Physical Activity in Women
How Much Is Good Enough?

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Earlier this year, England’s Paula Radcliffe ran a marathon in 2 hours 15 minutes 25 seconds, shattering her own previous record by 113 seconds, and considerably narrowed the gap between male (2:05:38, held by Moroccan-born American Khalid Khannouchi) and female world records.1 It is likely that women the world over cheered her accomplishment. Here in the United States, women’s statistics outpace men’s in a related area; sadly, this is no reason for celebration. Data from the 2000 National Health Interview Survey indicate that more women than men are physically inactive: 72% of women do not engage in regular leisure-time physical activity compared with 64% of men.2

Among both women and men, the high prevalence of physical inactivity is a cause for concern. Physical inactivity increases the risk of developing many chronic diseases, including coronary heart disease, stroke, type 2 diabetes, and certain cancers.3 Such sedentary behavior is also associated with higher body weight, and obesity increases the risk of many of the same chronic diseases.4 While substantial evidence accumulated over the past half century documents the adverse health consequences of physical inactivity, until recently studies on this subject have been conducted mainly in men. For example, in a 1990 meta-analysis quantifying the magnitude of increased risk for coronary heart disease with inactivity, fewer than one fifth of the studies included women.5 However, over the past decade or so, many studies have specifically examined the impact of sedentary behavior on the health of women.6-8

How much physical activity is needed for health benefits in women? Or, given the penchant for sedentary ways, a better question may be how little? Both physicians and the public may be confused by 2 apparently contradictory recommendations from well-respected entities, the Centers for Disease Control and Prevention (CDC) and the American College of Sports Medicine (ACSM) and the Institute of Medicine (IOM). The CDC/ACSM recommendation, released in 1995, called for a minimum of 30 min/d of moderate-intensity physical activity, such as brisk walking, most days of the week.9 This recommendation relied heavily on data from observational epidemiological studies of physical activity and chronic diseases involving more than 100,000 persons, in which information on physical activity was primarily self-reported. Weight control was not a major focus of the recommendation. While a healthy weight is desirable, even overweight women and men who exercise at levels that satisfy the CDC/ACSM recommendation experience lower rates of cardiovascular disease and premature mortality.6,7,10

In contrast to the CDC/ACSM recommendation, the IOM guideline, released in 2002, emphasized weight control and was part of a larger recommendation for a healthy diet.11 The IOM guideline stressed balancing diet with physical activity, and concluded that 60 min/d of moderate-intensity physical activity daily is needed to maintain healthy weight. This amount of physical activity was derived from normative data in 407 healthy adults with a body mass index of 18.5 to 25 (healthy weight range), among whom total daily energy expenditure was measured using doubly labeled water.

While self-reported physical activity is less precise than energy expenditure measured using doubly labeled water, it is unclear that these different methods of assessing physical activity are responsible for differences in the recommendations. Self-reported data, using validated questionnaires, are appropriate and feasible for large studies of thousands or tens of thousands of persons. Validation studies have compared self-reported physical activities with activities reported in daily diaries. With regard to activities performed for sport or recreation, the relative ranking of participants agrees well. When comparing absolute amounts of energy expenditure, the findings range from an underestimate of energy expenditure by about one third to an overestimate of 10% using questionnaires.12-14

Two reports in this week’s issue of The Journal, both involving women, provide more data for the debate regarding 30 min/d vs 60 min/d of physical activity. Jakicic et al15 report the results of a clinical trial testing different durations and intensities of physical activity on weight loss. The authors randomly assigned 196 sedentary women with a mean (SD) age of 37.0 (5.7) years and body mass index of 32.6 (4.2) to 4 exercise groups. The 4 groups were in-

See also pp 1323 and 1331.
structured to eventually work up to exercising 5 d/wk at 40 min/d for the vigorous intensity/high duration group; 60 min/d, moderate intensity/high duration; 40 min/d, moderate intensity/moderate duration; and 30 min/d, vigorous intensity/moderate duration. Exercise sessions were not supervised and walking was encouraged (it accounted for 87% of reported exercise bouts). Additionally, all women were asked to reduce their energy intake to between 1200 and 1500 kcal/d and fat intake to 20% to 30% of total energy intake.

In intention-to-treat analyses (ie, comparing the randomized groups), all groups lost weight from baseline to 12 months. Weight loss ranged from 6.3 (5.6) kg in the moderate intensity/moderate duration to 8.9 (7.3) kg in the vigorous intensity/high duration group; there were no significant differences between groups. Energy expenditure during the exercise sessions was not measured, and investigators hypothesize that the lack of difference in weight loss may be due to similar energy expenditure among all groups (there were no significant differences in self-reported energy and fat intake among the groups). Similarly, all groups improved in cardiorespiratory fitness from baseline to 12 months, ranging from 13.5% (16.9%) improvement in the moderate intensity/moderate duration group to 22.0% (19.9%) in the vigorous intensity/high duration group; there were no significant differences between groups.

As in any clinical trial, no matter how well conducted, participants did not comply perfectly. In secondary analyses, the investigators analyzed changes in weight and fitness according to participant self-reported activity levels. Women who reported exercising for greater total duration per week experienced greater weight loss. Those who reported exercising for 150 min/wk to 199 min/wk (30-39 min/d for 5 days) at both 6 and 12 months lost 8.5 kg compared with 11.6 kg in those reporting 200 min/wk or more moderate intensity/moderate duration to 8.9 (7.3) kg in the vigorous intensity/high duration group; there were no significant differences between groups.

These data support the CDC/ACSM recommendation showing that 30 min/d of physical activity is sufficient for weight loss provided caloric intake is restricted (at baseline, participants reported consuming 2118 kcal/d; at 12 months, 1456 kcal/d—a large decrease of 31%). The data also show a dose-response relationship, with greater weight loss observed when greater total duration of physical activity was reported. This suggests that following the IOM recommendation of 60 min/d of exercise while maintaining the same caloric restriction will result in greater weight loss than adhering to the CDC/ACSM recommendation.

A second study in this issue also supports the CDC/ACSM recommendation. The report by McTiernan et al from the Women’s Health Initiative (WHI) included 74,171 women aged 50 to 79 years at study entry and investigated the association between recreational physical activity and incidence of breast cancer in postmenopausal women.16 Although previous studies have shown that an inverse association exists, data on the kind, intensity, and duration of activity needed are sparse.17 Women in the WHI study who engaged in the equivalent of 1.25 to 2.5 hours per week of brisk walking had an 18% decreased risk of breast cancer (relative risk, 0.82; 95% confidence interval, 0.68-0.97) compared with women who reported no walking or recreational activities. At higher levels of physical activity, little additional risk decrement was observed among the WHI women. However, other studies have shown more of a dose-response relationship18; the difference may be because a relatively small proportion of the WHI women engaged in high levels of physical activity.

Investigators also examined, separately, the associations of moderate intensity and moderate to vigorous intensity activities with breast cancer risk. There was no clear evidence that one was more strongly related to decreased risk. While information on light-intensity activities and walking was collected, no data were presented on their associations with breast cancer risk.

Few women can—or want to—train at the level Ms Radcliffe does to set world records in elite sporting events. It is therefore encouraging to know that women can experience health benefits at far lower levels of physical activity. The 2 studies in this issue add to scientific data showing that modest and achievable levels of physical activity—30 min/d on most days—can decrease the risk of chronic diseases including breast cancer, and coupled with appropriate dietary restraint (this is crucial), can help overweight women lose weight. In fact, other studies have shown that decreased rates of coronary heart disease and premature mortality begin to occur at even lower levels—perhaps at 1 to 2 hours per week of moderate-intensity leisure-time activity.6,7,10 Thus, when prescribing physical activity, it is appropriate for physicians to set a goal of 30 min/d of moderate-intensity activity, which can be accumulated in bouts of at least 10 minutes, most days of the week. For patients willing to do more, and for whom no contraindications exist, greater duration and increased intensity of activity can bring additional health benefits.

REFERENCES

Postdiarrheal Shiga Toxin–Mediated Hemolytic Uremic Syndrome

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Postdiarrheal Shiga toxin–mediated hemolytic uremic syndrome (HUS) is the major cause of acute renal failure in infants and young children and is a substantial cause of mortality and chronic morbidity. Adolescents who develop postdiarrheal HUS fare as well as younger children but adults, especially elderly individuals, experience a higher incidence of death and disability.

There are no treatments of proven value, and care during the acute phase of the illness, which is merely supportive, has not changed substantially during the past 30 years. Although US mortality for infants and young children decreased markedly following the widespread availability of pediatric dialysis units and intensive care facilities, 3% to 5% of patients who develop HUS still die during the acute phase.

An equal number have severe brain damage or end-stage renal disease (ESRD) and require chronic renal replacement therapy (dialysis or renal transplantation) within the first few years. A much larger proportion (30%-50%) sustain less severe renal sequelae (proteinuria, low glomerular filtration rate [GFR], or both) but approximately 10% of this group have both overt proteinuria and low GFR and are therefore at high risk for experiencing a slow loss of renal function and eventual ESRD due to hyperfiltration injury.

Shiga toxins 1 and 2 are the most important virulence factors for development of postdiarrheal HUS and are required for disease expression. Shiga toxins produced by enterohemorrhagic Escherichia coli (EHEC) are very potent subunit cytotoxins that were previously referred to as Shiga-like toxins because of their similarity to the prototypic Shiga toxin produced by Shigella dysenteriae type 1. Enterohemorrhagic E.coli colonize the large intestine via a distinct attaching and effacing lesion that provides a tight junction between the EHEC and the surface of the intestinal epithelial cells. Toxins then translocate into the circulation, probably facilitated by the influx (transmigration) of neutrophils, which increases paracellular permeability.

Shiga toxin–mediated HUS is almost always preceded by colitis, which usually becomes hemorrhagic, but can occur without a diarrheal prodrome or can be subsequent to a urinary tract infection caused by Shiga toxin–producing strains of E.coli. Once in the circulation, there is evidence that Shiga toxin is transported by neutrophils to the kidneys, where it is transferred and bound via the toxin's B-subunits to neutral glycolipid globotriaosylceramide receptors on target cells (eg, glomerular endothelial and tubular epithelial cells). It is assumed that specific organ involvement depends on the presence of globotriaosylceramide receptors. The toxin is then internalized via receptor-mediated endocytosis and routed to the endoplasmic reticulum. The single A subunit enzymatically inactivates the ribosomes via depurination of the 28S ribosomal RNA component resulting in a blockade of peptide elongation. When protein synthesis is inhibited, the cell dies or is severely damaged. This leads to glomerular endothelial cell swelling and detachment from the underlying basement membrane with secondary activation of both platelets and the coagulation cascade. This sequence of events results in the classic lesions that characterize the thrombotic microangiopathy of Shiga toxin–mediated HUS. Apoptosis also may

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