1.4% in C, difference significant.	
	Hatori et al., 1993 (85)	Tmod = 9 Tint = 12 C = 12	58 56 58	7 mo, 3×/wk, 30 min, walking: Tmod = below anaerobic threshold, Tint: above anaerobic threshold	LS by DXA	BMD of LS increased significantly by 2.7% in Tint, nonsignificantly by 0.7% in Tmod, and C.	
	Iwamoto et al., 1998 (100)	T = 15 C = 20	65 65	12 mo, increased walking by ~45%, gymnastics 5×/wk	LS by DXA	BMD of LS increased significantly by 4.5% in T, insignificantly by 1% in C.	The subjects were osteoporotic.

TABLE 14. *Continued*

Type of Regimen	Reference	Subjects *N*	Subjects Age (yr)	Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
	Kohrt et al., 1995 (122)	T = 8 C = 8	65 66	11 mo, first 2 mo flexibility exercises, then 9 mo walking, jogging, stair climbing, 3–5×/wk, 45 min, 65–85% HRmax	LS and FN, Ward's triangle, trochanter by DXA	BMD of LS and FN increased significantly by 2.7%·yr^{-1} and 3.6%·yr^{-1}, respectively, in T, no significant changes in C. Significant increase of BMD also at Ward's triangle and trochanter in T, not in C.	
	Kohrt et al., 1995 (122)	THRT = 8 CHRT = 8	66 67	Same training program as above	See above	BMD of LS increased by 8.9%·yr^{-1} in THRT by 6.6%·yr^{-1} in CHRT, and BMD of FN increased by 2.3%·yr^{-1} in THRT and in CHRT.	
	Krölner et al., 1983 (127)	T = 16 c = 15	61 61	8 mo, 2×/wk, 1 h, supervised, variety of weight-bearing aerobic and calisthenic exercises	LS, radius by DPA	BMC increased by 3.5% in T, decreased by 2.7% in C, difference significant.	
	Nelson et al., 1991 (178)	Tmod. Ca = 9 Cmod Ca = 9	60	12 mo, 4×/wk, 50 min, walking, 70–80% HRmax, mean Ca intake 761 mg·d^{-1}	LS by QCT, LS, FN by DPA	No difference in LS or FN BMD changes between Tmod Ca and Cmod Ca groups by DXA but significant increase of LS BMD by QCT in Tmod Ca.	
	Nelson et al., 1991 (178)	Thigh Ca = 9 Chigh Ca = 9	60	As above, but mean Ca intake 1462 mg·d^{-1}	As above	BMD of LS increased by 2.4%·yr^{-1} in Thigh Ca and by 0.5%·yr^{-1} in Chigh Ca; corresponding changes in FN BMD 3.0%·yr^{-1} and 0.9%·yr^{-1}, respectively.	
	Tsukahara et al., 1994 (241)	T = 30 C = 15	64	12 mo, 1×/wk, 30 min walking, jumping aerobics in water, 65% $\dot{V}O_{2max}$	LS by DXA	BMD of LS increased by 1.3% in T and decreased by 2.6% in C, changes nonsignificant.	
Aerobic training regimens, RCTs	Bassey and Ramsdale, 1995 (7)	T = 20 C = 24	64 55	12 mo, 1×/wk high-impact exercise + 50 heel drops·d^{-1}; in C 1×/wk low-impact exercise + flexibility exercises	LS and FN, Ward's triangle, trochanter, radius by DXA	At 6 mo, significant increase in trochanteric BMD in T, no change in C. No other significant changes.	
	Bassey et al., 1998 (8)	Tpre = 30 Cpre = 25 Tpostdepl = 45 TpostHRT = 24 Cpostdepl = 32 CpostHRT = 22	38 36 56 54 55 53	20 wk 1×/wk supervised 50 jumps during 10 min, 6×/wk the same at home, ground reaction force 3 times body weight in younger and 4 times body weight in older subjects	LS and FN and trochanter by DXA	In the premenopausal women exercise resulted in significant 2.8% increase of femoral BMD, different from C. In postmenopausal women, no significant difference between exercise and control groups after 12 or 18 mo. HRT status did not affect this outcome.	
	Bravo et al., 1996 (22)	T = 61	60	12 mo, 3×/wk, 1 h weight-bearing activities, 60–70%	LS, FN by DXA	No significant change of BMD of LS or FN in T, significant decrease of BMD at LS in C.	

TABLE 14. *Continued*

Type of Regimen	Reference	Subjects		Exercise Regimen(s)	Bone Measurements Site and Method	Results	Comments
		N	Age (yr)				
		C = 63	60	HRR + flexibility exercises			
	Ebrahim et al., 1997 (55)	Tcompl = 49 Tdrop = 32 Ccompl = 48 Cdrop = 36	66 67 68 71	24 mo, 3×/wk, 40 min brisk walking, unsupervised Compl = compliant subjects; drop = drop outs from follow-up	LS, FN by DXA	In Ccompl BMD of FN had fallen more than in Tcompl, difference 2.4%. BMD of LS had increased to a similar extent in both Tcompl and Ccompl.	
	Grove and Londree, 1992 (75)	Tlow = 5 Thigh = 5 C = 5	57 54 56	12 mo low- or high-impact activities 3×/wk, 20 min; low impacts $\leq$1.5 × body weight, high impacts $\geq$1.5 × body weight	LS by DPA	No change of LS BMD in C, 1.0% increase in Tlow and 6.9% increase in Thigh.	
	Hatori et al., 1993 (85)	Tmod = 9 Thigh = 12	58 56	7 mo; moderate intensity exercise: 3×/wk, 30 min walking at 90% of the HR at anaerobic threshold (<60% of $\dot{V}O_{2max}$); high intensity: the same at 110% of the HR at anaerobic threshold	LS by DXA	BMD LS increased by 1.2% in Thigh, decreased by 1.2% in Tlow and C.	
	Lau et al., 1992 (139)	Tcalow = 11 Ccalow = 12 Tcahigh = 15 Ccahigh = 12	79 75 76 75	10 mo, 100 × stepping up and down a 22.5-cm block, 4×/wk, upper trunk exercises	LS, FN, Ward's triangle, trochanter by DXA	Exercise had no effect on bone loss at any site, but there was significant joint effect of Ca supplementation and exercise at FN.	
	Martin and Notelovitz, 1993 (165)	T30 = 20 T45 = 16 C = 19	60 58 57	12 mo treadmill running, 3×/wk, 70–85% HRmax 30 = 30 min/session; 45 = 45 min/session	LS, proximal and distal forearm by DPA	No significant changes of BMD at any site.	
	McMurdo et al., 1997 (169)	T+Ca = 44 C+Ca = 48	60–73	6 × 10 wk during 2 years, 3×/wk, 45-min classes, weight-bearing exercise	LS by QCT	LS BMC decreased by 0.9% in T+Ca and by 2.7% in C+Ca, difference not significant.	
	Prince et al., 1995 (198)	T+Ca = 42 C+Ca = 42	63 62	24 mo, 2×/wk, 1 h, 60% HRmax, supervised weight-bearing exercise, walking 2 extra h·wk^{-1}	LS, FN, trochanter, intertrochanter by DXA	T+Ca had slightly but significantly less bone loss and FN as compared with C+Ca.	

of intensive exercise training at various ages should be studied.

The potential of PA as an osteogenic stimulus can probably be best used by combining it with the effects of hormones and drugs. The effects of various combinations of PA and drugs, hormones, and eventually other measures should be studied in order to find optimal regimens to influence bone mass and strength.

When OP is seen as a clinical condition leading frequently to fractures, the scope of the studies on the effects of PA on OP and on related dose-response issues should be enlarged to include all possible effects of PA on the risk of osteoporotic fractures.

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REFERENCES

1. Alfredson, H., P. Nordström, and R. Lorentzon. Bone mass in female volleyball players: a comparison of total and regional bone mass in female volleyball players and nonactive females. *Calcif. Tissue Int.* 60:338–342, 1997.
2. Aloia, J. F., S. H. Cohn, J. A. Ostuni, R. Cane, and K. Ellis. Prevention of involutional bone loss by exercise. *Ann. Intern. Med.* 89:356–358, 1978.
3. Avlonitou, E., E. Georgiu, G. Douskas, and A. Louizi. Estimation of body composition in competitive swimmers by means of three different techniques. *Int. J. Sports Med.* 18:363–368, 1997.
4. Bailey, D. A., H. A. McKay, R. L. Mirwald, P. R. E. Crocker, and R. A. Faulkner. A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the University of Saskatchewan bone mineral accrual study. *J. Bone Miner. Res.* 14:1672–1679, 1999.
5. Barnekow-Bergkvist, M., G. E. Hedberg, U. Janlert, and E. Jansson. Determinants of self-reported neck shoulder and low back symptoms in a general population. *Spine* 123:235–243, 1998.
6. Bass, S., G. Pearce, M. Bradney, et al. Exercise before puberty may confer residual benefits in bone density in adulthood: studies in active prepubertal and retired female gymnasts. *J. Bone Miner. Res.* 13:500–507, 1998.
7. Bassey, E. J., and S. J. Ramsdale. Weight-bearing exercise and ground reaction forces: a 12-month randomized controlled trial of effects on BMD in healthy postmenopausal women. *Bone* 16:469–476, 1995.
8. Bassey, E. J., M. C. Rothwell, J. J. Littlewood, and D. W. Pye. Pre- and postmenopausal women have different BMD responses to the same high-impact exercise. *J. Bone Miner. Res.* 13:1805–1813, 1998.
9. Battie, M. C., T. Videman, L. E. Gibbons, L. D. Fisher, H. Manninen, and K. Gill. Determinants of lumbar disc degeneration. A study relating lifetime exposures and magnetic resonance imaging (MRI) findings in identical twins. *Spine* 20:2601–2612, 1995.
10. Bautch, J. C., D. G. Malone, and A. C. Vailas. Effects of exercise on knee joints with osteoarthritis: a pilot study of biologic markers. *Arthritis Care Res.* 10:48–55, 1997.
11. Bennell, K. L., S. A. Malcolm, J. D. Wark, and P. D. Brukner. Skeletal effects of menstrual disturbances in athletes. *Scand. J. Med. Sci. Sports* 7:261–273, 1997.
12. Bennell, K. L., S. A. Malcolm, K. M. Kahn, et al. Bone mass and bone turnover in power athletes, endurance athletes, and controls: a 12-month longitudinal study. *Bone* 20:477–484, 1997.
13. Bentsen, H., F. Lindgarde, and R. Manthorpe. The effect of dynamic strength back exercise and/or a home training program in 57-year old women with chronic low back pain: results of a prospective randomized study with a 3-year follow-up period. *Spine* 22:1494–1500, 1997.
14. Bérard, A., G. Bravo, and P. Gauthier. Meta-analysis of the effectiveness of physical activity for the prevention of bone loss in postmenopausal women. *Osteoporos. Int.* 7:331–337, 1997.
15. Beverly, M. D., T. A. Rider, M. J. Evans, and R. Smith. Local bone mineral response to brief exercise that stresses the skeleton. *BMJ* 299:233–235, 1989.
16. Biering-Sørensen, F. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine* 9:106–119, 1984.
17. Blimikie, C. J. R., S. Rice, C. E. Wegger, J. Martin, D. Levy, and C. L. Gordon. Effects of resistance training on bone mineral content (BMC) and density in adolescent females. *Can. J. Physiol. Pharmacol.* 74:1025–1033, 1996.
18. Bloomfield S. S., N. I. Williams, D. R. Lamb, R. D. Jackson. Non-weightbearing exercise may increase lumbar spine BMD in healthy postmenopausal women. *Am. J. Phys. Med. Rehabil.* 72:204–209, 1993.
19. Börjesson, M., E. Robertson, L. Weidenhielm, E. Mattson, and E. Olsson. Physiotherapy in knee osteoarthrosis: effect on pain and walking. *Physiother. Res. Intern.* 1:89–97, 1996.
20. Bradney, M., G. Pearce, G. Naughton, et al. Moderate exercise during growth in prepubertal boys: changes in bone mass, size, volumetric density and bone strength: a controlled prospective study. *J. Bone Miner. Res.* 13:1814–1281, 1998.
21. Braith, R. W., R. M. Mills, M. A. Welsch, J. W. Keller, and M. L. Pollock. Resistance exercise training restores BMD in heart transplant recipients. *J. Am. Coll. Cardiol.* 28:1471–1477, 1996.
22. Bravo, G., P. Gauthier, P. M. Roy, et al. Impact of a 12-month exercise program on the physical and psychological health of osteopenic women. *J. Am. Geriatr. Soc.* 44:756–762, 1996.
23. Bronfort, G., C. H. Goldsmith, C. Nelson, P. D. Boline, and A. V. Anderson. Trunk exercise combined with spinal manipulative or NSAID therapy for chronic low back pain: a randomized, observer-blinded clinical trial. *J. Manipulative Physiol. Ther.* 19:570–582, 1996.
24. Buckwalter, J. A., and N. E. Lane. Athletics and osteoarthritis. *Am. J. Sports Med.* 25:873–881, 1997.
25. Buswell, J. Low back pain: a comparison of two treatment programmes. *N. Z. J. Physiother.* 10:13–17, 1982.
26. Campelleo, M., M. Nodin, and S. Weiser. Physical exercise and low back pain. *Scand. J. Med. Sci. Sports* 6:63–72, 1996.
27. Caplan, G. A., J. A. Ward, and S. R. Lord. The benefits of exercise in postmenopausal women. *Aust. J. Public Health* 17: 23–26, 1993.
28. Casazza, B. A., J. L. Young, and S. A. Herring. The role of exercise in the prevention and management of acute low back pain. *Occup. Med.* 13:47–59, 1998.
29. Cassell, C., M. Benedict, and B. Specker. BMD in elite 7- to 9-yr-old female gymnasts and swimmers. *Med. Sci. Sports Exerc.* 28:1243–1246, 1996.
30. Cavanaugh, D. J., and D. E. Cann. Brisk walking does not stop bone loss in postmenopausal women. *Bone* 9:201–204, 1988.
31. Cheng, Y., C. A. Macera, D. R. Davis, B. E. Ainsworth, P. J. Troped, and S. N. Blair. Physical activity and self-reported, physician-diagnosed osteoarthritis: is physical activity a risk factor? *J. Clin. Epidemiol.* 53:315–322, 2000.
32. Cherkin, D. C., R. A. Deyo, M. Battie, J. Street, and W. A. Barlow. A comparison of physical therapy, chiropractic manipulation, and provision of an educational booklet for the treatment of patients with low back pain. *N. Engl. J. Med.* 339:1021–1029, 1998.
33. Chilibeck, P. D., D. G. Sale, and C. E. Webber. Exercise and BMD. *Sports Med.* 19:103–122, 1995.
34. Chok, B., R. Lee, J. Latimer, and S. B. Tan. Endurance training of the trunk extensor muscles in people with subacute low back pain. *Phys. Ther.* 79:1032–1042, 1999.

35. Chow, R., J. E. Harrison, and C. Notarius. Effect of two randomized exercise programmes on bone mass of healthy postmenopausal women. *BMJ* 295:1441–1444, 1987.
36. Cohen, B., P. J. Millett, B. Mist, M. A. Laskey, and N. Rushton. Effect of exercise training programme on BMD in novice college rowers. *Br. J. Sports Med.* 29:85–88, 1995.
37. Cooper, C., M. Cawley, A. Bhalla, et al. Childhood growth, physical activity, and peak bone mass in women. *J. Bone Miner. Res.* 10:940–947, 1995.
38. Courteix, D., E. Lespessailles, S. L. Peres, P. Obert, P. Germain, and C. L. Benhamou. Effect of physical training on BMD in prepubertal girls: a comparative study between impact-loading and non-impact-loading sports. *Osteoporos. Int.* 8:152–158, 1998.
39. Courteix, D., E. Lespessailles, C. Jaffre, P. Obert, and C. L. Benhamou. Bone mineral acquisition and somatic development in highly trained girl gymnasts. *Acta Paediatr.* 88:803–803, 1999.
40. Coxhead, C. E., H. Inskip, T. W. Meade, W. R. S. North, and J. D. G. Troup. Multicentre trial of physiotherapy in the management of sciatic symptoms. *Lancet* 1:1065–1068, 1981.
41. Croft, P. R., A. C. Papageorgiuo, E. Thomas, G. J. Macfarlane, and A. J. Silman. Short-term physical risk factors for new episodes of low back pain. *Spine* 24:1556–1561, 1999.
42. Cummings, S. R., D. M. Black, M. C. Nevitt, et al. Bone density at various sites for prediction of hip fractures. *Lancet* 341:72–75, 1993.
43. Dalsky, F. P., K. S. Stocke, A. A. Ehsani, E. Slatopolsky, W. C. Lee, and S. J. Birge, Jr. Weight-bearing exercise training and lumbar BMC in postmenopausal women. *Ann. Intern. Med.* 108:824–828, 1988.
44. Davee, A. M., C. J. Rosen, and R. A. Adler. Exercise patterns and trabecular bone density in college women. *J. Bone Miner. Res.* 5:245–250, 1990.
45. Davies, J. R., T. Gibson, and L. Tester. The value of exercises in the treatment of low back pain. *Rheumatol. Rehabil.* 18:243–247, 1979.
46. De Jong, N., M. J. Paw, L. C. De Groot, G. J. Hiddink, and W. A. Van Staveren. Dietary supplements and physical exercise affecting bone and body composition in frail elderly persons. *Am. J. Public Health* 90:947–954, 2000.
47. Delitto, A., M. T. Cibulka, R. E. Erhard, R. W. Bowling, and J. A. Tenhula. Evidence for use of an extension-mobilization category in acute low back syndrome: a prescriptive validation pilot study. *Phys. Ther.* 73:216–228, 1993.
48. Deyle, G. D., N. E. Henderson, R. L. Matekel, M. G. Ryder, M. B. Garber, and S. C. Allison. Effectiveness of manual physical therapy and exercise in osteoarthritis of the knee. A randomized, controlled trial. *Ann. Intern. Med.* 132:173–181, 2000.
49. Deyo, R. A., N. E. Walsh, D. C. Martin, L. S. Schoenfeld, and S. Ramamurthy. A controlled trial of transcutaneous electrical nerve stimulation (TENS) and exercise for chronic low back pain. *N. Engl. J. Med.* 322:1627–1634, 1990.
50. Donchin, M., O. Woolf, L. Kaplan, and T. Forman. Secondary prevention of low-back pain: a clinical trial. *Spine* 15:1317–1320, 1990.
51. Dook, J. E., C. James, N. K. Henderson, and R. I. Price. Exercise and BMD in mature female athletes. *Med. Sci. Sports Exerc.* 29:291–296, 1997.
52. Drinkwater, B. L. Physical activity, fitness, and osteoporosis. In: *Physical Activity, Fitness, and Health. International Proceedings and Consensus Statement.* C. Bouchard, R. J. Shephard, and T. Stephens (Eds.). Champaign, IL: Human Kinetics, 1994, pp. 724–736.
53. Düppe, H., P. Gärdsell, O. Johnell, and E. Ornstein. BMD in female junior, senior and former football players. *Osteoporos. Int.* 6:437–441, 1996.
54. Düppe, H., P. Gärdsell, O. Johnell, B. E. Nilsson, and K. Ringsberg. BMD, muscle strength and physical activity. *Acta Orthop. Scand.* 68:97–103, 1997.
55. Ebrahim, S., P. W. Thompson, V. Baskaran, and K. Evans. Randomized placebo-controlled trial of brisk walking in the prevention of postmenopausal osteoporosis. *Age Ageing* 26:253–260, 1997.
56. Elnaggar, I. M., M. Nordin, A. Sheikhzadeh, M. Parnianpour, and N. Kahanovitz. Effects of spinal flexion and extension exercises on low-back pain and spinal mobility in chronic mechanical low-back pain patients. *Spine* 16:967–972, 1991.
57. Emslander, H. C., M. Sinaki, J. M. Muhs, et al. Bone mass and muscle strength in female college athletes (runners and swimmers). *Mayo Clin. Proc.* 73:1151–1160, 1998.
58. Ernst, E. Exercise for female osteoporosis. A systematic review of randomised clinical trials. *Sports Med.* 25:359–368, 1998.
59. Ettinger, W. H., R. Burns, S. P. Messier, et al. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis. The fitness arthritis and seniors trial (FAST). *JAMA* 277:25–31, 1997.
60. Faas, A., A. W. Chavannes, J. T. M. Van Eijk, and J. W. Gubbels. A randomised, placebo-controlled trial of exercise therapy in patients with acute low back pain. *Spine* 18:1388–1395, 1993.
61. Faas, A. Exercises. Which ones are worth trying, for which patients, and when? *Spine* 21:2874–2879, 1996.
62. Farrell, J. P., and L. T. Twomey. Acute low back pain: comparison of two conservative treatment approaches. *Med. J. Aust.* 1:160–164, 1982.
63. Fehling, P. C., L. Alekel, J. Clasey, and A. Rector. A comparison of bone mineral densities among female athletes in impact loading and active loading sports. *Bone* 17:205–210, 1995.
64. Felson, D. T., Y. Zhang, M. T. Hannan, et al. Risk factors for incident radiographic knee osteoarthritis in the elderly. *Arthritis Rheum.* 40:728–733, 1997.
65. Flodgren, G., R. Hedelin, and K. Henriksson-Larsén. BMD in flatwater sprint kayakers. *Calcif. Tissue Int.* 64:374–379, 1999.
66. Friedlander, A. L., H. K. Genant, S. Sadowsky, N. N. Byl, and C.-C. Glüer. A two-year program of aerobics and weight training enhances BMD of young women. *J. Bone Miner. Res.* 10:574–585, 1995.
67. Frost, H. M. Bone "mass" and the "mechanostat": a proposal. *Anat. Rec.* 219:1–9, 1987.
68. Frost, H., J. A. Klaber Moffett, J. S. Moser, and J. C. T. Fairbank. Randomised controlled trial for evaluation of fitness programme for patients with chronic low back pain. *BMJ* 310: 151–154, 1995.
69. Frost, H., S. E. Lamb, J. A. Klaber Moffett, J. C. T. Fairbank, and J. S. Moser. A fitness programme for patients with chronic low back pain: 2-year follow-up of a randomised controlled trial. *Pain* 75:273–279, 1998.
70. Gelber, A. D., M. C. Hochberg, L. A. Mead, N. Y. Wang, F. M. Wigly, and M. J. Klag. Joint injury in young adults and risk for subsequent knee and hip osteoarthritis. *Ann. Intern. Med.* 133: 321–328, 2000.
71. Gibson, J. H., M. Harries, A. Mitchell, R. Godfrey, M. Lunt, and J. Reeve. Determinants of bone density and prevalence of osteopenia among female runners in their second to seventh decades of age. *Bone* 26:591–598, 2000.
72. Gilbert, J. R., D. W. Taylor, A. Hildebrand, and C. Evans. Clinical practice of common treatments for low-back pain. *BMJ* 291:789–792, 1985.
73. Gleeson, P. B., E. J. Protas, A. D. Leblanc, V. S. Schneider, and H. J. Evans. Effects of weight lifting on BMD in premenopausal women. *J. Bone Miner. Res.* 5:153–158, 1990.
74. Gross, P., and B. Marti. Sports activity and risk of arthritis [in German]. *Sweiz. Med. Wochenschr.* 127:967–977, 1997.
75. Grove, K. A., and B. R. Londree. Bone density in postmenopausal women: high impacts vs low impact exercise. *Med. Sci. Sports Exerc.* 24:1190–1194, 1992.
76. Gundewall, B., M. Liljeqvist, and T. Hansson. Primary prevention of back symptoms and absence from work: a prospective randomized study among hospital employees. *Spine* 18:587–594, 1993.
77. Gutin, B., S. Owens, T. Okuyama, S. Riggs, M. Fergusin, and M. Litaker. Effect of physical training and its cessation on percent

fat and bone density of children with obesity. *Obes. Res.* 7:208–214, 1999.

78. GYNTELBERG, F. One-year incidence of low back pain among male residents of Copenhagen aged 40–59. *Dan. Med. Bull.* 21:30–36, 1974.
79. HAAPASALO, H., P. KANNUS, H. SIEVANEN, A. HEINONEN, P. OJA, and I. VUORI. Long-term unilateral loading and BMD and content in female squash players. *Calcif. Tissue Int.* 54:249–255, 1994.
80. HÄKKINEN, A., T. SOKKA, A. KOTANIEMI, et al. Dynamic strength training in patients with early rheumatoid arthritis increases muscle strength but not BMD. *J. Rheumatol.* 26:1257–1263, 1999.
81. HANNAN, M. T., D. T. FELSON, J. J. ANDERSON, and A. NAIMARK. Habitual physical activity is not associated with knee osteoarthritis: the Framingham Study. *J. Rheumatol.* 20:704–709, 1993.
82. HANSEN, F. R., T. BENDIX, P. SKOV, C. V. JENSEN, J. H. KRISTENSEN, L. KROHN, and H. SCHIOELER. Intensive, dynamic back-muscle exercises, conventional physiotherapy, or placebo-control treatment of low-back pain. A randomized, observer-blind trial. *Spine* 18:98–108, 1993.
83. HARBER, V. I., L. E. WEBBER, J. R. SUTTON, and J. D. MACDOUGALL. The effect of amenorrhea on calcaneal bone density and total bone turnover in runners. *Int. J. Sports Med.* 12:505–508, 1991.
84. HARTIVGSEN, J., C. LEBOEUF-YDE, S. LINGS, and E. H. CORDER. Is sitting-while-at-work associated with low back pain? A systematic, critical literature review. *Scand. J. Public Health* 28:230–239, 2000.
85. HATORI, M., A. HASEGAWA, H. ADACHI, et al. The effects of walking at the anaerobic threshold level on vertebral bone loss in postmenopausal women. *Calcif. Tissue Int.* 52:411–414, 1993.
86. HEIKKINEN, J., E. KYLLONEN, E. KURTILLA-MATERO, et al. HRT and exercise: effects on bone density, muscle strength and lipid metabolism. A placebo-controlled 2-year prospective trial on two estrogen-progestin regimens in healthy postmenopausal women. *Maturitas* 26:139–149, 1997.
87. HEINONEN, A., P. OJA, P. KANNUS, H. SIEVANEN, A. MÄNTTARI, and I. VUORI. BMD in female athletes of different sports. *Bone Miner.* 23:1–14, 1993.
88. HEINONEN, A., P. OJA, P. KANNUS, et al. BMD in female athletes representing sports with different loading characteristics of the skeleton. *Bone* 17:197–203, 1995.
89. HEINONEN, A., P. KANNUS, H. SIEVANEN, et al. Randomised controlled trial of effect of high-impact exercise on selected risk factors for osteoporotic fractures. *Lancet* 348:1343–1347, 1996.
90. HEINONEN, A., P. OJA, H. SIEVANEN, M. PASANEN, and I. VUORI. Effect of 2 training regimens on BMD in healthy perimenopausal women: a randomized controlled trial. *J. Bone Miner. Res.* 13:483–490, 1998.
91. HEINONEN, A., P. KANNUS, H. SIEVÄNEN, M. PASANEN, P. OJA, and I. VUORI. Good maintenance of high-impact activity-induced bone gain by voluntary, unsupervised exercises: an 8-month follow-up of a randomized controlled trial. *J. Bone Miner. Res.* 14:125–128, 1999.
92. HEINRICH, C. H., S. B. GOING, R. W. PAMENTER, C. D. PERRY, T. W. BOYDEN, and T. G. LOHMAN. BMC of cyclically menstruating female resistance and endurance trained athletes. *Med. Sci. Sports Exerc.* 22:558–563, 1990.
93. HELEWA, A., C. H. GOLDSMITH, P. LEE, H. A. SMYTHE, and L. FORWELL. Does strengthening the abdominal muscles prevent low back pain—a randomized controlled trial. *J. Rheumatol.* 26:1808–1815, 1999.
94. HELMINEN, H. J., I. KIVIRANTA, A.-M. SÄÄMÄNEN, M. TAMMI, K. PAUKKONEN, and J. JURVELIN (Eds.). *Joint Loading—Biology and Health of Articular Structures.* Bristol: Wright, 1987, p. 440.
95. HEMMILA, H. M., S. M. KEINANEN-KIUKAANNIEMI, S. LEVOSKA, and P. PUSKA. Does folk medicine work? A randomized clinical trial on patients with prolonged back pain. *Arch. Phys. Med. Rehabil.* 78:571–577, 1997.
96. HILDE, G., and K. BØ. Effect of exercise in the treatment of chronic low back pain: a systematic review, emphasising type and dose of exercise. *Phys. Ther. Rev.* 3:107–117, 1998.
97. HO, S. C., E. WONG, S. G. CHAN, J. LAU, C. CHAN, and P. C. LEUNG. Determinants of peak bone mass in Chinese women aged 21–40 years. III. Physical activity and BMD. *J. Bone Miner. Res.* 12:1262–1271, 1997.
98. HUMPHRIES, B., R. U. NEWTON, R. BRONKS, et al. Effect of exercise intensity on bone density, strength, and calcium turnover in older women. *Med. Sci. Sports Exerc.* 32:1043–1050, 2000.
99. IMEOKPARIA, R. L., J. P. BARRETT, M. I. ARRIETA, et al. Physical activity as a risk factor for osteoarthritis of the knee. *Ann. Epidemiol.* 4:221–230, 1994.
100. IWAMAOTO, J., T. TAKEDA, T. OTANI, and Y. YABE. Effect of increased physical activity on BMD in postmenopausal osteoporotic women. *Keio J. Med.* 47:157–161, 1998.
101. JACOBSON, P. C., W. BEAVER, S. A. GRUBBS, T. N. TAFT, and R. V. TALMAGE. Bone density in women: college athletes and older athletic women. *J. Orthop. Res.* 2:328–332, 1984.
102. JOHANSEN, R. L. REMVIK, P. KRYGER, et al. Exercises for chronic low back pain: a clinical trial. *J. Orthop. Sports Phys. Ther.* 22:52–59, 1995.
103. JONES, G., and T. DWYER. Bone mass in prepubertal children: gender differences and the role of physical activity and sunlight exposure. *J. Clin. Endocrinol. Metab.* 83:4274–4279, 1998.
104. KANDERS, B., D. W. DEMSTER, and R. LINDSAY. Interaction of calcium nutrition and physical activity on bone mass in young women. *J. Bone Miner. Res.* 3:145–149, 1988.
105. KANKAANPAA, M., S. TAIMELA, O. AIRAKSINEN, and O. HÄNNINEN. The efficacy of active rehabilitation in chronic low back pain. Effect on pain intensity, self-experienced disability, and lumbar fatigability. *Spine* 24:1034–1042, 1999.
106. KANNUS, P., H. HAAPASALO, M. SANKELO, et al. Effects of starting age of physical activity on bone mass in dominant arm of tennis and squash players. *Ann. Intern. Med.* 123:27–31, 1995.
107. KARLSSON, M. K., O. JOHNELL, and K. J. OBRANT. Is BMD advantage maintained long-term in previous weight lifters? *Calcif. Tissue Int.* 57:325–328, 1995.
108. KARLSSON, M. K., R. HASSERIUS, and K. J. OBRANT. BMD in athletes during and after career: a comparison between loaded and unloaded skeletal regions. *Calcif. Tissue Int.* 59:245–248, 1996.
109. KARLSSON, M. K., C. LINDEN, C. KARLSSON, O. JOHNELL, K. OBRANT, and E. SEEMAN. Exercise during growth and BMD and fractures in old age. *Lancet* 355:469–470, 2000.
110. KELLET, K. M., D. A. KELLETT, and L. A. NORDHOLM. Effects of an exercise program on sick leave due to pack pain. *Phys. Ther.* 71:283–293, 1991.
111. KELLEY, G. Aerobic exercise and lumbar spine BMD in postmenopausal women: a meta-analysis. *J. Am. Geriatr. Soc.* 46:143–152, 1998.
112. KELLEY, G. A. Aerobic exercise and bone density at the hip in postmenopausal women: a meta-analysis. *Prev. Med.* 27:798–807, 1998.
113. KELLEY, G. A. Exercise and regional BMD in postmenopausal women. A meta-analytic review of randomized trials. *Am. J. Phys. Med. Rehabil.* 77:76–87, 1998.
114. KEMPER, H. C. G., J. W. R. TWISK, W. VAN MECHELEN, G. P. POST, J. C. ROOS, and P. LIPS. A fifteen-year longitudinal study in young adults on the relation of physical activity and fitness with the development of the bone mass: the Amsterdam growth and health longitudinal study. *Bone* 27:847–853, 2000.
115. KENDALL, P. H., and J. M. JENKINS. Exercises for backache: a double-blind controlled trial. *Physiotherapy* 54:154–157, 1968.
116. KERR, D., A. MORTON, I. DICK, and R. PRINCE. Exercise effects on bone mass in postmenopausal women are site-specific and load-dependent. *J. Bone Miner. Res.* 11:218–225, 1996.
117. KIRCHNER, E. M., R. D. LEWIS, and P. J. O'CONNOR. BMD and dietary intake of female college gymnasts. *Med. Sci. Sports Exerc.* 27:543–549, 1995.
118. KIRCHNER, E. M., R. D. LEWIS, and P. J. O'CONNOR. Effect of past gymnastics participation on adult bone mass. *J. Appl. Physiol.* 80:226–232, 1996.
119. KIRK, S., C. F. SHARP, N. ELBAUM, et al. Effect of long-distance running on bone mass in women. *J. Bone Miner. Res.* 4:515–522, 1989.

120. Klaber Moffett, J., D. Torgerson, S. Bell-Syer, et al. Randomised controlled trial of exercise for low back pain: clinical outcomes, costs, and preferences. *BMJ* 319:279–283, 1999.
121. Koes, B. W., L. M. Bouter, H. Beckerman, G. J. Van Der Heijden, and P. G. Krupschild. Physiotherapy exercises and back pain: a blinded review. *BMJ* 302:1572–1576, 1991.
122. Kohrt, W. M., D. B. Snead, E. Slatopolsky, and S. J. Birge, Jr. Additive effects of weight-bearing exercise and estrogen on BMD in older women. *J. Bone Miner. Res.* 10:1303–1311, 1995.
123. Kohrt, W. M., K. E. Yarasheski, and J. O. Hollszy. Effects of exercise training on bone mass in elderly women and men with physical frailty. *Bone* 23:S499, 1998.
124. Konradsen, L., E. M. Hansen, and L. Sondergaard. Long distance running and osteoarthrosis. *Am. J. Sports Med.* 18:379–381, 1990.
125. Kontulainen, S., P. Kannus, H. Haapasalo, et al. Changes in BMC with decreased training in competitive young adult tennis players and controls: a prospective 4-yr follow-up. *Med. Sci. Sports Exerc.* 31:646–652, 1999.
126. Kovar, P. A., J. P. Allegrante, C. R. MacKenzie, M. G. E. Peterson, B. Gutin, and M. E. Charlson. Supervised fitness walking in patients with osteoarthritis of the knee. A randomized, controlled trial. *Ann. Intern. Med.* 116:529–534, 1992.
127. Krolner, B., B. Toftr, S. P. Nielsen, and E. Tondevold. Physical exercise as prophylaxis against involutional vertebral bone loss: a controlled trial. *Clin. Sci.* 64:541–546, 1983.
128. Kujala, U. M, J. Kaprio, and S. Sarna. Osteoarthritis of weight bearing joints of lower limbs in former elite male athletes. *BMJ* 308:231–234, 1994.
129. Kujala, U. M., J. Kettunen, H. Paananen, T. Aalto, and M. C. Battie. Knee osteoarthritis in former runners, soccer players, weight lifters and shooters. *Arthritis Rheum.* 38:539–546, 1995.
130. Kujala, U. M., S. Taimela, T. Viljanen, et al. Physical loading and performance as predictors of back pain in healthy adults. A 5-year prospective study. *Eur. J. Appl. Physiol.* 73:452–458, 1996.
131. Lahad, A., A. D., Malter, A. O. Berg, and R. A. Deyo. The effectiveness of four interventions for the prevention of low back pain. *JAMA* 272:1286–1291, 1994.
132. Lane, N. E. Exercise. A cause of osteoarthritis. *J. Rheumatol.* 43:3–6, 1995.
133. Lane, N. E., D. A. Bloch, H. H. Jones, W. H. Marshall, P. D. Wood, and J. F. Fries. Long distance running, bone density and osteoarthritis. *JAMA* 255:1147–1151, 1986.
134. Lane, N. E., D. A. Bloch, P. D. Wood, and J. E. Fries. Aging, long distance running, and the development of musculoskeletal disability. *Am. J. Med.* 82:772–780, 1987.
135. Lane, N. E., D. A. Bloch, H. B. Hubert, et al. Running, osteoarthritis, and bone density: initial 2-year longitudinal study. *Am. J. Med.* 88:452–459, 1990.
136. Lane, N. E., B. A. Micheli, A. Bjorkengren, et al. The risk of osteoarthritis with running and aging: a five year longitudinal study. *J. Rheumatol.* 20:461–468, 1993.
137. Lane, N. E., J. W. Oehlert, D. A. Bloch, and J. F. Fries. The relationship of running to osteoarthritis of the knee and hip and BMD of the lumbar spine: a 9 year longitudinal study. *J. Rheumatol.* 25:334–341, 1998.
138. Lane, N. E., M. C. Hopchberg, A. Pressman, J. C. Scott, and M. C. Nevitt. Recreational physical activity and the risk of osteoarthritis of the hip in elderly women. *J. Rheumatol.* 26:849–854, 1999.
139. Lau, E. M. C., J. Woo, P. C. Leung, R. Swaminathan, and D. Leung. The effects of calcium supplementation and exercise on bone density in elderly Chinese women. *Osteoporos. Int.* 2:168–173, 1992.
140. Layne, J. E., and M. E. Nelson. The effects of progressive resistance training on bone density: a review. *Med. Sci. Sports Exerc.* 31:25–30, 1999.
141. Lee, E. J., K. A. Long, W. L. Risser, H. B. W. Poindexter, W. E. Gibbons, and J. Goldzierh. Variations in bone status of contralateral and regional sites in young athletic women. *Med. Sci. Sports Exerc.* 27:1354–1361, 1995.
142. Leichter, I., A. Simkin, J. Y. Margulies, et al. Gain in mass density of bone following strenuous physical activity. *J. Orthop. Res.* 7:86–90, 1989.
143. Leino, P. I. Does leisure time physical activity prevent low back disorder. *Spine* 18:863–871, 1993.
144. Lidstrom, A., and M. Zachrison. Physical therapy on low back pain and sciatica. *Scand. J. Rehabil. Med.* 2:37–42, 1970.
145. Lindstrom, I., C. Öhlund, C. Eek, et al. The effect of graded activity on patients with subacute low back pain: a randomized prospective clinical study with an operant-conditioning behavioral approach. *Phys. Ther.* 72:279–293, 1992.
146. Lindstrom, I., C. Öhlund, C. Eek, L. Wallin, L.-E. Peterson, and A. Nachemson. Mobility, strength, and fitness after a graded activity program for patients with subacute low back pain. A randomized prospective clinical study with a behavioral therapy approach. *Spine* 17:641–652, 1992.
147. Linton, S. J., L. A. Bradley, I. Jsensen, E. Spangfort, and L. Sundel. The secondary prevention of low back pain. *Pain* 36:197–207, 1989.
148. Linton, S. J., A. L. Hellsing, and G. Bergstrom. Exercise for workers with musculoskeletal pain: does enhancing compliance decrease pain? *J. Occup. Rehabil.* 6:177–190, 1996.
149. Ljunggren, A. E., H. Weber, O. Kogstad, E. Thom, and G. Kirkesola. Effect of exercise on sick leave due to low back pain: a randomized, comparatives, long-term study. *Spine* 22:1610–1617, 1997.
150. Lloyd, T., V. M. Chinchilli, N. R. N. Johnson-Rollings, K. R. D. Kieselhorst, D. F. Eggli, and R. Marcus. Adult female hip bone density reflects teenage sports-exercise patterns but not teenage calcium intake. *Pediatrics* 106:40–44, 2000.
151. Lohman, T., S. Going, R. Pamenter, et al. Effects of resistance training on regional and total BMD in premenopausal women: a randomized prospective study. *J. Bone Miner. Res.* 10:1015–1024, 1995.
152. Lord, S. R., J. A. Ward, P. Williams, and E. Zivanovic. The effects of a community exercise program on fracture risk factors in older women. *Osteoporos. Int.* 6:361–367, 1996.
153. Luoto, S., M. Heliovaara, H. Hurri, and H. Alaranta. Static back endurance and the risk of low back pain. *Clin. Biomechanics* 10:323–324, 1995.
154. Lynch, P., and J. O. Judge. Relationship of prolonged resistance training and femur bone density in older women. *J. Am. Geriatr. Soc.* 40:SA29, 1992.
155. Maddalozzo, G. F., and C. M. Snow. High intensity resistance training: effects on bone in older men and women. *Calcif. Tissue Int.* 66:399–404, 2000.
156. Malmivaara, A., U. Häkkinen, T. Aro, et al. The treatment of acute low back pain—bed rest, exercises or ordinary activity? *N. Engl. J. Med.* 332:351–355, 1995.
157. Mangione, K. K., K. McCully, A. Gloviak, I. Lefebvre, M. Hofmann, and R. Graik. The effects of high-intensity and low-intensity cycle ergometry in older adults with knee osteoarthritis. *J. Gerontol. A. Biol. Sci. Med. Sci.* 54:M184–M190, 1999.
158. Manniche, C., G. Hesselsoe, L. Bentzen, I. Christensen, and E. Lundberg. Clinical trial of intensive muscle training for chronic low back pain. *Lancet* 2:1473–1476, 1988.
159. Manniche, C., K. Asmussen, B. Lauritsen, et al. Intensive dynamic back exercises with or without hyperextension in chronic back pain after surgery for lumbar disc protrusion: a clinical trial. *Spine* 18:560–567, 1993.
160. Mannion, A. F., M. Müntener, S. Taimela, and J. Dvorak. A randomized clinical trial of 3 active therapies for chronic low back pain. *Spine* 24:2435–2448, 1999.
161. Margulies, J. Y., A. Simkin, I. Leichter, et al. Effect of intense physical activity on the bone-mineral content in the lower limbs of young adults. *J. Bone Joint Surg. Am.* 68:1090–1093, 1986.
162. Marshall, D., O. Johnell, and H. Wedel. Meta-analysis of how measures of BMD predict occurrence of osteoporotic fractures. *BMJ* 312:1254–1259, 1996.
163. Marti, B., M. Knobloch, A. Tschopp, A. Jucker, and H. Howald. Is excessive running predictive of degenerative hip disease? Controlled study of former elite athletes. *BMJ* 299:91–93, 1989.

164. Martin, P. R., M. J. Rose, P. J. R. Nichols, P. L. Russel, and J. G. Hughes. Physiotherapy exercises for low back pain: process and clinical outcome. *Int. Rehabil. Med.* 8:34–38, 1980.
165. Martin, D., and M. Notelovitz. Effects of aerobic training on BMD of postmenopausal women. *J. Bone Miner. Res.* 8:931–936, 1993.
166. McAlindon, T. E., P. W. F. Wilson, P. Aliabadi, B. Weissman, and D. T. Felson. Level of physical activity and the risk of radiographic and symptomatic knee osteoarthritis in the elderly: the Framingham Study. *Am. J. Med.* 106:151–157, 1999.
167. McCartney, N., A. L. Hicks, J. Martin, and C. E. Webber. Long-term resistance training in the elderly: effects on dynamic strength, exercise capacity, muscle, and bone. *J. Gerontol*. 50: B97-B104, 1995.
168. McDermott, M., and P. Freyne. Osteoarthrosis in runners with knee pain. *Br. J. Sports Med.* 17:84–87, 1983.
169. McMurdo, M. E. T., P. A. Mole, and C. R. Paterson. Controlled trial of weight-bearing exercise in older women. *BMJ* 314:569, 1997.
170. Menkes, A., S. Mazel, R. A. Redmond, et al. Strength training increases regional BMD and bone remodeling in middle-aged and older men. *J. Appl. Physiol.* 74:2478–2484, 1993.
171. Minor, M. A., J. E. Hewett, R. R. Weber, S. K. Anderson, and D. R. Kay. Efficacy of physical conditioning exercise in patients with rheumatoid arthritis and osteoarthritis. *Arthritis Rheum.* 32:1396–1405, 1989.
172. Morris, F. L., G. A. Naughton, J. L. Gibbs, J. S. Carslon, and J. D. Wark. Prospective ten-month exercise intervention in premenarcheal girls: positive effects on bone and lean mass. *J. Bone Miner. Res.* 12:1453–1462, 1997.
173. Morris, F. L., W. R. Payne, and J. D. Wark. The impact of intense training on endogenous estrogen and progesterone concentrations and bone mineral acquisition in adolescent rowers. *Osteoporos. Int.* 10:361–368, 1999.
174. Mundt, D. J. J. L. Kelsey, A. L. Golden, et al. An epidemiologic study of sports and weight lifting as possible risk factors for herniated lumbar and cervical discs. *Am. J. Sports Med.* 21:854–860, 1993.
175. Murray, R. O., and C. Duncan. Athletic activity in adolescence as an etiological factor in degenerative hip disease. *J. Bone Joint Surg. Br.* 53:406–419, 1971.
176. Myerson, M., B. Gutin, M. P. Warren, et al. Total body bone density in amenorrheic runners. *Obstet. Gynecol.* 79:973–978, 1992.
177. National Osteoporosis Foundation. Osteoporosis. review of the evidence for prevention, diagnosis, and treatment and cost-effectiveness analysis. Status report. *Osteoporos. Int.* 8:S1–S88, 1998.
178. Nelson, M. E., E. C. Fisher, F. A. Dilmanian, G. E. Dallal, and W. J. Evans. A 1-y walking program and increased dietary calcium in postmenopausal women: effects on bone. *Am. J. Nutr.* 53:1304–1311, 1991.
179. Nelson, M. E., M. A. Fiatarone, C. M. Morganti, I. Trice, R. A. Greenbert, and W. J. Evans. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: a randomized controlled trial. *JAMA* 272:1909–1914, 1994.
180. Nichols, D. L., C. F. Sanborn, S. L. Bonnick, V. Benezra, B. Gench, and N. M. Dimarco. The effects of gymnastics training on BMD. *Med. Sci. Sports Exerc*. 26:1220–1225, 1994.
181. Nichols, D. L., C. F. Sanborn, S. L. Bonnick, B. Gench, and N. DiMarco. Relationship of regional body composition to BMD in college females. *Med. Sci. Sports Exerc.* 27:178–182, 1995.
182. Nickols-Richardson, S. M., C. M. Modlesky, P. J. O'Connor, and R. D. Lewis. Premenarcheal gymnasts possess higher BMD than controls. *Med. Sci. Sports Exerc.* 32:63–69, 2000.
183. Nordstrom, P., G. Nordstrom, and R. Lorentzon. Correlation of bone density to strength and physical activity in young men with a low or moderate level of physical activity. *Calcif. Tissue Int.* 60:332–337, 1997.
184. Notelovitz, M., D. Martin, R. Tesar, et al. Estrogen therapy and variable-resistance weight training increases bone mineral in surgically menopausal women. *J. Bone Miner. Res.* 6:583–590, 1991.
185. Nwuga, V. C. B. Relative therapeutic efficacy of vertebral manipulation and conventional treatment in back pain management. *Am. J. Phys. Med.* 61:273–278, 1982.
186. Nwuga, G., and V. Nwuga. Relative therapeutic efficacy of the Williams and McKenzie protocols in back pain management. *Physiother. Pract.* 1:99–105, 1985.
187. O'Reilly, S. C., K. R. Muir, and M. Doherty. Effectiveness of home exercise on pain and disability from osteoarthritis of the knee: a randomised controlled trial. *Ann. Rheum. Dis.* 58:15–19, 1999.
188. Orwoll, E. S., J. Ferar, S. K. Oviatt, M. R. McClung, and K. Huntington. The relationship of swimming exercise to bone mass in men and women. *Arch. Intern. Med.* 149:2197–2200, 1989.
189. O'Sullivan, P. B., L. T. Twomey, and G. T. Allison. Evaluation of specific stabilising exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine* 22:2959–2967, 1997.
190. Panush, R. S., and J. D. Inzinna. Recreational activities and degenerative joint disease. *Sports Med.* 17:1–5, 1994.
191. Panush, R. S., C. Schmidt, J. R. Caldwell, et al. Is running associated with degenerative joint disease? *JAMA* 255:1152–1154, 1986.
192. Pearce, G., S. Bass, N. Young, C. Formica, and E. Seeman. Does weight-bearing exercise protect against the effect of exercise induced oligomenorrhea on bone density? *Osteoporos. Int.* 6:448–452, 1996.
193. Peterson, S. E., M. D. Peterson, G. Raymond, C. Gilligan, M. M. Checovich, and E. L. Smith. Muscular strength and bone density with weight training in middle-aged women. *Med. Sci. Sports Exerc.* 23:449–504, 1991.
194. Pettersson, U., H. Alfredson, P. Nordstrom, K. Henriksson-Larsen, and R. Lorentzon. Bone mass in female cross-country skiers: relationship between muscle strength and different MBD sites. *Calcif. Tissue Int.* 67:199–206, 2000.
195. Pettersson, U., P. Nordstrom, H. Alfredson, K. Henriksson-Larsen, and R. Lorentzon. Effect of high impact activity on bone mass and size in adolescent females: a comparative study between two different types of sports. *Calcif. Tissue Int.* 67:207–214, 2000.
196. Picavet, H. S. J., and S. A. G. Schouten. Physical load in daily life and low back problems in the general population—the MORGEN Study. *Prev. Med.* 31:506–512, 2000.
197. Preisinger, E., Y. Alacamlioglu, K. Pils, et al. Exercise therapy for osteoporosis: results of a randomised controlled trial. *Br. J. Sports Med.* 30:209–212, 1996.
198. Prince E. T. R., A. Devine, I. Dick, et al. The effects of calcium supplementation (milk powder or tablets) and exercise on bone density in postmenopausal women. *J. Bone Miner. Res.* 10: 1068–1075, 1995.
199. Pruitt, L. A., R. D. Jackson, R. L. Bartels, and H. J. Lehnhard. Weight training effects on BMD in early postmenopausal women. *J. Bone Miner. Res.* 7:179–185, 1992.
200. Pruitt, L. A., D. R. Taaffe, and R. Marcus. Effects of a one-year high-intensity versus low-intensity resistance training program on BMD in older women. *J. Bone Miner. Res.* 10:1788–1795, 1995.
201. Puranen, J., L. Ala-Ketola, P. Peltokallio, and J. Saarela. Running and primary osteoarthritis of the hip. *BMJ* 1:424–425, 1975.
202. Recker, R. R., K. M. Davies, S. M. Hinders, R. P. Heaney, M. R. Stegman, and D. B. Kimmel. Bone gain in young adult women. *JAMA* 268:2403–2408, 1992.
203. Revel, M., M. A. Mayoux-Benhamou, J. P. Rabourdin, F. Bagheri, and C. Roux. One-year psoas training can prevent lumbar bone loss in postmenopausal women: a randomized controlled trial. *Calcif. Tissue Int.* 53:307–311, 1993.
204. Rhodes, E. C., A. D. Martin, J. E. Taunton, M. Donnelly, J. Warren, and J. Elliot. Effects of one year of resistance training on the relation between muscular strength and bone density in elderly women. *Br. J. Sports Med.* 34:18–22, 2000.
205. Riihimaki, H., E. Viikari-Juntura, G. Moneta, J. Kuha, T. Videman, and S. Tola. Incidence of sciatic pain among men in

machine operating, dynamic physical work, and sedentary work. A three-year follow-up. *Spine* 19:138–142, 1994.

206. Risch, S. V., N. K. Norvell, M. L. Pollock, et al. Lumbar strengthening in chronic low back pain patients. Physiologic and psychological benefits. *Spine* 18:232–238, 1993.
207. Risser, W. L., E. J. Lee, A. Leblanc, H. B. W. Poindexeter, J. M. H. Risser, and V. Schneider. Bone density in eumenorrheic female college athletes. *Med. Sci. Sports Exerc.* 22:570–574, 1990.
208. Robinson, T. L., C. Snow-Hartger, D. R. Taaffe, D. Gillis, J. Shaw, and R. Marcus. Gymnasts exhibit higher bone mass than runners despite similar prevalence of amenorrhea and oligomenorrhea. *J. Bone Miner. Res.* 10:26–35, 1995.
209. Rockwell, J. C., A. M. Sorensen, S. Baker, et al. Weight training decreases vertebral bone density in premenopausal women: a prospective study. *J. Endocrinol. Metabol.* 71:988–993, 1990.
210. Roos, H., H. Lindberg, P. Gärdsell, L. S. Lohmander, and H. Wingstrand. The prevalence of gonarthrosis and its relation to meniscectomy in former soccer players. *Am. J. Sports Med.* 22:219–222, 1994.
211. Ruiz, J. C., C. Mandel, and M. Garabedian. Influence of spontaneous calcium intake and physical exercise on the vertebral and femoral BMD of children and adolescents. *J. Bone Miner. Res.* 10:675–682, 1995.
212. Rutherford, O. M. Is there a role for exercise in the prevention of osteoporotic fractures? *Br. J. Sports Med.* 33:378–386, 1999.
213. Säämanen, A.-M. *Articular Cartilage Proteoglycans and Joint Loading. A Study in Young Rabbits and Dogs.* Doctoral dissertation. Kuopio, Finland: University of Kuopio, 1989.
214. Sachs, B. L., S. S. Ahmad, M. Lacroix, D. Olimpi, R. Heat, J. A. David, and A. D. Scala. Objective assessment for exercise treatment on the B-200 isostation as part of work tolerance rehabilitation: a random prospective blind evaluation with comparison control population. *Spine* 19:49–52, 1994.
215. Salamone, L. M., J. A. Cauley, D. M. Black, et al. Effect of a lifestyle intervention on BMD in premenopausal women: a randomized trial. *Am. J. Clin. Nutr.* 70:97–103, 1999.
216. Sandmark, H., and E. Vingård. Sports and risk for severe osteoarthrosis of the knee. *Scand. J. Med. Sci. Sports* 9:279–284, 1999.
217. Sandstrom, P., P. Jonsson, R. Lorentzon, and K. Thorsen. BMD and muscle strength in female ice hockey players. *Int. J. Sports Med.* 21:524–528, 2000.
218. Saxon, L., C. Finch, and S. Bass. Sports participation, sports injuries and osteoarthritis. Implications for prevention. *Sports Med.* 28:123–135, 1999.
219. Schilke, J. M., G. O. Johnson, T. J. Housh, and J. R. O'Dell. Effects of muscle-strength training on the functional status of patients with osteoarthritis of the knee joint. *Nurs. Res.* 45:68–72, 1996.
220. Seferlis, T., G. Nemeth, A. M. Carlsson, and P. Gillstrom. Conservative treatment in patients sicklisted for acute low-back pain: a prospective randomised study with 12 months' follow-up. *Eur. Spine J.* 7:461–470, 1998.
221. Simkin, A., J. Ayalon, and I. Leichter. Increased trabecular bone density due to bone-loading exercises in postmenopausal osteoporotic women. *Calcif. Tissue Int.* 40:59–63, 1987.
222. Sinaki, M., H. W. Wahner, K. P. Offord, and S. F. Hodgson. Efficacy of nonloading exercises in prevention of vertebral bone loss in postmenopausal women: a controlled trial. *Mayo Clin. Proc.* 64:762–769, 1989.
223. Slemenda, C. W., and C. C. Johnston. High intensity activities in young women: site specific bone mass effects among male figure skaters. *Bone Miner.* 20:125–132, 1993.
224. Smidt, G. L., S. Lin, K. D. O'Dwyer, and P. R. Blanpied. The effect of high-intensity trunk exercise on BMD of postmenopausal women. *Spine* 17:280–285, 1992.
225. Smith, E. L., and C. Gilligan. Dose-response relationship between physical loading and mechanical competence of bone. *Bone* 18:45S–50S, 1996.
226. Snook, S. H., B. S. Webster, R. W. McGorry, M. T. Fogleman, and B. McCann. The reduction of chronic nonspecific low back pain through the control of early morning lumbar flexion. A randomized controlled trial. *Spine* 23:2601–2607, 1998.
227. Snow-Harter, C., M. L. Bouxsein, B. T. Lewis, D. R. Carter, and R. Marcus. Effects of resistance and endurance exercise on bone mineral status of young women: a randomized exercise intervention trial. *J. Bone Miner. Res.* 7:761–769, 1992.
228. Söderman, K., E. Bergstrom, R. Lorentzon, and H. Alfredson. Bone mass and muscle strength in young female soccer players. *Calcif. Tissue Int.* 67:297–303, 2000.
229. Sohn, R. S., and L. J. Micheli. The effect of running on the pathogenesis of osteoarthritis of the hips and knees. *Clin. Orthop.* 198:106–109, 1985.
230. Spector, T. D., P. A. Harris, D. J. Hart, et al. Risk of osteoarthritis associated with long-term weight-bearing sports. *Arthritis Rheum.* 39:988–995, 1996.
231. Stankovic, R., and O. Johnell. Conservative treatment of acute low back pain: a prospective randomized trial. *Spine* 15:120–123, 1990.
232. Stankovic, R., and O. Johnell. Conservative treatment of acute low-back pain: a 5-year follow-up study of two methods of treatment. *Spine* 20:469–472, 1995.
233. Statens Beredning För Medicinsk Utvardering (SBU). *Pain in Back, Pain in Neck. Summary of Scientific Evidence,* Vol 1–2. [in Swedish]. Stockholm: Statens Beredning för Medicinsk Utvärdering, 2000, p. 806.
234. Suni, J. *Health-Related Fitness Test Battery for Middle-Aged Adults with Emphasis on Musculoskeletal and Motor Tests.* Doctoral Dissertation. Jyväskylä, Finland: University of Jyväskylä, January 2000.
235. Suominen, H., S. Cheng, and P. Rahkila. Physical training and bone in elderly male and female athletes. In: *Abstracts of the 3rd International Conference of Physical Activity, Aging and Sports.* Jyväskylä, Finland: University of Jyväskylä, 1992, pp.90–91.
236. Taaffe, D. R., C. Snow-Harter, D. A. Connolly, T. L. Robinson, M. D. Brown, and R. Marcus. Differential effects of swimming vs. weight-bearing activity on bone mineral status of eumenorrheic athletes. *J. Bone Miner. Res.* 10:586–593, 1995.
237. Taaffe, D. R., T. L. Robinson, C. M. Snow, and R. Marcus. High-impact exercise promotes bone gain in well-trained female athletes. *J. Bone Miner. Res.* 12:255–260, 1997.
238. Taaffe, D. R., and R. Marcus. Regional and total body BMD in elite collegiate male swimmer. *J. Sports Med. Phys. Fitness* 39:154–159, 1999.
239. Teegarden, D., W. R. Proulx, M. Kern, et al. Previous physical activity relates to bone mineral measures in young women. *Med. Sci. Sports Exerc.* 28:105–113, 1996.
240. Torstensen, T. A., A. E. Ljunggren, H. D. Meen, E. Odland, P. Mowinckel, and S. Geijerstam. Efficiency and costs of medical exercise therapy, conventional physiotherapy, and self-exercise in patients with chronic low back pain: a pragmatic, randomized, single-blinded, controlled trial with 1-year follow-up. *Spine* 23:2616–2624,1998.
241. Tsukahara, N., A. Toda, J. Goto, and I. Ezawa. Cross-sectional and longitudinal studies on the effect of water exercise in controlling bone loss in Japanese postmenopausal women. *J. Nutr. Sci. Vitaminol.* 40:37–47, 1994.
242. Turner, J. A., S. Clancy, K. J. McQuade, and D. D. Cardenas. Effectiveness of behavioral therapy for chronic low back pain: a component analysis. *J. Consult. Clin. Psychol.* 58:573–579, 1990.
243. Turner, C. H. Three rules for bone adaptation to mechanical stimuli. *Bone* 23:399–407, 1998.
244. Underwood, M. R., and J. Morgan. The use of a back class teaching extension exercises in the treatment of acute low back pain in primary care. *Fam. Pract.* 15:9–15, 1998.
245. Valdimarsson, Ö., J. Ö. Krsitinsson, S. Ö. Stefansson, S. Valdimarsson, and G. Sigurdsson. Lean mass and physical activity as predictors of BMD in 16–20-year old women. *J. Intern. Med.* 245:489–496, 1999.
246. Välimaki, M. J., M. Kärkkäinen, C. Lamberg-Allartdt, et al. Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. *BMJ* 309:230–235, 1994.

247. Van Baar, M. E., J. Dekker, R. A. B. Oostendorp, et al. The effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee: a randomized clinical trial. *J. Rheumatol.* 25: 2432–2439, 1998.
248. Van Baar, M. E., W. J. J. Assendeleft, J. Dekker, R. A. B. Oostendorp, and J. W. J. Bijlsma. Effectiveness of exercise therapy in patients with osteoarthritis of the hip or knee. A systematic review of randomized clinical trials. *Arthritis Rheum.* 42:1361–1369, 1999.
249. Van Tulder, M. W., W. J. J. Assendelft, B. W. Koes, L. M. Bouter, and The Editorial Board of the Cochrane Collaboration Back Review Group. Method guidelines for systematic reviews in the Cochrane Collaboration Back Review Group for Spinal Disorders. *Spine* 22:2323–2330, 1997.
250. Van Tulder, M. W., A. Malmivaara, R. Esmail, and B. W. Koes. Exercise therapy for low back pain. (Cochrane Review). In: *The Cochrane Library,* Issue 4. Oxford: Update Software, 2000.
251. Videman, T., T. Nurminen, and J. D. G. Troup. Lumbar spine pathology in cadaveric material in relation to history of back pain, occupation, and physical loading. *Spine* 15:728–737, 1990.
252. Videman T., S. Sarna, M. C. Battie, et al. The long term effects of physical loading and exercise lifestyle on back-related symptoms, disability, and spinal pathology among men. *Spine* 20: 699–709, 1995.
253. Vingård, E., L. Alfredsson, I. Goldie, and C. Hogstedt. Occupation and osteoarthrosis of the hip and knee: a register-based cohort study. *Int. J. Epidemiol.* 20:1025–1031, 1991.
254. Vingård, E., L. Alfredsson, I. Goldie, and C. Hogstedt. Sports and osteoarthrosis of the hip. An epidemiologic study. *Am. J. Sports Med.* 21:195–200, 1993.
255. Vingård, E., L. Alfredsson, and H. Malchau. Osteoarthrosis of the hip in women and its relationship to physical load from sports activities. *Am. J. Sports Med.* 26:78–82, 1998.
256. Vuori, I., A. Heinonen, H. Sievanen, P. Kannus, M. Pasanen, and P. Oja. Effects of unilateral strength training and detraining on BMD and content in young women: a study of mechanical loading and deloading on human bones. *Calcif. Tissue Int.* 55: 59–67, 1994.
257. Vuori, I. Peak bone mass and physical activity: a short review. *Nutr. Rev.* 54:S11–S14, 1996.
258. Vuori, I., and A. Heinonen. Sport and bone. In: *Women in Sport,* B. L. Drinkwater (Ed.). Oxford: Blackwell Science, 2000, pp. 280–300.
259. Waddell, G., G. Feder, and M. Lewis. Systematic reviews of bed rest and advice to stay active for acute low back pain. *Br. J. Gen. Pract.* 47:647–652, 1997.
260. Wallace, B. A., and R. G. Cumming. Systematic review of randomized trials of the effect of exercise on bone mass in pre- and postmenopausal women. *Calcif. Tissue Int.* 67:10–18, 2000.
261. Waterworth, R. F., and I. A. Hunter. An open study of diflunisal, conservative and manipulative therapy in the management of acute mechanical low back pain. *N. Z. Med. J.* 95:373–375, 1985.
262. Welten, D. C., H. C. G. Kemper, G. B. Post, et al. Weight-bearing activity during youth is a more important factor for peak bone mass than calcium intake. *J. Bone Miner. Res.* 9:1089–1096, 1994.
263. White, A. W. M. Low back pain in men receiving workmen's compensation. *Can. Med. Assoc. J.* 95:50–56, 1996.
264. Winters, K. M., M. Titus, and C. M. Snow. Progressive jump and lower body resistance training hip bone mass in premenopausal women (Abstract). ACSM Annual Meeting, 1999.
265. Witzke, K. A., and C. M. Snow. Effects of plyometric jump training on bone mass in adolescent girls. *Med. Sci. Sports Exerc.* 32:1051–1057, 2000.
266. Wolff, I., J. Van Croonenborg, H. C. G. Kemper, P. J. Kostense, and J. W. R. Twisk. The effect of exercise training programs on bone mass: a meta-analysis of published controlled trials in pre- and postmenopausal women. *Osteoporos. Int.* 9:1–12, 1999.
267. Wolman, R. L., L. Faulman, P. Clark, R. Hesp, and M. G. Harries. Different training patterns and BMD of the femoral shaft in elite, female athletes. *Ann. Rheum. Dis.* 50:487–489, 1991.
268. World Health Organization. *Assessment of Fracture Risk and its Application to Screening for Postmenopausal Osteoporosis.* WHO Technical Report Series, No. 843. Geneva: WHO, 1994.
269. Zylbergold, R. S., and M. C. Piper. Lumbar disc disease: comparative analysis of physical therapy treatments. *Arch. Phys. Med. Rehabil.* 62:176–179, 1981.