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論文名	A prospective study of overweight, physical activity, and depressive symptoms in young women
著 者	Ball K, Burton NW, Brown WJ.
雑誌名	Obesity (Silver Spring).
巻・号・頁	17 1 66-71
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PubMedリンク	http://www.ncbi.nlm.nih.gov/pubmed/18997676

対象の内訳	ヒト	動物	地 域	その他	研究の種類	縦断研究
対象	一般健常者	空白		( )		コホート研究
性別	女性	( )		( )		( )
年齢	22-27(24.5歳)	[redacted]		( )		前向きコホート
対象数	5000～10000			( )		( )
調査の方法	質問紙	( )				

アウトカム						
予 防	なし	なし	なし	なし		( )
維持・改善	な し	な し	な し	心理的指標改善	( )	( )

図 表

Table 3 Unadjusted and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for 2003 CESD-10 scores ≥10 by 2000 BMI (Model 1) and 2000 physical activity (Model 2) categories

	N	Unadjusted OR	95% CI		OR adjusted for covariates[a]	95% CI		OR fully adjusted[b]	95% CI	
Model 1: 2000 BMI										
Healthy weight	3,858	1.00			1.00			1.00		
Underweight	401	1.26	0.99	1.60	1.09	0.84	1.43	1.09	0.83	1.42
Overweight	1,207	**1.32**	**1.14**	**1.53**	**1.21**	**1.02**	**1.43**	**1.21**	**1.03**	**1.43**
Obese	610	**1.81**	**1.50**	**2.18**	**1.34**	**1.09**	**1.67**	**1.33**	**1.08**	**1.65**
Missing	601	1.33	1.10	1.62	1.07	0.85	1.34	1.07	0.85	1.34
Model 2: 2000 physical activity										
None	583	1.00			1.00			1.00		
Very low	1,164	**0.72**	**0.58**	**0.90**	**0.90**	0.70	1.15	0.91	0.71	1.16
Low	1,061	**0.58**	**0.46**	**0.72**	**0.76**	**0.59**	**0.99**	**0.77**	**0.59**	**0.99**
Moderate	1,573	**0.60**	**0.49**	**0.74**	0.82	0.65	1.04	0.82	0.65	1.05
High	2,145	**0.54**	**0.45**	**0.66**	**0.78**	**0.62**	**0.98**	**0.79**	**0.63**	**1.00**
Missing	151	**0.61**	**0.41**	**0.93**	1.28	0.76	2.14	1.29	0.77	2.17

Significant associations shown in boldface.
[a]Covariates were 2003 highest educational qualification, 2003 marital status, 2003 occupation, 2003 smoking status, parity status across all surveys, 2003 health problem/disability and 2000 CESD-10 score. [b]Covariates were those above, as well as 2000 physical activity (Model 1) and 2000 BMI (Model 2).

図表掲載箇所	
概 要 (800字まで)	＜目的＞BMIと余暇身体活動とうつ発症との関係を前向きに検討すること。＜方法＞コホート名：Australian Longitudinal Study on Women's Health、対象者数：6677人、追跡期間：3年、身体活動評価方法詳細：余暇身体活動。先週の通勤や余暇での歩行と中高強度身体活動の時間と頻度を思い出し法で評価。、因子の単位：メッツ・分/週。余暇身体活動量により5分位に別けた。分位1：0-39メッツ・分／週、分位2：40-299メッツ・分／週、分位3：300-599メッツ・分／週、分位4：600-1199メッツ・分／週、分位5：1200-無制限メッツ・分／週であった。＜結果＞余暇身体活動とうつ発症リスクとの関係は、分位1：1、分位2：0.91(0.71-1.16)、分位3：0.77(0.59-0.99)、分位4：0.82(0.65-1.05)、分位5：0.79(0.63-1)であった。過体重や肥満ではうつ発症のリスクが増加することも明らかとなった。身体活動によるうつリスクの低下はBMIで補正しても有意であった。
結 論 (200字まで)	歩行を含む中強度以上の身体活動が300メッツ分/週≒5メッツ時/週以上なら、若者女性のうつ発症リスクが低い。
エキスパートによるコメント (200字まで)	若い女性を対象として、うつの発症を週5メッツ・時／週という運動基準の運動量の基準値とほぼ同量の余暇身体活動で予防できることを示した研究。

担当者 宮地元彦

Prospective Study of Recreational Physical Activity and Ovarian Cancer

Elizabeth R. Bertone, Walter C. Willett, Bernard A. Rosner, David J. Hunter, Charles S. Fuchs, Frank E. Speizer, Graham A. Colditz, Susan E. Hankinson

Background: **It has been hypothesized that physical activity may reduce the risk of ovarian cancer by decreasing estrogen levels, reducing body fat, and reducing the frequency of ovulation. Epidemiologic studies of this relationship have obtained inconsistent results. The only prospective study to date reported a positive association between frequent vigorous exercise and ovarian cancer risk. We further evaluated this relationship in the Nurses' Health Study cohort.** ***Methods:*** **Participation in recreational physical activity was assessed by questionnaire in 1980, 1982, 1986, 1988, 1992, and 1994, with questions assessing exercise frequency, duration, and intensity. Results were adjusted for age, parity, oral contraceptive use, tubal ligation, and other risk factors for ovarian cancer. All statistical tests were two-sided.** ***Results:*** **During a 16-year follow-up (from 1980 to 1996), 1.2 million person-years were accrued by 92 825 cohort members, and 377 cases of epithelial ovarian cancer were confirmed. The relative risk (RR) of ovarian cancer for women engaging in recreational physical activity for 7 hours or more per week compared with those reporting less than 1 hour per week was 0.80 (95% confidence interval [CI] = 0.49 to 1.32; P_{trend} = .59). When both the frequency and intensity of activity were taken into account, activity level was also not associated with a reduced risk of ovarian cancer. Compared with inactive women, participants reporting high activity in terms of metabolic equivalent task hours (MET hours) were at greater risk of ovarian cancer (RR for 20 to <30 MET hours/week = 1.84 [95% CI = 1.12 to 3.02]; RR for >30 MET hours/week = 1.27 [95% CI = 0.75 to 2.14]).** ***Conclusions:*** **Overall, results did not suggest an inverse association between recreational physical activity and ovarian cancer. The possibility of a modest increase in risk with frequent vigorous activity requires further investigation.** **[J Natl Cancer Inst 2001;93:942–8]**

It has been hypothesized that participation in recreational physical activity may reduce a woman's risk of ovarian cancer by decreasing estrogen levels, reducing body fat, and, with extreme exercise, reducing the frequency of ovulation *(1–3)*. Several case–control studies *(4–8)* have evaluated this association with inconsistent results, some studies reporting a reduced risk of ovarian cancer with frequent activity and other studies *(9,10)* reporting no association or a slight increase in risk with greater exercise. The single prospective study *(11)* to address this relationship found a statistically significant increased risk of ovarian cancer in postmenopausal women with frequent and vigorous activity, with evidence of a dose–response relationship. Women engaging in vigorous physical activity four or more times per week experienced a 2.5-fold increase in risk compared with those rarely engaging in vigorous activity. These results have raised important questions as to how intensity, type, and timing of exercise may relate to risk of ovarian cancer. We further evaluated the association between physical activity and risk of ovarian cancer in the Nurses' Health Study (NHS) cohort.

Subjects and Methods

NHS Cohort

The NHS is a cohort of 121 700 U.S. female registered nurses who responded to a mailed questionnaire in 1976 *(12)*. The participants were 30–55 years old at the time of the initial mailing and provided information on their medical history and health-related behaviors, such as use of oral contraceptives and smoking status; they have completed questionnaires every 2 years thereafter to update information on various risk factors and to identify new diagnoses of cancer and other diseases. As of 1996, the rate of follow-up was 90%, with vital status data available for more than 98% of the original cohort.

The protocol for the study was approved by the Human Research Committees of the Brigham and Women's Hospital and the Harvard Medical School, Boston, MA.

Exposure Assessment

On the 1980 NHS questionnaire, participants were asked about the number of hours per weekday and weekend day they spent involved in vigorous physical activity (e.g., vigorous sports, brisk walking, and bicycling on hills) and moderate activity (e.g., bicycling on level ground, walking, and light sports). The participants also were asked if they ever exercised intensely enough to work up a sweat and, if so, to report the type of activity that led to sweating and the number of times per week they engaged in it. A similar question was included on the 1982 questionnaire, which asked how many hours per week they engaged in physical activity that led to sweating. These questions have been validated previously *(13–16)* and have been found to predict resting heart rate, obesity, and levels of high-density lipoprotein-bound cholesterol *(15,16)*.

Beginning in 1986, the participants were asked about the amount of time each week that they engaged in specific activities. These activities included jogging (i.e., slower than 10 minutes per mile), running (i.e., 10 minutes per mile or faster), bicycling (including on a stationary machine), lap swimming, playing tennis, squash, or racquetball, performing aerobic activities (including calisthenics, aerobic dance, and rowing on a machine), and walking or hiking outdoors (including walking at golf). In addition, the participants were asked about their usual walking pace and the number of flights of stairs that they climbed each day. These questions were repeated on the 1988, 1992, and 1994 questionnaires, with questions on lower intensity activity (e.g., yoga and stretching) and other vigorous activities (e.g., lawn mowing) added in 1994. These questions have been validated for use in a similar population, the NHS II cohort, described in detail elsewhere *(17)*. Briefly, in a sample of 153 cohort members, correlations were 0.59 (95% confidence interval [CI] = 0.48 to 0.69), for a total weekly activity score as measured by two questionnaires completed 1 year apart, and 0.79 (95% CI = 0.64 to 0.88) for four past-week activity recalls and the second questionnaire.

Covariate Assessment

Information on ovarian cancer risk factors was collected by questionnaire throughout the 16-year

Affiliations of authors: E. R. Bertone, G. A. Colditz, S. E. Hankinson, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, and Department of Epidemiology, Harvard School of Public Health, Boston, MA; W. C. Willett, D. J. Hunter, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, and Departments of Epidemiology and Nutrition, Harvard School of Public Health; B. A. Rosner, F. E. Speizer, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School; C. S. Fuchs, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, and Dana-Farber Cancer Institute, Boston.

Correspondence to: Elizabeth R. Bertone, Sc.D., Department of Biostatistics and Epidemiology, University of Massachusetts, 403 Arnold House, 715 N. Pleasant St., Amherst, MA 01003 (e-mail: ebertone@schoolph.umass.edu).

Reprint requests to: Susan E. Hankinson, Sc.D., Channing Laboratory, 181 Longwood Ave., Boston, MA 02115 (e-mail: Sue.Hankinson@channing.harvard.edu).

See "Notes" following "References."

follow-up. We updated data on most factors every 2 years, including menopausal status, postmenopausal hormone use, smoking status, hysterectomy, and body mass index (BMI) [weight in kilograms/(height in m)^2 or kg/m^2]. A semiquantitative food-frequency questionnaire was included on the 1980 questionnaire to measure the intake of lactose, caffeine, alcohol, and total energy. We updated dietary intake information with subsequent food-frequency questionnaires in 1984, 1986, and 1990. Information on several risk factors was collected during only part of the follow-up period. For example, questions on oral contraceptive use were included on questionnaires through 1982, at which point fewer than 1% of the participants were still using oral contraceptives. We considered women who reported past use of oral contraceptives in 1982 to be past users for the remainder of follow-up and those who were current users in 1982 to be past users from 1984 onward. We updated parity (measured until 1984) and tubal ligation (measured until 1982) in a similar fashion. Information on age at menarche, age at birth of the first child, talc use, and menstrual irregularity was collected once and then carried forward throughout the follow-up period.

Disease Assessment

On each follow-up questionnaire, the participants were asked whether they had been diagnosed with ovarian cancer by a physician during the previous 2 years. Women reporting a diagnosis of ovarian cancer were asked for permission to review their medical records. Records were reviewed by physicians blinded to the participant's exposure status to confirm the diagnosis and to identify histologic type and subtype and invasiveness. Only confirmed cases of epithelial ovarian cancer (International Classification of Diseases for Oncology: 183) *(18)* were included in the analysis. In addition, the National Death Index was searched systematically to identify women who died of ovarian cancer before reporting a diagnosis by questionnaire; we then contacted family members to obtain medical records. Approximately 98% of all deaths among cohort members are identified by searching the National Death Index *(19)*.

During the 16-year follow-up, 511 cases of ovarian cancer were reported by cohort members. We received medical records for 441 (86.3%) of these cases, record receipt was still pending for six cases, 23 women denied the diagnosis when contacted, 35 women refused to release their records, three women could not be contacted, and death certificates alone were available for three women. After review of medical records, the diagnosis of ovarian cancer was confirmed for 402 (91.2%) of the 441 participants. Of the 39 cases not confirmed, 11 diagnoses were rejected, 18 were changed to a more correct diagnosis, and 10 were found to be metastases from other tumors. Overall, 377 (93.8%) of the 402 confirmed cases were classified as epithelial tumors. This group included 224 tumors of the serous subtype, 65 of the mucinous subtype, and 53 of the endometrioid subtype. Ninety percent (n = 338) of the tumors were invasive.

Statistical Analysis

Participants were excluded from the analysis at baseline if they reported a diagnosis of cancer or a bilateral oophorectomy before the start of follow-up in 1980. Person-years of follow-up accrued from the date of return of the 1980 questionnaire until a diagnosis of ovarian or other cancer, a report of bilateral oophorectomy, death, or the end of the follow-up period on June 1, 1996, whichever came first. For each participant, follow-up time equal to the number of months between the return of the 1980 questionnaire and the return of the 1982 questionnaire was assigned to a category of physical activity participation on the basis of the level reported in 1980 (e.g., hours per week of physical activity in 1980). Similarly, for each subsequent 2-year interval, additional months of follow-up were assigned according to the updated information on physical activity reported at the beginning of the interval. For exposures reported only once (e.g., hours per week of physical activity at baseline), the initial response level was carried forward throughout the follow-up period.

Several variables were used to address how frequency, intensity, and consistency of participation in physical activity affected ovarian cancer risk. We evaluated the total amount of time the participants reported engaging in physical activity each week (<1, 1 to <2, 2 to <4, 4 to <7, and ≥7 hours/week) in 1980, 1982, 1986, 1988, 1992, and 1994. Thus, in addition to evaluating the risk associated with baseline activity level in 1980, we were able to assess the effect of recent activity participation by using the activity level reported on each questionnaire to predict risk in the subsequent 2-year period. Person-time was allocated to each of the five categories of physical activity participation in 2-year blocks, allowing each participant to change exposure status every 2 years. For the years in which physical activity questions were not included on the questionnaire (1984 and 1990), the level reported during the previous period was carried forward another 2 years. For example, hours of activity per week reported in 1980 were used to predict cancer risk from 1980 to 1982; similarly, the level reported in 1982 was used to predict risk from 1982 to 1984 and from 1984 to 1986. If a participant did not provide information on activity level in a given year, she was assigned a missing value for that year.

To best represent the overall level of physical activity during the follow-up period, we calculated the cumulative average of the exercise levels reported on all previous questionnaires. For example, the average physical activity (hours/week) in 1980 and 1982 was used to predict ovarian cancer risk from 1982 to 1986. Similarly, the average activity level in 1980, 1982, and 1986 was used to predict risk from 1986 to 1988. For the years in which physical activity questions were not included on the questionnaire (1984 and 1990), the cumulative average calculated during the previous time period was carried forward another 2 years (e.g., the average of 1980 and 1982 was used to predict cancer risk from 1982 to 1984 and from 1984 to 1986). If a participant did not complete the physical activity section of the questionnaire on any given year, she was assigned a missing value for that year; the cumulative average for that period was then calculated by use of data from all other previous years.

To compare the risk of ovarian cancer in women who were consistently very active over several years with that in women who were consistently inactive, we averaged the number of hours of activity per week that each woman reported in 1980, 1982, and 1986. This average was then used to predict cancer risk from 1986 to 1996. For participants who only completed two of the three questionnaires, we used the average of the two measurements; women who responded to only one questionnaire during this period were assigned a missing value for this analysis.

In addition, we classified participants on the basis of the type and intensity of activity reported. On the basis of their response to the 1980 questionnaire, we divided participants into three groups by whether they reported engaging in exercise intensely enough to produce sweating and by the intensity of the specific activity that they reported engaging in most frequently. Women who reported never sweating were classified as having low activity. Women were considered to have moderate physical activity if they reported sweating and engaged in a moderately intense activity [i.e., a metabolic equivalent task (MET) score of <5, as defined by Ainsworth et al. *(20)*]. The participants were classified as having high activity if they reported sweating and engaged in a vigorous activity (i.e., a MET score of ≥5). Within the moderate- and high-intensity categories, we further divided women by the frequency with which they exercised (i.e., <2, 2–4, and >4 times/week).

Beginning in 1986, we were able to evaluate the effect of time spent participating in each of the specific recreational activities queried. We divided women into five categories (<1, 1 to <2, 2 to <4, 4 to <7, and ≥7 hours/week) based on the amount of time they reported walking each week, and we used data from 1986, 1998, 1992, and 1994 to calculate an averaged updated measure of time spent walking. This procedure was then repeated with each activity and then with all nonwalking activities combined.

In addition, from 1986 onward, we were able to calculate the total number of MET hours of activity for each participant. For this calculation, we first assigned each of the activities queried an intensity score on the basis of the index defined by Ainsworth et al. *(20)*. Intensity scores (in METs) are based on an activity's required energy expenditure, with an MET score of 1.0 equal to the amount of energy expended while sitting quietly. Then, for each participant, we multiplied the number of hours per week she spent at each activity by its respective MET score to calculate MET hours/week of each activity. We then summed the contributions of all activities to calculate each woman's activity level in terms of total MET hours/week. For each woman, this measure took into account the frequency, duration, and intensity of physical activity. Participants were then divided into categories on the basis of their total weekly MET hours (0 to <2.5, 2.5 to <5, 5 to <10, 10 to <20, 20 to <30, and ≥30 hours/week). We used total MET hours in 1986, 1988, 1992, and 1994 to create a cumulative average measure of MET hours/week from 1986 to 1996.

To examine whether changes in physical activity level preceding a diagnosis of ovarian cancer biased results, in one analysis, we excluded cases diagnosed in the first 2 years after each report of activity level. In addition, we used analyses lagged by 4, 8, and 12 years to evaluate the association between physical activity in the more distant past and cancer risk. For example, in the analysis lagged by 12 years, physical activity in 1980 was used to predict cancer risk from 1992 to 1994.

We performed several subanalyses to determine whether associations between physical activity and ovarian cancer were limited to particular subtypes of ovarian cancer or to subgroups of our population. We evaluated the relationship of physical activity to serous, mucinous, and endometrioid subtypes individually and the relationship to all nonmucinous tumors and all invasive tumors.

To determine whether the relationship between physical activity and ovarian cancer varied by levels of other risk factors, we stratified our data by age (<50 and ≥50 years), menopausal status (premenopausal and postmenopausal), postmenopausal hormone use (in postmenopausal women only: ever use and never use), BMI (<25 and ≥25 kg/m^2), oral contraceptive use (ever use and never use), smoking status (never, current, and past), and regularity of menstrual cycles in 1982 (very or usually regular [defined as within 2 days of expected] and very or usually irregular). We further examined the influence of the timing of activity in relation to menopause. For each interval, we used current menopausal status to classify the activity reported by each woman as premenopausal or postmenopausal. We then compared the risk of ovarian cancer associated with activity (hours/week) reported by premenopausal and postmenopausal women in the subsequent interval.

Incidence rates for each category of physical activity were calculated by dividing the number of incident cases by the person-years in each category. Relative risks (RRs) compared the incidence rate of ovarian cancer in the second through highest category of each physical activity variable with that in the lowest category (referent) by use of pooled logistic regression analysis with 2-year increments *(21)*. We calculated 95% CI and used the two-sided Mantel extension test for trend to evaluate the presence of a linear trend in the RRs across categories. In the test for trend analyses, the median value for each exposure category was modeled as a continuous variable in the multivariable regression, with one exception; for analyses of activity intensity and frequency based on data from 1980, we used the category number as a continuous variable in the regression model (*see* Table 2). All statistical tests were two-sided.

We evaluated the potential confounding effects of other ovarian cancer risk factors, including age, current BMI, BMI at age 18 years, height, smoking status, parity, age at menarche, tubal ligation, hysterectomy, oral contraceptive use and duration, age at menarche, menstrual cycle regularity, age at menopause, postmenopausal hormone use and duration, talc use, and intake of total calories, lactose, caffeine, and alcohol. Variables were included in multivariable regression models if they were found to alter the RR for the physical activity–ovarian cancer relationship by 10% or more when compared with age-adjusted RRs or if they were determined to be statistically significant predictors of ovarian cancer independent of physical activity level. The final model included age, parity, age at menarche, tubal ligation, oral contraceptive use and duration, menopausal status, and postmenopausal hormone use and duration. Despite their association with activity level, current BMI and BMI at age 18 years did not confound the exposure–disease relationship or predict overall ovarian cancer incidence and, therefore, were not included in multivariable regression models.

Results

From 1980 to 1996 (a 16-year follow-up), more than 1.2 million person-years were accrued by the 92 825 cohort members who provided information on their participation in physical activities (97.8% of total possible person-years of observation), and 377 cases of epithelial ovarian cancer were confirmed.

Baseline characteristics of the NHS cohort according to physical activity level in 1980 are shown in Table 1. Women reporting more frequent exercise were slightly more likely to be users of oral contraceptives or to have had a tubal ligation than less active women. Inactive women were more likely to be current smokers, to be obese (i.e., BMI ≥30 kg/m^2), and to consume less alcohol than more active women. Other characteristics were similar across categories of physical activity.

The total duration of physical activity (hours/week) was not substantially associated with risk of ovarian cancer (Table 2). Women in the highest category of activity (i.e., ≥7 hours/week) experienced a statistically nonsignificant decrease in cancer risk compared with the least active women (RR = 0.80; 95% CI = 0.49 to 1.32; P_{trend} = .59). Results for hours of total activity measured in 1980 only and for the most recent level of physical activity (from 1980 to 1996; results not shown) also suggested no reduction in risk with greater activity.

Next we examined the follow-up period from 1986 to 1996, for which we had more detailed data on type of activity (Table 2). Women who engaged in activity 7 hours or more per week had a statistically nonsignificant 26% increase in ovarian cancer risk (RR = 1.26; 95% CI = 0.80 to 1.97), with no evidence of a dose–response relationship (P_{trend} = .59). Risk was also elevated for women in the highest category of activity measured in 1986 only (RR = 1.64; 95% CI = 1.05 to 2.58; P_{trend} = .13).

We evaluated the association of consistent participation in physical activity on cancer risk by using the average of activities in 1980, 1982, and 1986 to predict cancer incidence from 1986 onward (Table 2). Women whose average activity

Table 1. Baseline characteristics of the study population according to category of physical activity in 1980*

Characteristic	Category of physical activity, physical activity in h/wk				
	<1	1 to <2	2 to <4	4 to <7	≥7
No. of participants	18 950	21 411	12 327	12 365	9176
Mean age, y	46.4	46.2	46.3	46.2	46.1
Mean daily intake					
Total fat, g/day	68.2	69.9	69.3	68.0	66.6
Total calories, kcal/day	1539	1576	1594	1572	1561
Alcohol, g/day	6.3	6.1	5.7	6.5	8.3
Lactose, g/day	12.8	13.2	13.9	13.9	13.8
Mean parity, No. of childbirths	3.0	3.1	3.1	2.9	3.0
Mean age at birth of first child, y†	24.6	24.5	24.4	24.3	24.3
Mean age at menarche, y	12.5	12.5	12.5	12.6	12.6
Mean current body mass index (BMI), kg/m^2	24.6	24.5	24.4	23.8	22.9
BMI ≥30 kg/m^2, %	14.4	13.0	11.9	8.3	4.1
Oral contraceptive (OC) ever use, %	48.9	50.0	47.3	51.8	53.9
Mean duration of OC use, mo‡	49.3	49.9	49.7	51.1	52.7
Premenopausal, %	65.5	66.8	66.1	66.2	67.3
Current smokers, %	31.8	29.9	31.1	26.1	22.1
Past smokers, %	25.4	25.7	25.1	29.3	35.0
Tubal ligation, %	16.2	16.0	16.5	16.8	17.6
Simple hysterectomy, %	10.6	11.0	11.3	11.0	10.7
Postmenopausal hormone (PMH) ever use, %	30.7	30.1	30.4	30.4	30.2
Mean duration of PMH use, mo§	6.2	6.9	6.7	7.7	7.0

*Age was standardized to the distribution of the entire cohort by use of the direct method. Numbers of participants do not sum to 92 825 because of missing physical activity data in 1980.

†Among parous women.

‡Among OC users.

§Among PMH users.

Table 2. Analysis of the association of ovarian cancer and total physical activity (by categories of hours/week) from 1980 to 1996 and from 1986 to 1996, and average hours of physical activity per week from 1980 to 1986 (follow-up 1986–1996)

Physical activity level	No. of cases*	Age-adjusted RR (95% CI)†	MV RR (95% CI)‡
1980–1996 (cumulative average), h/wk			
<1	85	1.00 (referent)	1.00 (referent)
1 to <2	83	0.76 (0.56 to 1.03)	0.80 (0.59 to 1.08)
2 to <4	101	0.80 (0.60 to 1.06)	0.86 (0.65 to 1.15)
4 to <7	89	1.01 (0.75 to 1.36)	1.10 (0.82 to 1.49)
≥7	19	0.72 (0.44 to 1.19)	0.80 (0.49 to 1.32)
			P = .59§
1980 (only), h/wk			
<1	97	1.00 (referent)	1.00 (referent)
1 to <2	76	0.74 (0.54 to 1.00)	0.75 (0.56 to 1.02)
2 to <4	52	0.84 (0.60 to 1.18)	0.86 (0.61 to 1.20)
4 to <7	59	0.99 (0.72 to 1.38)	1.01 (0.73 to 1.40)
≥7	35	0.85 (0.57 to 1.28)	0.92 (0.62 to 1.36)
			P = .74§
1986–1996 (cumulative average), h/wk			
<1	60	1.00 (referent)	1.00 (referent)
1 to <2	47	1.08 (0.73 to 1.58)	1.13 (0.77 to 1.65)
2 to <4	53	1.06 (0.73 to 1.53)	1.10 (0.76 to 1.60)
4 to <7	33	0.93 (0.60 to 1.42)	0.98 (0.64 to 1.50)
≥7	28	1.24 (0.79 to 1.93)	1.26 (0.80 to 1.97)
			P = .59§
1986 (only), h/wk			
<1	52	1.00 (referent)	1.00 (referent)
1 to <2	44	1.40 (0.94 to 2.10)	1.41 (0.94 to 2.11)
2 to <4	40	1.21 (0.80 to 1.82)	1.23 (0.81 to 1.85)
4 to <7	23	1.11 (0.68 to 1.81)	1.12 (0.69 to 1.84)
≥7	30	1.63 (1.04 to 2.55)	1.64 (1.05 to 2.58)
			P = .13§
Average h/wk, 1980–1986‖			
<1	50	1.00 (referent)	1.00 (referent)
1 to <2	44	0.72 (0.48 to 1.08)	0.72 (0.48 to 1.08)
2 to <4	74	0.98 (0.68 to 1.40)	1.00 (0.70 to 1.43)
4 to <7	45	0.94 (0.63 to 1.41)	0.97 (0.64 to 1.45)
≥7	15	1.42 (0.80 to 2.54)	1.46 (0.82 to 2.60)
			P = .18§

*Numbers of cases may not sum to 377 because of missing data.

†RR = relative risk.

‡MV RR adjusted for age (<50, 50–54, 55–59, 60–64, and ≥65 years), parity (no, one or two, three or four, and ≥five births), age at menarche (<12, 12, 13, 14, and ≥15 years), oral contraceptive use and duration (never use, use <3 years, use 3–5 years, and use >5 years), menopausal status/postmenopausal hormone use (premenopausal/never use, current use <5 years, current use ≥5 years, past use <5 years, and past use ≥5 years), tubal ligation (yes and no), smoking status (never, past, current and <15 cigarettes/day, and current and ≥15 cigarettes/day). MV = multivariable; CI = confidence interval.

§Two-sided P_{trend} across categories, calculated by use of the median of each category as a continuous variable in the MV regression model.

‖Follow-up from 1986 to 1996.

level was 7 hours or more per week had an RR of 1.46 (95% CI = 0.82 to 2.60) compared with those reporting less than 1 hour/week.

We then categorized participants to compare the effects of frequency of activity and intensity of activity, as reported in 1980 (Table 3). Women participating in moderate activity less than two times per week had an RR of 0.57 (95% CI = 0.36 to 0.92) compared with women reporting low activity. Reductions were not found for more frequent or intense exercise; risk appeared modestly elevated in women who exercised vigorously three or more times per week.

We further addressed the influence of activity frequency and intensity by use of data on MET hours. Compared with inactive women, participants in the second through sixth categories of MET hours/week experienced a greater risk of ovarian cancer (Table 4), although we saw no evidence of a linear trend across categories (P_{trend} = .52). Women reporting 20 to less than 30 MET hours/week had an RR of 1.84 (95% CI = 1.12 to 3.02) compared with those reporting less than 2.5 MET hours/week, while those in the highest category (≥30 MET hours/week) had an RR of 1.27 (95% CI = 0.75 to 2.14).

We compared the risk of ovarian cancer associated specifically with walking, as well as with frequent participation in all other activities. We saw no increase in risk among women who walked frequently compared with those who walked infrequently, even after controlling for time spent in other nonwalking activities (RR for walking ≥7 versus <1 hour/week = 1.17; 95% CI = 0.74 to 1.86). Similarly, frequent participation in nonwalking activities was not associated with an increase in risk (RR for nonwalking ≥7 versus <1 hour/week = 1.10; 95% CI = 0.62 to 1.95).

We evaluated the association between duration of physical activity and three subtypes of ovarian cancer. The RRs for serous tumors tended to be slightly higher than those for the main analyses (e.g., RR for ≥7 versus < 1 hour/week of total activity = 1.11; 95% CI = 0.61 to 2.02). Although we had limited statistical power to assess the effect of physical activity on mucinous and endometrioid tumors, results for these subtypes also did not differ substantially from the main analysis (results not shown). Furthermore, we observed little difference in the associations for mucinous tumors compared with all nonmucinous tumors (results not shown), and results from a subanalysis limited to invasive tumors did not differ from those including all cases (results not shown).

To determine whether results were affected by participants having reduced their activity level preceding a diagnosis of ovarian cancer, we evaluated the association between the cumulative average of physical activity (from 1980 to 1996), excluding cases diagnosed during the first 2 years after each report of activity level. Results did not differ substantially from those presented in Table 2; the RR for participants reporting 7 hours or more per week was 1.08 (95% CI = 0.68 to 1.72). Similarly, results lagged by 4 and 8 years did not differ from the main analysis (results not shown). Although power was greatly limited by the small number of cases diagnosed from 1992 to 1996 (n = 91), physical activity level 12 years earlier was associated with a modest, statistically nonsignificant reduction in risk. Compared with women who reported activity of less than 1 hour/week, the RRs

Table 3. Analysis of the association of ovarian cancer and light-, moderate-, and high-intensity physical activity in 1980 (follow-up from 1980 to 1996)

Physical activity level, intensity versus frequency—1980*	No. of cases†	Age-adjusted RR‡ (95% CI)	MV RR (95% CI)§
Low intensity	192	1.00 (referent)	1.00 (referent)‖
Moderate intensity, ≤2 times/wk	19	0.56 (0.35 to 0.90)	0.57 (0.36 to 0.92)
Moderate intensity, 3–4 times/wk	26	1.31 (0.87 to 1.97)	1.35 (0.89 to 2.03)
Moderate intensity, >4 times/wk	17	0.93 (0.57 to 1.53)	0.94 (0.57 to 1.54)
Vigorous intensity, ≤2 times/wk	23	0.98 (0.64 to 1.51)	1.05 (0.68 to 1.63)
Vigorous intensity, 3–4 times/wk	26	1.48 (0.98 to 2.24)	1.58 (1.05 to 2.38)
Vigorous intensity, >4 times/wk	16	1.38 (0.83 to 2.31)	1.48 (0.89 to 2.48)

*Intensity categories are defined as follows: low = activity not leading to sweating; moderate = activity of less than five metabolic equivalent tasks (METs) leading to sweating; high = activity of five METs or more leading to sweating.

†Numbers of cases may not sum to 377 because of missing data.

‡RR = relative risk.

§MV RR adjusted for age, parity, age at menarche, menopausal status/postmenopausal hormone use, tubal ligation, and smoking status. Categories for covariates are shown in Table 2. MV = multivariable; CI = confidence interval.

‖Each category compared with the referent of low activity. Two-sided P_{trend} across categories was calculated separately for low + three categories of moderate activity (P_{trend} = .93) and for low + three categories of vigorous activity (P_{trend} = .03).

Table 4. Analysis of the association of oavarian cancer and MET*,† hours/week from 1986 to 1996 and in 1986 only

Physical activity level	No. of cases‡	Age-adjusted RR (95% CI)	MV RR (95% CI)§
MET (cumulative average), h/wk			
0 to <2.5	28	1.00 (referent)	1.00 (referent)
2.5 to <5	34	1.37 (0.83 to 2.25)	1.42 (0.86 to 2.34)
5 to <10	42	1.24 (0.77 to 2.00)	1.34 (0.83 to 2.17)
10 to <20	50	1.22 (0.77 to 1.94)	1.32 (0.83 to 2.10)
20 to <30	37	1.71 (1.05 to 2.78)	1.84 (1.12 to 3.02)
≥30	29	1.19 (0.71 to 1.99)	1.27 (0.75 to 2.14)
			P = .52‖
MET (1986 only), h/wk			
0 to <2.5	38	1.00 (referent)	1.00 (referent)
2.5 to <5	32	1.18 (0.74 to 1.89)	1.11 (0.75 to 1.66)
5 to <10	36	1.38 (0.87 to 2.17)	1.30 (0.89 to 1.90)
10 to <20	32	1.07 (0.67 to 1.71)	1.02 (0.68 to 1.51)
20 to <30	25	1.47 (0.89 to 2.44)	1.41 (0.90 to 2.18)
≥30	26	1.25 (0.76 to 2.05)	1.16 (0.75 to 1.80)
			P = .48‖

*MET = metabolic equivalent task; MV = multivariable; RR = relative risk; CI = confidence interval.

†Metabolic equivalents, as defined by Ainsworth et al. *(20)*.

‡Numbers of cases and person-years may not sum to totals because of missing data.

§MV RR adjusted for age, parity, age at menarche, menopausal status/postmenopausal hormone use, and tubal ligation and smoking status. Categories for covariates are shown in Table 2.

‖Two-sided P_{trend} over quintiles, calculated by using the median of each quintile as a continuous variable in the MV model.

for 1 to less than 2 hours/week, 2 to less than 4 hours/week, 4 to less than 7 hours/week, and greater than or equal to 7 hours/week were 0.91, 0.63, 0.53, and 0.72, respectively (P_{trend} = .14).

Finally, we evaluated whether the association between physical activity and ovarian cancer varied according to level of other ovarian cancer risk factors, particularly, current BMI and menopausal status. Results suggested that the observed increase in risk may be greatest in leaner women (i.e., BMI <25 kg/m^2), but these interactions were not statistically significant. We did not observe any difference in the association by menopausal status, age, oral contraceptive use, postmenopausal hormone use, regularity of menstrual cycles, or smoking status (results not shown). Furthermore, we found no difference in the risk of ovarian cancer for activity taking place before menopause compared with after menopause (results not shown).

Discussion

We did not find evidence of an inverse association between recreational physical activity and risk of ovarian cancer. The analysis of cumulative averaged MET hours/week (from 1986 to 1996), which probably provides the best overall measure of activity during the follow-up period, suggests that women who frequently engage in vigorous activity may experience a modest increase in ovarian cancer risk compared with inactive women. We did not find that walking, the activity most commonly reported by our participants, was associated with an increase in risk.

Physical activity has been hypothesized to reduce the incidence of ovarian cancer through one of several mechanisms. Intense physical activity can disrupt regular ovarian cycling, leading to luteal phase inadequacy, anovulation, and amenorrhea *(1)*. Vigorous exercise may thus alter the risk of ovarian cancer by reducing the frequency of ovulation and damage to epithelial tissue *(22)*. However, suppression of ovulation may only be achieved with extreme activity levels. Other data suggest that regular vigorous activity of the intensity level practiced by most women may instead increase ovulation frequency. Rich-Edwards et al. *(23)* found that frequent vigorous but not moderate activity was inversely associated with ovulatory infertility. A positive relationship between physical activity and ovulation frequency could potentially explain the modest increase in risk of ovarian cancer that we observed in women engaging in vigorous activity.

Physical activity may decrease circulating levels of endogenous estrogens *(2,24,25)*, exposure to which has been proposed as a contributing factor to the development of ovarian cancer *(26)*. Activity can also decrease body fat, thereby reducing the extragonadal production of estrogen, a major source of estrogen after menopause *(3)*. However, activity may also plausibly increase ovarian cancer risk through hormone-related pathways. By reducing estrogens, exercise may increase pituitary production of gonadotropins as part of a negative-feedback relationship *(26)*; exposure to high levels of gonadotropins has been suggested as a mechanism for ovarian cancer development *(27)*. An alternative hypothesis, proposed by Risch *(28)*, suggests that even moderate amounts of activity may increase risk

of ovarian cancer by reducing progesterone levels and by increasing androgen levels.

Several studies *(4–11)* have evaluated the association between physical activity and ovarian cancer, with inconsistent results. Such inconsistencies may result from variation in the definition of physical activity between studies, misclassification of activity level, or bias in these studies. Of the seven case–control studies of activity and ovarian cancer *(4–8)*, five observed a reduction in cancer risk with frequent activity ranging in magnitude from 25% to 50%. Two other studies *(9,10)* reported a positive association. It is possible that selection and recall biases may have influenced the results of these case–control studies. Because ovarian cancer is often diagnosed at an advanced stage and survival is generally poor *(29)*, it may be difficult to attain high participation rates. Case recall of physical activity may be subject to bias. Such biases are greatly reduced by prospective exposure measurement.

The only other prospective cohort study *(11)* to address this relationship reported a statistically significant positive association between activity and ovarian cancer. In the Iowa Women's Health Study, women in the highest category of a physical activity index had a twofold increase in risk compared with women in the lowest category. Risk increased linearly with frequency of both moderate and vigorous physical activity, with exercise of greater intensity conferring greater risk.

The extent to which occupational physical activity and physically demanding tasks like child rearing may confound studies of recreational exercise remains unclear. Recreational and nonrecreational activity may be inversely associated if women who hold physically demanding jobs have less energy for recreation and if those with more sedentary jobs are more likely to participate in leisure-time activity. We were able to include some information relating to occupational activity by including weekly time spent walking and stair climbing on questionnaires from 1986 onward and including these activities in the calculation of MET hours. However, the inability to comprehensively measure all contributions to activity is a limitation of any study of recreational activity.

Since the etiologic period of ovarian cancer has not yet been identified, it is unclear when physical activity may have the greatest impact on risk. Because of its effect on ovulation, premenopausal physical activity may affect risk differently from that after menopause. We saw no substantial difference in cancer risk among premenopausal and postmenopausal women or among women who exercised before as opposed to after menopause. Vigorous activity in young adulthood may also modify risk by delaying the onset of menarche and by increasing the frequency of anovulation in adolescence *(1)*. Although we were unable to specifically address the effect of adolescent activity, we attempted to evaluate the effect of disease latency with a lagged analysis. Although we were severely limited by low power, our results suggested the possibility of an inverse association between cancer risk and participation in physical activity 12 years earlier.

In conclusion, we did not find evidence of an inverse association between recreational physical activity and risk of ovarian cancer. Our results concerning the possibility of a modest increase in risk with frequent vigorous activity suggest that additional research is needed to explore in greater depth the relationship between ovarian cancer and the frequency and intensity of physical activity.

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NOTES

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対象の内訳			地 域		研究の種類	
	ヒト	動物		欧米		縦断研究
対象	一般健常者	空白		( )		コホート研究
性別	女性	( )		( )		( )
年齢	30-55歳(46歳)	[redacted]		( )		前向きコホート
対象数	10000以上			( )		( )
調査の方法	質問紙	( )				
アウトカム						
予 防	なし	なし	ガン予防	なし		( )
維持・改善	な し	な し	な し	なし	(	( )

図 表

Table 4. Analysis of the association of ovarian cancer and MET*,† hours/week from 1986 to 1996 and in 1986 only

Physical activity level	No. of cases‡	Age-adjusted RR (95% CI)	MV RR (95% CI)§
MET (cumulative average), h/wk			
0 to <2.5	28	1.00 (referent)	1.00 (referent)
2.5 to <5	34	1.37 (0.83 to 2.25)	1.42 (0.86 to 2.34)
5 to <10	42	1.24 (0.77 to 2.00)	1.34 (0.83 to 2.17)
10 to <20	50	1.22 (0.77 to 1.94)	1.32 (0.83 to 2.10)
20 to <30	37	1.71 (1.05 to 2.78)	1.84 (1.12 to 3.02)
≥30	29	1.19 (0.71 to 1.99)	1.27 (0.75 to 2.14)
			P = .52‖
MET (1986 only), h/wk			
0 to <2.5	38	1.00 (referent)	1.00 (referent)
2.5 to <5	32	1.18 (0.74 to 1.89)	1.11 (0.75 to 1.66)
5 to <10	36	1.38 (0.87 to 2.17)	1.30 (0.89 to 1.90)
10 to <20	32	1.07 (0.67 to 1.71)	1.02 (0.68 to 1.51)
20 to <30	25	1.47 (0.89 to 2.44)	1.41 (0.90 to 2.18)
≥30	26	1.25 (0.76 to 2.05)	1.16 (0.75 to 1.80)
			P = .48‖

*MET = metabolic equivalent task; MV = multivariable; RR = relative risk; CI = confidence interval.
†Metabolic equivalents, as defined by Ainsworth et al. (20).
‡Numbers of cases and person-years may not sum to totals because of missing data.
§MV RR adjusted for age, parity, age at menarche, menopausal status/postmenopausal hormone use, and tubal ligation and smoking status. Categories for covariates are shown in Table 2.
‖Two-sided P_{trend} over quintiles, calculated by using the median of each quintile as a continuous variable in the MV model.

図表掲載箇所	P946, Table4
概 要 (800字まで)	本研究はthe Nurses' Health Study cohortに参加している30-55歳の女性92,825名を対象に16年間の追跡調査を行い、余暇身体活動と卵巣がんとの関係について検討を行っている。余暇身体活動量については、ジョギング、ランニング、自転車、水泳、テニス、スカッシュ、ラケットボール、エアロビックな活動、ウォーキング、アウトドアハイキングにおける週当たりの時間が聞き取られ、さらに通常の歩行ペースや階段の昇る段数も聞き取った。強度はMETsに換算され、余暇身体活動は0-2.49メッツ・時/週、2.5-4.99メッツ・時/週、5-9.99メッツ・時/週、10-19.99メッツ・時/週、20-29.99メッツ・時/週、30メッツ・時/週以上に分類された。卵巣がん発症のリスクは、それぞれ、1.0、1.11(0.75-1.66)、1.3(0.89-1.9)、1.02(0.68-1.51)、1.41(0.9-2.18)、1.16(0.75-1.8)であり、有意なリスク低下は認められなかった。一方、10年間の身体活動の累積平均と卵巣がん発症との関連について検討を行ったところ、週0-2.5メッツ・時と比較して、週20-30メッツ・時の人では、そのリスクが1.84(1.12-3.02)であった。しかしながら、週30メッツ・時行っている人では、1.27(0.75-2.14)で有意なリスク増加は認められなかった。
結 論 (200字まで)	卵巣がんの発症には、身体活動量は関与していなかった。一部、高強度の身体活動や、累積身体活動が影響している可能性も見られたが、これについては、更なる検討が必要である。
エキスパートによるコメント (200字まで)	女性に特有なガンに関して、身体活動による予防との関連が検討されているが、本研究の結果からは卵巣がんに対しては、身体活動による予防効果は認められていない。今後は身体活動強度や総身体活動量についての検討や他のリスクファクターとの相互作用についての検討が必要であろう。

担当者　　村上晴香

Prospective Study of Physical Activity and Depressive Symptoms in Middle-Aged Women

Wendy J. Brown, PhD, Jessica H. Ford, MSc, Nicola W. Burton, MPsych(Clinical), Alison L. Marshall, PhD, Annette J. Dobson, PhD

Background: Although many studies support an inverse association between physical activity (PA) and depressive symptoms, prospective relationships between these variables have been confounded by pre-existing psychological and physical health problems.

Methods: This study examined the dose–response relationships between self-reported PA and depressive symptoms, using cross-sectional and prospective data from a population-based cohort of middle-aged women who participated in the Australian Longitudinal Study on Women's Health (ALSWH) between 1996 and 2001. Participants completed three mailed surveys (S1, 1996; S2, 1998; S3, 2001), which included questions about time spent in walking, moderate- and vigorous-intensity PA, and measures of psychological health (Center for Epidemiologic Studies Depression scale [CESD-10], and Mental health [MH] subscale of the Short Form 36 survey). Relationships between previous (S1, S2), current (S3), and habitual (S1, S2, S3) PA and "depressive symptoms" were examined, adjusting for sociodemographic and health-related variables (n=9207).

Results: Mean CESD-10 scores decreased, and MH scores increased with increasing levels of previous, current, and habitual activity. Odds ratios for CESD-10 scores ≥10 or MH scores ≤52 at S3 were 30% to 40% lower among women who reported the equivalent of ≥60 minutes of moderate-intensity PA per week, compared with those who reported less PA than this. Women who were in the lowest PA category at S1, but who subsequently reported ≥240 metabolic equivalent minutes (MET.mins) per week had lower odds of CESD-10 scores of ≥10 or MH scores ≤52 at S3 than those who remained in the very low PA category.

Conclusions: These data suggest that there is a clear relationship between increasing PA and decreasing depressive symptoms in middle-aged women, independent of pre-existing physical and psychological health.

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Introduction

The U.S. Surgeon General's report on physical activity (PA) and health cited four prospective longitudinal studies that examined the relationships between PA and depressive symptoms in the general population.[1] In the First National Health and Nutritional Examination Survey (NHANES I) study, men and women who reported little or no PA and few depressive symptoms at baseline were almost twice as likely to report depressive symptoms after 8 years of follow-up.[2] Similar results were found in the Alameda County study in which 1799 men and women were followed for 9 years.[3] The Harvard alumni study followed 21,596 men for 20 years and provided evidence of a dose–response relationship between PA and physician-diagnosed depression.[4] The fourth study however, found no relationship between PA at baseline and psychiatrist-diagnosed depression after 5 years of follow-up, in a cohort of more than 1500 Bavarian men and women.[5]

Since publication of the U.S. Surgeon General's report,[1] several reviews have concluded that there is an inverse association between PA and depressive symptoms.[6–9] From these reviews, it is clear that much of the evidence for this relationship is limited by cross-sectional research designs, small clinical samples, a focus on vigorous-intensity exercise, and inadequate follow-up beyond 12 months. Where cohorts have been followed for >12 months, there are conflicting results. For example, when a cohort of 937 middle-aged physicians was followed for 15 years, there was no relationship between PA and depressive symptoms.[10] In contrast, a study of 1947 older adults who were followed for 5 years found that PA was protective for both prevalent and incident depressive symptoms.[11]

From the School of Human Movement Studies (Brown, Burton, Marshall), and School of Population Health (Ford, Dobson), The University of Queensland, Brisbane, Queensland, Australia

Address correspondence and reprint requests to: Wendy Brown, PhD, School of Human Movement Studies, The University of Queensland, St Lucia QLD 4072, Australia. E-mail: wbrown@hms.uq.edu.au.

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The confounding role of pre-existing depressive symptoms and ongoing chronic health problems has also been raised as a potential limitation of this evidence. For example, in 1996, Mobily et al.[12] found that older men and women who walked daily were less likely to report depressive symptoms after 3 years, but that the association only existed among those participants who reported a high number of depressive symptoms at baseline. When clinically depressed or physically limited participants were excluded from their cohort of 944 older adults, Kritz-Silverstein et al.[13] also found no statistically significant longitudinal effects of PA on depression. In contrast, other studies have reported that the protective effect of PA on depressive symptoms persists after adjustment for chronic conditions,[11] as well as for a range of other behavioral, social, and demographic characteristics.[2,3,11,14]

On balance, most of these studies, which have been conducted primarily with mixed gender cohorts, suggest a protective role for PA in the primary prevention of depressive symptoms. If this is true, quantification of the amount of PA needed to prevent or ameliorate depressive symptoms (i.e., exploration of dose–response relationships) would assist with the development of public health recommendations and health promotion messages. The Harvard Alumni researchers[4] have reported that men who expend 1000 to 2499 or ≥2500 kcal per week are 17% and 28%, respectively, less likely to develop clinically diagnosed depression than men who expend <1000 kcals per week. These data suggest that moderate to high levels of weekly energy expenditure in men are associated with decreased likelihood of depression.

The aim of the present study was to explore the dose–response relationships between PA and depressive symptoms through analyses of cross-sectional and prospective data from a large population-based cohort of middle-aged women participating in the Australian Longitudinal Study on Women's Health (ALSWH). The large size and comprehensive nature of the ALSWH enabled us to control for a wide range of potential confounding variables to an extent that has not been possible in previous studies. The longitudinal design helps to elucidate the directions of causation.

Methods

Overview of the Australian Longitudinal Study on Women's Health

The ALSWH is a longitudinal study of factors affecting the health and well-being of three national cohorts of women aged 18 to 23 years (young), 45 to 50 years (middle age), and 70 to 75 years (older) at the time of the baseline survey (S1) in 1996. The study is designed to continue for 20 years with self-report (survey) data collection at 2- to 3-year intervals. It is providing longitudinal data on health outcomes for about 40,000 women living in all States and Territories of Australia. (Details of the study can be found at www.sph.uq.edu.au/alswh.) In this paper, the middle-aged cohort is the only focus because (1) the incidence of depression is highest in middle-aged women[15]; (2) there was a high rate of missing data in the Center for Epidemiologic Studies Depression Scale (CESD-10) responses from the older cohort; and (3) the life circumstances and hence potential covariates and confounders differ substantially between the cohorts.

Study Sample

Women were selected randomly from the national Medicare health insurance database (which includes all permanent residents of Australia) with intentional over-representation of women living in rural and remote areas.[16] Written informed consent was obtained from all participants. In 1996 (S1), 14,099 middle-aged women returned the survey (response rate 54%), with 12,338 (88%) and 11,202 (79%) of these completing follow-up surveys in 1998 (S2) and 2001 (S3), respectively. A total of 10,400 women completed all three surveys. The original respondents were reasonably representative of the general population of Australian women in this age group, but there was over-representation of Australian-born, employed, and university-educated women.[16] There were few differences between continuing respondents and those lost to follow-up, with the exception that continuing respondents were slightly more likely to be married, employed, have higher education, and be nonsmokers than those who were lost to follow-up (see www.newcastle.edu.au/centre/wha/InfoData/technical_datareports.html). Further details of the recruitment methods and response rates are described elsewhere.[16]

Of the 10,400 women who completed all three surveys, 1193 were excluded because they had missing data on the main response or exposure variables or because they reported having difficulty walking 100 meters; this left 9207 (89%) women with data for inclusion in these analyses.

The Surveys

Complete details of >300 items in each survey are on the study website (http://www.newcastle.edu.au/centre/wha). Of relevance to this paper are the questions about depressive symptoms, physical activity; sociodemographic characteristics (country of birth, education, marital status, occupation, and area of residence); height and weight; health behaviors (smoking); menopause status; and physical health.

Assessment of depressive symptoms. The 1998 (S2) and 2001 (S3) surveys included the CESD-10, a ten-item self-report scale designed to identify depressive symptoms (experienced in the last week) in the general population. The measurement properties of this instrument have been assessed, with satisfactory test–retest correlations and good predictive accuracy when compared with the full-length 20-item version of the CES-D.[17] Responses are recorded using a four-point Likert scale ranging from rarely (scored 0) to most of the time (scored 3), and summed across the ten items to provide a total CESD-10 score. Respondents who provided data on either nine or ten of the CESD-10 items were included in the analyses, with mean imputation used to replace the one missing item. CESD-10 scores of ≥10 were used to indicate "depressive symptoms."[17]

The Medical Outcomes Study's[18] short-form functioning and well-being profile (SF-36) was included in each of the three surveys to assess general physical and psychological health and well-being over the last 4 weeks. This comprehensive health-related quality of life measure includes 36 items to assess eight different health domains, and has been evaluated as a practical, reliable, and valid instrument for use in postal surveys of the general population.[19] The mental health (MH) subscale is based on five items with responses recorded on a six-point Likert scale, and possible scores ranging from 0 to 100.[18] Because MH scores of ≤52 can be used to indicate the presence of depressive symptoms, and as the results are very similar for CESD-10 scores ≥10 and MH scores ≤52, the term "depressive symptoms" is used for both measures.

Assessment of physical activity. At S1, two questions about PA were used. They asked how many times in a normal week respondents engaged in vigorous exercise (e.g., aerobics, jogging) or less vigorous exercise (e.g., walking, swimming) for ≥20 minutes.[20] Responses were used to derive a PA score based on frequency of participation in "vigorous" (7.5 metabolic equivalents [METs]) and "less vigorous" (4 METs) PA lasting ≥20 minutes:

$$\text{PA score} = \Sigma\{\text{frequency} * 20 * 4(\text{less vigorous}) + \text{frequency} * 20 * 7.5(\text{vigorous})\}.$$

The items are known to have acceptable test–retest reliability.[22] Very few women reported undertaking no activity. PA scores at S1 were categorized as "very low" (PA score of <200; no reported PA or one session per week); "low" (PA score of 200 to <400; two to three sessions of moderate or vigorous PA per week); "moderate" (PA score of 400 to <560; e.g., five sessions per week of moderate-intensity PA or three sessions per week of vigorous PA); or "high" (PA score of ≥560; e.g., daily moderate-intensity PA or four or more sessions of vigorous PA).

At S2 and S3, PA was assessed using questions developed for the evaluation of the national Active Australia campaign in 1997, and for national monitoring of PA in Australia.[23] The questions ask about the frequency and total duration of walking (for recreation or transport), and of vigorous (e.g., aerobics, jogging) and moderate-intensity activity (e.g., swimming, golf) in the last week. These items have been shown to have acceptable reliability and validity for population measurement of PA.[24,25] A PA score (MET minutes) was derived from reported duration of time spent in each form of PA during the last week:[21]

$$\text{MET mins} = \Sigma\{(\text{walking minutes} * 3.5) + (\text{moderate minutes} * 4) + (\text{vigorous minutes} * 7.5)\}.$$

PA scores at S2 and S3 were categorized as "very low" (PA score of <240, commensurate with <60 minutes of moderate-intensity PA per week); "low" (PA score of 240 to <600 or 60 to <150 minutes/week); "moderate" (PA score of 600 to <1200, or 150 to <300 minutes/week); or "high" (PA score of ≥1200 or >300 minutes/week). The cut-point for the "moderate" category was based on current Australian and U.S. guidelines for PA, indicating compliance with current national guidelines of 150 minutes of moderate-intensity PA per week.[26]

For this paper, three different PA scores were created: (1) "current" PA (S3 data only); (2) "previous" PA (sum of PA scores at S1 and S2); and (3) "habitual" PA (sum of PA scores at S1, S2, and S3). Previous PA scores were categorized as "very low" (<440; e.g., PA scores at S1 of <200 and at S2 of <240); "low" (440 to <1000; e.g., PA scores at S1 of 200 to <400 and at S2 of 240 to <600); "moderate" (1000 to <1760; e.g., PA scores at S1 of 400 to <560, and at S2 of 600 to <1200); or "high" (≥1760; PA scores at S1 of ≥560 and at S2 of ≥1200). With the addition of S3 data, habitual PA scores were categorized as "very low" (<680); "low" (680 to <1600); "moderate" (1600 to <2960); or "high" (≥2960).

For women in the "very low" PA category at S1, a transitional variable was derived to reflect changes in PA between S1 and S3: PA score of <200 at S1 and <240 at S3; PA score of <200 at S1 and 240 to <600 at S3; and PA score of <200 at S1 and ≥600 at S3.

Covariates. To assess the robustness of the association between PA and the CESD-10 and MH scores, the following sociodemographic and health-related variables were included in the analyses as potentially confounding/mediating variables: country of birth (S1); highest educational qualification (S1); marital status (S3); occupation (S3); area of residence (S3); smoking status (S3); body mass index (BMI) (S3); menopause status (S3); MH score of ≤52 at S1; and diagnosis of symptoms, chronic conditions or procedures (stiff and painful joints, back pain, arthritis, hypertension, diabetes, cancer, heart disease, osteoporosis, thrombosis, low iron, bronchitis, prolapse, cholecystectomy) at S3. All responses were categorized as shown in Table 1. An additional category was created for missing responses to the questions about occupation, BMI (when either height or weight was missing), and PA.

Statistical Analyses

Chi-square tests were used to examine associations between the sociodemographic and health characteristics, and depressive symptoms and very low PA. The observed-margins least-squares mean option in the SAS general linear model procedure was used to estimate crude and adjusted means, and 95% confidence intervals using multiple regression separately for CESD-10 and MH scores at S3 for each PA variable (current, previous, and habitual). Crude and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were then calculated for CESD-10 score of ≥10 and MH score of ≤52 for each PA variable, using the logistic procedure in SAS. Means and ORs were adjusted for country of birth, highest educational qualification, marital status, occupation, area of residence, current smoking status, body mass index, menopause status, MH score of ≤52 at S1, and chronic conditions at S3.

Finally, for women who were in the "very low" PA category at S1, ORs and 95% CIs for a CESD-10 score of ≥10 and MH score of ≤52 at S3 were calculated to compare depressive symptoms in women with "low" or "moderate/high" PA at S3 with those who remained in the "very low" category. Once again, data were adjusted for the potential confounders that were included in the previous analyses.

All statistical analyses were performed using SAS, version 8 (SAS Institute Inc., Cary NC, 1999) in 2004 and 2005.

Results

Sociodemographic characteristics of the women included in the analysis sample (n=9207) and for women who were categorized as having "depressive

Table 1. Selected sociodemographic and health characteristics of women in analysis sample, and percentages of women in each category who had CESD-10 score ≥10 at S3, MH ≤52 at S3, or "very low" PA at S1[a]

Overall prevalence	Analysis sample n=9207	CESD≥10 (S3) n=1905 (20.7%)	MH≤52 (S3) n=1215 (13.2%)	"Very low" PA (S1) n=2407 (26.3%)
S1 Country of birth		**0.0058****	0.25	**0.0022****
Australia	78.1	20.4	12.8	26.1
Other English speaking background	13.7	20.8	13.2	24.9
Europe	5.5	23.7	18.0	27.0
Asia	1.9	22.9	13.1	39.1
Other	0.7	27.0	20.6	30.2
S1 Education		**<0.0001*****	**<0.0001*****	**<0.0001*****
No formal qualification/Year 10	47.2	23.1	15.1	29.0
Year 12	17.0	18.6	12.2	24.9
Trade/apprentice	20.4	19.1	11.7	23.2
University degree	15.4	17.8	10.4	23.4
S3 Marital status		**<0.0001*****	**<0.0001*****	0.40
Separated/single/divorced/widowed	23.6	28.1	17.7	27.0
Married/de facto	76.4	18.4	11.8	26.1
S3 Occupation		**<0.0001*****	**<0.0001*****	**0.0031****
No paid job	23.1	26.1	18.0	26.4
Sales/transport	11.6	20.1	13.8	25.4
Trade and advanced clerical	24.2	19.7	12.1	29.3
Manager/professional	33.9	16.9	9.8	24.7
Missing	7.2	25.7	16.7	24.6
S3 Area of residence		0.22	**0.0170***	**0.0005****
Urban	37.5	20.9	14.1	28.3
Large rural	14.1	23.7	14.6	24.8
Small rural	43.3	19.8	12.2	25.6
Remote	5.1	18.6	11.2	20.6
Current smoking status		**<0.0001*****	**<0.0001*****	**<0.0001*****
Smoking	13.7	27.8	19.8	34.6
Not smoking	86.3	19.6	12.2	25.0
S3 BMI		**0.0016****	**<0.0001*****	**<0.0001*****
Underweight (BMI ≤20)	4.5	21.5	14.6	26.0
Healthy weight (BMI >20 and ≤25)	36.8	17.9	11.6	22.7
Overweight (BMI >25 and ≤30)	29.2	19.4	13.1	25.2
Obese (BMI >30)	21.7	25.5	15.4	32.1
Missing	7.8	24.9	14.2	30.8
S3 Menopausal status		**<0.0001*****	**<0.0001*****	0.16
Hysterectomy and/or oopherectomy	28.5	16.1	10.4	25.0
Premenopause	13.9	19.4	12.3	25.8
Perimenopause	25.3	19.4	12.0	25.8
Postmenopause	32.3	25.6	16.7	27.9
S1 Mental health		**<0.0001*****	**<0.0001*****	**<0.0001*****
MH ≤52	12.7	53.2	41.9	38.2
MH ≤52	87.3	15.9	9.0	24.5
S3 Chronic condition		**<0.0001*****	**<0.0001*****	**0.0380***
Yes (one or more chronic conditions)	58.1	25.9	16.4	27.1
No (no chronic conditions)	41.9	13.4	8.8	25.2

Numbers vary due to missing values; p values are from χ^2 for differences in proportions. The p values refer to differences in proportions between categories in row variables.
*p<0.05 (bolded);
**p<0.01;
***p<0.0001.
BMI, body mass index; CESD, Center for Epidemiologic Studies Depression scale; MH, mental health.

symptoms" (CESD-10 score of ≥10 or MH score of ≤52) are shown in Table 1. About one fifth of the sample had a CESD-10 score of ≥10 (20.7%) and 13.2% had an MH score of ≤52. Women who were born in Europe, were un-partnered (i.e., divorced, separated, etc.), or had no formal qualifications or no paid job, were over-represented in these "depressive symptoms" subgroups. These women were also more likely to have MH scores of ≤52 at S1; to be smokers, underweight or obese, postmenopausal; and to have one or more chronic health problems. The most commonly reported chronic health problems were arthritis, hypertension, stiff and painful joints, back pain, asthma, and low iron. Characteris-

Table 2. Means and 95% CI for CESD-10 and MH at follow-up (S3: 2001) for current (S3), previous (S1, S2), and habitual (S1, S2, S3) PA[a]

		Unadjusted		Adjusted[a]	
	n	Mean	95% CI	Mean	95% CI
CESD-10					
Current PA					
Very low	2860	7.0	6.8, 7.2	6.7	6.5, 6.8
Low	1957	5.8	5.6, 6.1	5.9	5.7, 6.1
Moderate	1834	5.4	5.2, 5.7	5.6	5.4, 5.8
High	2204	5.1	4.9, 5.3	5.3	5.1, 5.5
Missing	*352*	*6.2*	*5.7, 6.8*	*6.0*	*5.5, 6.5*
Previous PA					
Very low	2019	6.8	6.6, 7.1	6.4	6.2, 6.6
Low	2343	5.9	5.7, 6.1	6.0	5.8, 6.2
Moderate	2061	5.7	5.4, 5.9	5.8	5.6, 6.0
High	2476	5.4	5.2, 5.6	5.6	5.4, 5.8
Missing	*308*	*6.6*	*6.0, 7.2*	*6.3*	*5.7, 6.8*
Habitual PA					
Very low	1686	7.1	6.9, 7.4	6.7	6.5, 7.0
Low	2302	6.0	5.7, 6.2	6.0	5.8, 6.2
Moderate	2281	5.7	5.4, 5.9	5.8	5.6, 6.0
High	2312	5.3	5.1, 5.5	5.4	5.3, 5.6
Missing	*626*	*6.3*	*5.9, 6.7*	*6.0*	*5.7, 6.4*
MH					
Current PA					
Very low	2860	71.1	70.5,71.8	72.1	71.5,72.7
Low	1957	74.8	74.1,75.6	74.5	73.8,75.2
Moderate	1834	76.3	75.5,77.1	75.8	75.0,76.5
High	2204	77.8	77.0,78.5	77.1	76.5,77.8
Missing	*352*	*74.1*	*72.2,75.9*	*74.6*	*72.9,76.3*
Previous PA					
Very low	2019	71.2	70.4,71.9	72.5	71.8,73.8
Low	2343	74.7	74.0,75.4	74.5	73.9,75.2
Moderate	2061	76.0	75.2,76.8	75.5	74.8,76.2
High	2476	76.5	75.8,77.2	75.9	75.3,76.5
Missing	*308*	*72.9*	*71.0,74.9*	*73.8*	*72.0,75.6*
Habitual PA					
Very low	1686	70.5	69.6,71.3	71.7	70.9,72.4
Low	2302	74.4	73.7,75.1	74.3	73.6,74.9
Moderate	2281	75.6	74.9,76.3	75.2	74.5,75.8
High	2312	77.2	76.5,77.9	76.7	76.0,77.4
Missing	*626*	*73.8*	*72.4,75.1*	*74.4*	*73.1,75.6*

[a]Adjusted for country of birth, highest educational qualification, marital status, occupation, area of residence, current smoking status, body mass index, menopause status, MH score ≤52 at S1, and chronic condition at S3.
CESD, Center for Epidemiologic Studies Depression scale; CI, confidence interval; MH, mental health; PA, physical activity.

tics of the women in the "very low" PA category are also provided in Table 1. In this group, which comprised 26.3% of the sample, women born in Asia, those with no qualifications, those in a trade or clerical occupation, or living in urban areas, as well as smokers, obese women, and women with a MH score of ≤52, were all over-represented (Table 1). Because of these associations, all the variables shown in this table were included as covariates in subsequent analyses.

The crude and adjusted means and 95% CIs for CESD-10 and MH scores at S3 are shown in Table 2, for each PA variable. Adjustment for all the potential confounding variables, including MH score of ≤52 at S1, did not markedly affect the results. Both the unadjusted and adjusted results indicate a clear reduction in depressive symptoms, indicated by both lower CESD-10 scores and higher MH scores, with increasing current, previous, and habitual PA.

The results of the logistic regression analyses that examined the relationship between CESD-10 score of ≥10 and MH score of ≤52 and current, previous, and habitual PA, with adjustment for the covariates, are shown in Figure 1. The data show a clear and significant inverse association between PA and depressive symptoms. For MH score of ≤52, the greatest reductions in ORs were evident between the "very low" and "low" PA categories, with the OR for "low" PA significantly <1 for previous (OR=0.79, 95%CI=0.66–0.85), habitual (OR=0.70, 95%CI=0.59–0.85), and current (OR=0.68, 95% CI=0.57–0.81) PA (Figure 1). Increasing previous, habitual, and current PA was associated with further

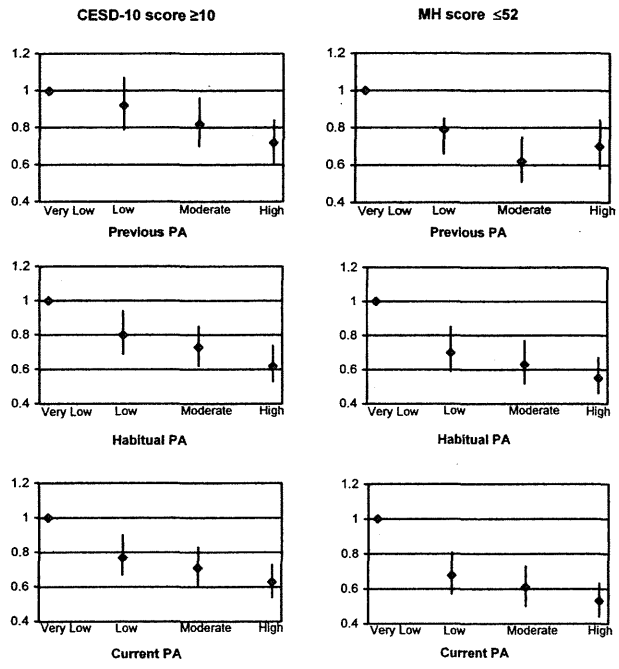

Figure 1. Odds ratios (and 95% confidence intervals) for CESD-10 score of ≥10 (left column) and mental health (MH) score of ≤52 (right column) for "low," "moderate," and "high" levels of previous, habitual, and current PA (referent category is "very low" PA). CESD, Center for Epidemiologic Studies Depression scale; PA, physical activity.

reductions in ORs, with the exception that the OR for "high" previous PA was slightly (but not significantly) higher than for "moderate" previous PA (Figure 1). Patterns of association were similar for PA and CESD-10; with the exception of the OR for low previous PA, all ORs were significantly <1, and decreased with increasing PA.

The final analyses, which included only those women who were in the "very low" PA category at S1, found that those who reported more PA at S3 had lower odds of CESD-10 score of ≥10 and MH score of ≤52 at S3 (Table 3).

Discussion

The aim of this study was to explore the dose–response relationships between PA and depressive symptoms using data from a large population-based cohort of middle-aged Australian women. The findings indicate a clear relationship between "current," "previous," and "habitual" PA, and two indicators of depressive symptoms in this sample, even after adjustment for a several potential confounders, and for MH scores at baseline. The findings also indicate that women who were in the lowest category for PA at baseline, but who reported PA at S3 that met recommended levels, were less likely to report depressive symptoms after 5 years than women whose PA levels remained very low.

The magnitude of the reduction in risk in this study (OR=0.62 for CESD-10 score of ≥10 and OR=0.55 for MH score of ≤52, with "high" levels of habitual PA) is consistent with results from previous prospective studies.[2–4,11] However, in this study "low" PA was also associated with significant reductions in risk of depressive symptoms. As the "low" category of PA equates with approximately 60 to 150 minutes of moderate-intensity PA per week, the results suggest that PA at levels below the currently recommended guidelines may be beneficial for the prevention of depressive symptoms. The analysis of change in PA for the women who were least active S1 also suggests that adoption of low-moderate levels of PA may be helpful in preventing the development of depressive symptoms.

The protective effect of PA remained even after adjustment for many of the sociodemographic and health-related variables that may also impact on PA levels and depressive symptoms. Based on reviews of

Table 3. ORs and 95% CIs for depressive symptoms (CESD-10 score ≥10; MH score ≤52) at S3, for women in "very low" PA category at S1, and with higher PA level at follow-up, compared with those who remained in "very low" (<240 MET.mins) category at S3

		Unadjusted		Adjusted[a]	
	n	OR	95% CI	OR	95% CI
CESD-10					
S1 (category) → S3 (MET.mins)					
Very low → <240	1225	1.00		1.00	
Very low → 240–<600	495	0.85	0.66–1.08	0.88	0.67–1.14
Very low → ≥600	591	0.75	0.59–0.94	0.78	0.61–1.01
MH					
S1 (category) → S3 (MET.mins)					
Very low → <240	1225	1.00		1.00	
Very low → 240–<600	495	0.73	0.55–0.96	0.76	0.56–1.02
Very low → ≥600	591	0.62	0.48–0.82	0.64	0.47–0.85

[a]Adjusted for country of birth, highest educational qualification, marital status, occupation, area of residence, current smoking status, body mass index, menopause status, depressive symptoms, MH score ≤52 at S1, and chronic conditions at S3.
CESD, Center for Epidemiologic Studies Depression scale; CI, confidence interval; MET.mins, metabolic equivalent in minutes (see text for full description); MH, mental health; PA, physical activity.

factors associated with physical activity,[27,28] this study identified a priori a range of possible covariates including socioeconomic disadvantage (lower level of education, lower status occupation), smoking, menopause status, baseline mental health, and the coexistence of chronic conditions, that might confound the relationship between PA and depressive symptoms. This is important, as the women in this sample who were categorized as having depressive symptoms were more likely to be disadvantaged on a number of socioeconomic variables, to be postmenopausal, and to have one or more chronic health problems. Consistent with other research that has examined some but not all of these potentially confounding variables and reported no subsequent effects,[2,3,11,14] the statistical adjustment for these variables in combination did not significantly alter the relationship between PA and depressive symptoms, indicating that this is a relatively robust association.

The main strength of this study is that it included a large population-based sample of women who are generally representative of Australian women in this age group. Both their PA levels and the prevalence of "depressive symptoms" were similar to values reported for women of this age in other national surveys.[23,28] Because of the scope of the ALSWH, it was possible to adjust for a large number of variables that have the potential to confound the relationship between PA and depressive symptoms. This is important because there is high comorbidity between affective disorders and chronic health problems (such as heart problems, cancer, or diabetes[29]). Adjustment for both MH scores and chronic health conditions at baseline, however, did not significantly alter the positive relationship between PA and depressive symptoms.

The main limitation of this study is that it relied on self-report measures of PA and depressive symptoms. All the measures used, however, are widely accepted, and evaluations of the CESD-10,[17,30] the mental health index from SF-36,[19,31] and previous week recall of PA[24,25] have indicated acceptable psychometric properties. The fact that a different measure of PA was used at S1 means that the composite variables for "previous," and "habitual" PA do not provide a precise measure of total duration of activity. As it was assumed that "session" duration was 20 minutes for the S1 PA scores, and because most women reported sessions of ≥30 minutes at S2 and S3, it is likely that the PA levels are underestimated. Moreover, it is acknowledged that the PA measure does not "capture" incidental or unstructured PA,[25] which can be substantial in some occupations in which women are "on their feet all day" (e.g., teaching, nursing).

Several researchers have called for more population-based longitudinal studies with a specific focus on the optimal frequency, intensity, and duration of PA needed to produce reduced depressive symptoms.[6,7,9]

What This Study Adds . . .

Depression is predicted to become the second leading contributor to the global burden of disease by 2020.

Prospective data from this longitudinal study of middle-aged Australian women showed a clear dose–response relationship between increasing physical activity and decreasing depressive symptoms, independent of pre-existing physical and psychological health.

Given the typically low levels of physical activity among middle-aged women, increasing physical activity could be an important strategy for the prevention of depressive symptoms in this group.

In this study, **volume** of PA was chosen as the focus because (1) an accurate measure of duration at S1 was not available; (2) there was relatively little variation in intensity of activity among the women in this sample (the majority [78%] reported only walking or other moderate-intensity activity); and (3) previous studies have shown that PA volume is an important predictor of a range of health outcomes.[32]

The results of this research highlight the need for tightly controlled intervention trials to explore the prevention and treatment of depressive symptoms in at-risk populations. Trials with different combinations of frequency, intensity, and duration of PA, such as those being conducted by Dunn et al.[33] are now required to confirm whether the benefits of a "low-moderate" volume of activity can be confirmed.

Prevention and management of depressive symptoms constitute an important public health priority. Depressive symptoms are often recurrent and chronic, and can generate substantial individual and societal costs. Depression is ranked as the leading cause of nonfatal disease burden in Australia.[34] It is predicted to become the second leading cause, after ischemic heart disease, of the global disease burden by 2020.[15] The results presented here suggest that promoting low to moderate levels of PA could be an important strategy for the prevention of depressive symptoms among middle-aged women.

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対象の内訳	ヒト	動物	地 域	その他	研究の種類	縦断研究
対象	一般健常者	空白		( )		コホート研究
性別	女性	( )		( )		( )
年齢	45-50(47.5歳)	[redacted]		( )		前向きコホート
対象数	5000～10000			( )		( )

調査の方法	質問紙	( )				
アウトカム	予 防	なし	なし	なし	なし		( )
	維持・改善	な し	な し	な し	心理的指標改善	( )	( )

図 表

Table 3. ORs and 95% CIs for depressive symptoms (CESD-10 score ≥10; MH score ≤52) at S3, for women in "very low" PA category at S1, and with higher PA level at follow-up, compared with those who remained in "very low" (<240 MET.mins) category at S3

		Unadjusted		Adjusted[a]	
	n	OR	95% CI	OR	95% CI
CESD-10					
S1 (category) → S3 (MET.mins)					
Very low → <240	1225	1.00		1.00	
Very low → 240–<600	495	0.85	0.66–1.08	0.88	0.67–1.14
Very low → ≥600	591	0.75	0.59–0.94	0.78	0.61–1.01
MH					
S1 (category) → S3 (MET.mins)					
Very low → <240	1225	1.00		1.00	
Very low → 240–<600	495	0.73	0.55–0.96	0.76	0.56–1.02
Very low → ≥600	591	0.62	0.48–0.82	0.64	0.47–0.85

[a]Adjusted for country of birth, highest educational qualification, marital status, occupation, area of residence, current smoking status, body mass index, menopause status, depressive symptoms, MH score ≤52 at S1, and chronic conditions at S3.
CESD, Center for Epidemiologic Studies Depression scale; CI, confidence interval; MET.mins, metabolic equivalent in minutes (see text for full description); MH, mental health; PA, physical activity.

図表掲載箇所	
概 要 (800字まで)	＜目的＞身体活動(PA)と抑うつ症状の関係を検討すること。＜方法＞コホート名：Australian Longitudinal Study on Women's Health、対象者数：9207人、追跡期間：5年、因子評価方法詳細：身体活動量を先週の通勤や余暇での歩行と中高強度身体活動の時間と頻度を思い出し法で評価した。高強度は7.5メッツ、中強度は4メッツ、歩行は3．5メッツに換算して、それに実施時間をかけてメッツ分/週でPAスコアを算出した。これに基づき、分位1：240メッツ分/週未満、分位2：240-600メッツ分/週、分位3：600メッツ分/週以上の3群に分類した。＜結果＞身体活動量と抑うつリスクとの関係は、分位1：1、分位2：0.76(0.56-1.02)、分位3：0.64(0.47-0.85)であった。
結 論 (200字まで)	通勤や余暇時間での中高強度身体活動が多いほど、MHスコア52以下で評価した低いうつ兆候のリスクと関連したいた。
エキスパートによるコメント (200字まで)	定量化された身体活動と抑うつとの関係を示した価値のある研究である。

担当者 宮地元彦

Activity Level and Risk of Overweight in Male Health Professionals

ABSTRACT

Objectives. This study undertook to examine relationships between nonsedentary activity level, time spent watching television (TV)/videocassette recorder (VCR), and risk of overweight among men.

Methods. Men participating in the Health Professionals Follow-Up Study were mailed surveys. Cross-sectional analyses examined the prevalence and odds of being overweight; prospective analyses determined cumulative incidence rates and relative risks of becoming overweight over 2 years of follow-up.

Results. Cross-sectionally, odds of being overweight were 50% (95% confidence interval [CI] = 45%, 55%) lower for men in the highest quintile of nonsedentary activity level when compared with men in the lowest quintile. Among men watching 41 or more hours of TV/VCR per week, the odds of being overweight were 4.06 (95% CI = 2.67, 6.17) times greater than those for men watching no more than 1 hour per week. Prospectively, higher levels of nonsedentary activity and lower levels of TV/VCR viewing were independently associated with lower relative risks for becoming overweight between survey years.

Conclusions. Both a lack of nonsedentary activity and time spent watching TV/VCR contribute to the development of overweight in men. Sedentary and nonsedentary activities represent separate domains, each with independent risks for overweight. (*Am J Public Health.* 1996;86: 25–30)

Pamela L.Y.H. Ching, ScD, RD, Walter C. Willett, MD, Eric B. Rimm, ScD, Graham A. Colditz, MBBS, Steven L. Gortmaker, PhD, and Meir J. Stampfer, MD

Introduction

National surveys indicate that the US male population is becoming increasingly overweight. Among men 20 to 74 years of age, the prevalence of overweight has increased from 23.2% in the National Health Examination Surveys of 1960–1962 to 31.3% in the National Health and Nutrition Examination Surveys of 1988–1991 (NHANES III, phase 1).[1,2]

One approach to the prevention and treatment of overweight is to increase energy expenditure through regular physical activity. Physical conditioning reduces fat mass and total body weight, increases lean body weight, and is associated with weight control independent of dietary factors.[3–9] Exercise improves cardiorespiratory functional capacity and decreases serum lipid, glucose, and insulin levels.[9–12]

Although the relationship between moderate or vigorous physical activity and overweight has been examined in several studies, less attention has focused on sedentary behaviors and risk of overweight. Nearly 60% of the US adult population reports engaging in little or no leisure-time physical activity.[13] Inactivity is associated with reduced levels of lean body mass and increased body fat in adults, and with obesity in children and adolescents in the United States.[14–16] Watching television (TV) represents a major sedentary behavior; the average adult male watches more than 29 hours weekly.[17] Several cross-sectional studies have reported an association between TV viewing and obesity in adults,[18–20] but prospective data exist only for children.[21]

In the current study, we examined cross-sectional and prospective relationships between nonsedentary and sedentary activity levels and risk of overweight in a cohort of male health professionals surveyed in 1988 and 1990.

Methods

Study Sample

The Health Professionals Follow-Up Study has been described elsewhere.[22–24] Briefly, it is a longitudinal study that examines various lifestyle risk factors for cardiovascular disease, cancer, and other health and disease outcomes among a cohort of US male health professionals. In 1986, 51 529 male health professionals, 40 to 75 years of age, were enrolled in the study. The cohort consists of dentists (58%), veterinarians (20%), pharmacists (8%), optometrists (7%), osteopaths (4%), and podiatrists (3%) who completed a six-page baseline questionnaire in 1986 concerning their medical history, current diet, and other lifestyle habits, including physical activity patterns. Follow-up questionnaires were sent every 2 years to update information on health status parameters and health practices, including patterns of nonsedentary and sedentary activity. This analysis begins with the 1988 questionnaire because that was the first

Pamela L.Y.H. Ching and Steven L. Gortmaker are with the Department of Health and Social Behavior; Walter C. Willett, Eric B. Rimm, Graham A. Colditz, and Meir J. Stampfer are with the Department of Epidemiology; and Drs Willett, Rimm, and Stampfer are with the Department of Nutrition, Harvard School of Public Health, Boston, Mass. Drs Willett, Colditz, and Stampfer are also with the Channing Laboratory, Harvard Medical School and Brigham and Women's Hospital, Boston.

Requests for reprints should be sent to Pamela Ching, ScD, RD, National Immunization Program, Mailstop E-62, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Atlanta, GA 30333.

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