The Role of Free-Living Daily Walking in Human Weight Gain and Obesity

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OBJECTIVE—Diminished daily physical activity explains, in part, why obesity and diabetes have become worldwide epidemics. In particular, chair use has replaced ambulation, so that obese individuals tend to sit for ~2.5 h/day more than lean counterparts. Here, we address the hypotheses that free-living daily walking distance is decreased in obesity compared with lean subjects and that experimental weight gain precipitates decreased daily walking.

RESEARCH DESIGN AND METHODS—During weight-maintenance feeding, we measured free-living walking using a validated system that captures locomotion and body movement for 10 days in 22 healthy lean and obese sedentary individuals. These measurements were then repeated after the lean and obese subjects were overfed by 1,000 kcal/day for 8 weeks.

RESULTS—We found that free-living walking comprises many (~47) short-duration (<15 min), low-velocity (~1 mph) walking bouts. Lean subjects walked 3.5 miles/day more than obese subjects (n = 10, 10.3 ± 2.5 vs. n = 12, 6.7 ± 1.8 miles/day; P = 0.0009). With overfeeding, walking distance decreased by 1.5 miles/day compared with baseline values (−1.5 ± 1.7 miles/day; P = 0.0005). The decrease in walking that accompanied overfeeding occurred to a similar degree in the lean (−1.4 ± 1.9 miles/day; P = 0.04) and obese (−1.6 ± 1.7 miles/day; P = 0.008) subjects.

CONCLUSIONS—Walking is decreased in obesity and declines with weight gain. This may represent a continuum whereby progressive increases in weight are associated with progressive decreases in walking distance. By identifying walking as pivotal in weight gain and obesity, we hope to add credence to an argument for an ambulatory future. ***Diabetes*** **57:548–554, 2008**

Obesity is epidemic in developed countries and is emerging in middle- and even low-income countries; this in part explains the unprecedented increase in type 2 diabetes worldwide (1). It is widely agreed that this partially reflects mounting sedentariness (2,3). At the beginning of the 20th century 90% of the population of the world was rural. However, over the last century, more than two billion agriculturalists have become city dwellers (4). In the latter transition, physical activity has declined (5). In particular, chair use has replaced ambulation (6) such that obese individuals tend to sit for ~2.5 h/day more than lean counterparts (7). Walking is the principal component of nonexercise activity thermogenesis (NEAT), which is the energy expenditure association with free-living daily activities (8,9). This is because people walk for several hours each day and, even at slow velocity, walking doubles energy expenditure (10); hence, the product of the time engaged in walking and its energetic equivalent is numerically substantial. We therefore wondered whether walking might be mechanistically important in weight gain and obesity. To address the hypothesis that free-living daily walking distance is decreased in obesity, we examined the characteristics of free-living walking in lean and obese people. Then, to address the hypothesis that weight gain is associated with a decrease in walking distance, we compared free-living walking before and after overfeeding.

RESEARCH DESIGN AND METHODS

Twenty-two healthy, sedentary volunteers were recruited aged (mean ± SD) 39 ± 8 years. Ten subjects (5 women and 5 men) were lean (BMI <25 kg/m^2), and 12 subjects (7 women and 5 men) were obese (BMI >29 kg/m^2) (Table 1). All of the subjects worked in sedentary jobs, 19 were white, 2 were black, 1 was Hispanic, and the majority of the subjects were of middle socioeconomic class and lived within 10 miles of the laboratory. None of the subjects worked in our laboratories or on the study team. Subjects were excluded if they used any medication at the time of the study or within 6 months before the study, exercised more than twice each week, smoked, used alcohol, were pregnant, had any acute or chronic illness, complained of joint problems or pain, failed psychological evaluation for depression and eating disorder, reported mental illness, or had unsteady body weight (>2 kg fluctuation over the 6 months before study).

The subjects were studied as outpatients for 11 weeks. Meals were prepared in the metabolic kitchen at the Mayo Clinic General Clinical Research Center (GCRC). All foods were weighed to within 1 g. For the first 3 weeks, volunteers were fed to establish the dietary intake necessary to maintain steady-state body weight. The diet composition was 45% carbohydrate, 35% fat, and 20% protein. Subjects were instructed not to adopt new exercise practices and to continue their usual daily activities and occupation. For the last 8 weeks of the study, subjects received 1,000 kcal/day above their weight-maintenance needs. For the last 10 days of weight maintenance feeding and for the last 10 days of overfeeding, subjects wore a physical activity monitoring system (PAMS; described below) to characterize the duration, number, and intensity of the bouts of walking and to measure other body postures and movements. When the study was complete, all subjects were assisted, as needed, to lose any excess weight that had been gained. Informed written consent was obtained after the nature and possible consequences of the study were explained, and the study was approved by the Mayo institutional review board.

Measurements of body composition. The volunteer's body weight was measured each morning under standardized conditions (with an empty bladder, without shoes, and wearing consistent, light clothing). These measures were performed by trained GCRC personnel using the same calibrated scale (ScaleTronix 5005; S/N 5-1700; ScaleTronix, Wheaton, IL). Body fat was measured in duplicate using dual X-ray absorptiometry (Lunar, Madison, WI) after the 3 weeks of baseline feeding and after 8 weeks of overfeeding. The test-retest difference for duplicate measurements of fat mass was <2%.

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Received for publication 17 June 2007 and accepted in revised form 4 November 2007.

Published ahead of print at http://diabetes.diabetesjournals.org on 16 November 2007. DOI: 10.2337/db07-0815.

Additional information for this article can be found in an online appendix at http://dx.doi.org/10.2337/db07-0815.

GCRC, General Clinical Research Center; ICC, intraclass correlation coefficient; NEAT, nonexercise activity thermogenesis; PAMS, physical activity monitoring system.

TABLE 1
Demographic details of lean and obese participants

Subject	Sex	Age (years)	Weight maintenance energy intake (kcal/day)	BMI (kg/m^2)	Change in BMI (kg/m^2)
Lean (BMI <25 kg/m^2)					
1	W	44	1,815	19	1.55
2	W	39	2,360	20	0.54
3	M	33	2,710	21	2.27
4	M	27	2,037	22	0.24
5	W	41	2,142	22	0.24
6	M	34	2,706	23	0.87
7	W	29	2,354	23	2.13
8	M	53	2,589	24	0.77
9	W	55	1,904	25	2.17
10	M	29	2,628	25	1.22
Obese (BMI >29 kg/m^2)					
11	M	40	2,885	29	1.73
12	M	29	3,068	31	1.13
13	W	47	2,428	32	1.11
14	W	42	2,402	32	1.01
15	W	41	2,423	33	0.48
16	M	36	3,470	33	2.25
17	W	38	2,551	34	1.01
18	W	41	3,220	35	1.41
19	W	41	2,362	35	1.00
20	M	41	2,303	35	2.09
21	M	27	4,266	37	1.35
22	W	31	2,623	38	1.02

Data are *n*. M, man; W, woman. The change in BMI is the change in BMI with overfeeding.

Measurement of daily body posture and movement. We used a validated PAMS that captures data on body posture and movement in duplicate continuously every half second for 10 consecutive days (7,11–13). PAMS comprises six sensors (Fig. 1), four inclinometers (each of which captures two axes of acceleration against the gravitational field of the Earth) (CXTA02; Crossbow Technology, San Jose, CA), and two triaxial accelerometers (each captures motion in x, y, and z axes) (CXL02LF3-R; Crossbow Technology). The 14 axes of data were binned and stored every half second on two data loggers (Ready DAQ AD2000; Crossbow Technology). The inclinometers were attached to the right and left outer aspect of the trunk and right and left outer aspect of the thigh. The two accelerometers were placed over the base of the spine. Specially designed underwear (Fig. 1) was used to attach the sensors. The two data loggers were stored in a pouch worn around the waist. The PAMS weighed <1 kg. Every 24 h, study staff removed the sensors while the subject showered for 15 min. During this time, data from the data loggers were downloaded to a personal computer and analyzed using Matlab scripts (Mathworks). The time taken showering was taken to represent "standing" for this period of time. The accelerometers were calibrated for walking velocity daily, and the inclinometers were validated for accuracy and precision daily both using an electronic bench-testing protocol and while each subject walked on a calibrated treadmill at 1, 2, and 3 mph each for 2 min.

Sensor determination of body posture using PAMS was correct for 700 of 700 measurements of posture compared with written responses by two observers and for the 440 daily tests. There were log linear relationships between accelerometer output and velocity with $r^2 > 0.98$ in all cases. The relationship between the paired accelerometers showed an intraclass correlation coefficient (ICC) of 0.99. We have reported previously that factorial determinations of NEAT from PAMS showed a linear positive relationship with total NEAT calculated from doubly labeled water (corrected ICC = 0.90, $P < 0.001$) (7) and with room calorimeter measurements (12,13). This approach was validated using a room calorimeter (12) and doubly labeled water (7,14).

Data analysis and statistics. We were able to distinguish walking from other activities for every half second that this occurred. All six sensors that we use to define body posture and movement were exactly time stamped together. Thus, for any given half second of a day the posture sensors for the thighs and chest and the back accelerometers were aligned. Three postures were thereby defined: *1*) If the chest posture sensors and the thigh posture sensors indicated horizontal, the person was lying down. *2*) If the chest

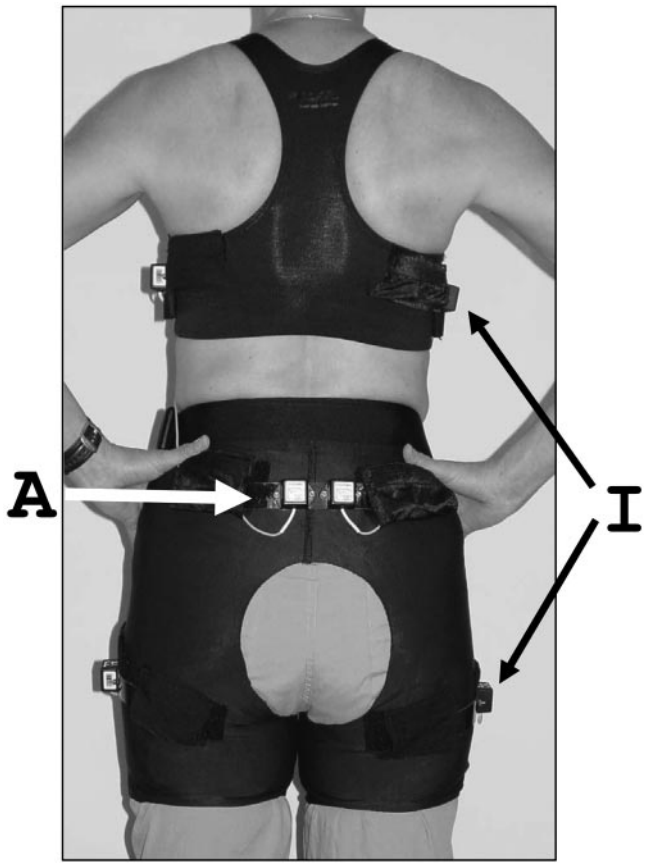

FIG. 1. Harnesses and sensor placement. Harnesses for accelerometers (*A*) and inclinometers (*I*). The harnesses are worn as "underwear."

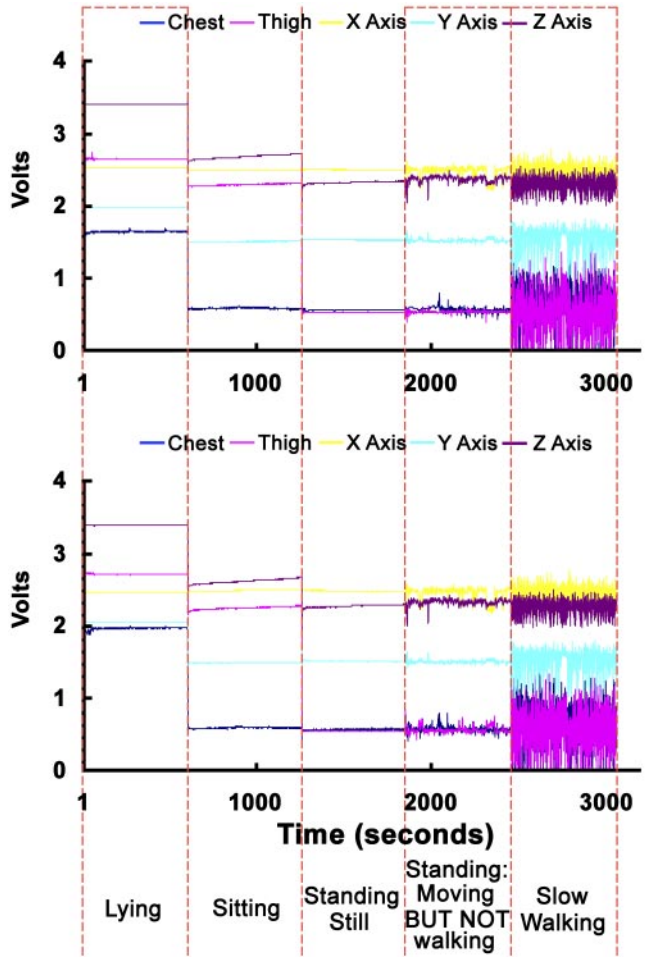

FIG. 2. Raw data stream from six sensors for different body postures and movements. A raw data stream from a subject lying still, sitting, standing still, standing while moving (but not walking), and walking. The *top panel* is from the left side of the body and the *bottom panel* from the right side of the body. (Please see http://dx.doi.org/db07-0815 for a high-quality digital representation of this figure.)

posture sensors indicated vertical and the thigh posture sensors indicated horizontal, the person was sitting down. *3*) If the chest posture sensors indicated vertical and the thigh posture sensors indicated vertical, the person was standing or walking.

How was standing distinguished from walking? The two triaxial accelerometers on the back record data on body movement. The principal acceleration with walking is in the vertical axis as a person elevates their body weight against gravity (the *z*-axis). However, if someone is walking forward, there is also acceleration in the forward-backward axis. Thus, when a person rhythmically elevates and descends during walking, the *z*-axis defines walking easily. This can be independently confirmed by the fact that we can also detect the forward motion of walking. Figure 2 illustrates the raw voltages from the six sensors for lying, sitting, standing still, standing while moving (but not walking), and walking. Walking can clearly be distinguished from other body postures using the accelerometer data streams labeled "*x*-axis, *y*-axis, and *z*-axis." Figure 2 illustrates an important additional verification of walking. The way posture sensors function is to use an electronic accelerometer orientated toward the gravitational field of the Earth. This is shown in the pink data streams labeled thigh (Figure 2, *top* [left thigh] and *bottom* [right thigh]). The pink-colored signals independently attest to the presence of walking because the swing of the thigh is measured. Furthermore, all data are gathered in duplicate because the sensors are paired and there was >99% concordance between the matched data pairs. We were therefore reassured that walking was likely to have been captured for the half second that it occurred.

Fat-free mass was calculated from the difference between body weight and fat mass. The regression equations between velocity and accelerometer output allowed mean free-living ambulatory accelerometer output to be translated into mean free-living velocity (11–13). The product of walking time and velocity is distance. A "bout" of walking was defined as when a person was standing for >2 s and showed locomotion. To address the primary hypotheses that walking distance was different between lean and obese individuals, an unpaired, two-tailed *t* test was used. To address the hypothesis that free-living walking distance decreased with overfeeding and whether this was different for lean and obese individuals, ANOVA and post hoc two-tailed *t* tests were used assuming normal distribution. Regression analyses were used elsewhere as indicated. Statistical significance was defined as $P < 0.05$. The Matlab programs used for data analysis are available at no cost from P.H.K.

RESULTS

All of the subjects tolerated the sensor load and study protocol. Subjects continued their normal occupations, hobbies, and other daytime and nighttime activities.

The 10-day walking characteristics of 22 free-living, sedentary, healthy subjects of varying weight were initially examined while the subjects were weight stable. When the subjects' 10,312 bouts of walking were analyzed, we found that free-living walking comprises many short-duration, low-velocity walking bouts (Fig. 3). On average, a participant took 47 (range 46–62) walks per day: 85% were <15 min in duration, and 88% occurred at <2 mph; this is consistent with the observation that free-living people predominantly exhibit low-amplitude, high-frequency movements (15). We were quite surprised that on average, free-living people walked about 7 miles/day (Table 2). However, when one considers that people are ambulating for 6.5 h/day and that free-living velocity just exceeds 1 mph, sedentary people indeed walk ~7 miles per day. Thus, in sedentary people, free-living walking represents

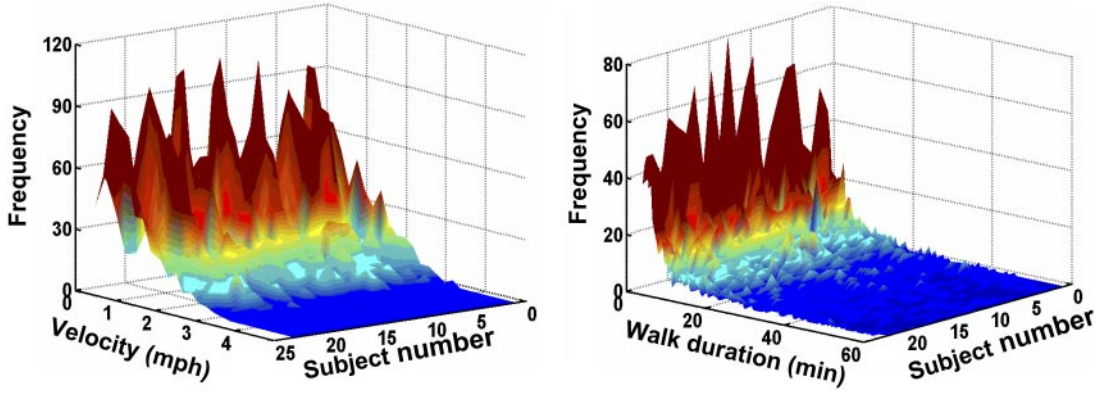

FIG. 3. Distribution of velocity and duration of walks in free-living people. The distribution of velocity and duration for 10,312 bouts of walking taken by 22 separate subjects (1–22) of varying weight during 10 days of weight-maintenance feeding. See online appendix for additional data (available at http://dx.doi.org/10.2337/db07-0815, where a high-quality digital representation of this figure can also be accessed.)

TABLE 2
Free-living walking characteristics for obese (n = 12) and lean (n = 10) subjects and the total group (n = 22)

	Walking bouts (n/day)	Time engaged in walking (minutes/day)	Average distance of a walking bout (miles)	Free-living walking velocity (mph)
Baseline				
Lean	46 ± 8	448 ± 111	0.22 ± 0.04	1.19 ± 0.21
Obese	47 ± 5	339 ± 74*	0.14 ± 0.04†	1.10 ± 0.20
Total	47 ± 6	389 ± 106	0.18 ± 0.06	1.14 ± 0.20
Overfed				
Lean	48 ± 11	459 ± 119	0.19 ± 0.06‡	1.09 ± 0.28
Obese	47 ± 9	334 ± 79*	0.11 ± 0.03§¶	0.96 ± 0.15‖
Total	47 ± 10	391 ± 116	0.15 ± 0.06**	1.02 ± 0.20**
Change with overfeeding				
Lean	1.6 ± 5	11 ± 43	−0.03 ± 0.04	−0.10 ± 0.14
Obese	−0.3 ± 7	−5 ± 51	−0.03 ± 0.05	−0.14 ± 0.15
Total	0.6 ± 6	2 ± 47	−0.03 ± 0.04	−0.12 ± 0.14

Data are means ± SD. Data were gathered every 0.5 s for 10 days continuously during weight-maintenance feeding and after 8 weeks of overfeeding by 1,000 kcal/day using validated posture and motion sensors worn under clothing (7). Statistical differences are indicated: obese subjects compared with lean, *P = 0.01, †P = 0.002, §P = 0.001; overfed compared with baseline, ‡P = 0.05, ¶$P < 0.005$, ‖P = 0.01, **$P <$ 0.001.

the cumulative effect of many short-duration, low-intensity walks.

Our primary hypothesis was that in obese subjects, free-living walking is decreased compared with sedentary lean controls. The subjects with obesity walked one-third less distance per day than lean individuals (P = 0.0009; Table 2). This difference represented ~3.5 miles (P = 0.0009) or ~2 h (P = 0.01) of walking per day (Table 2). When expressed relative to BMI units (calculated as weight in kilograms divided by the square of height in meters), for the lean subjects, walking distance was 0.46 ± 0.12 miles/day per BMI unit and for the obese subjects, 0.20 ± 0.05 miles/day per BMI unit ($P <$ 0.0001). These differences occurred because the distance of each walking bout walked was one-third shorter (P = 0.002) in the obese subjects compared with the lean, whereas the number of walking bouts per day and free-living walking velocity was similar between the groups (Table 2). For the 22 subjects, there was a significant negative linear correlation between walking distance and body fat (Fig. 4*A*). This suggests that walking distance progressively declines with increases in body fat. The subjects with obesity in these studies had mild obesity and did not have any joint pain or disease, so this did not explain the differences. Thus, people with obesity walk less distance than lean, sedentary counterparts because their bouts of walking were shorter.

If the mechanism(s) that underlies obesity is associated with a decline in walking distance, we wondered whether experimental weight gain would be associated with decreased walking. To examine this, we compared the walking characteristics of all of the volunteers after 8 weeks of overfeeding by 1,000 kcal/day above weight-maintenance needs. We thereby analyzed 10,438 bouts of walking after 56,000 kcal of overfeeding in a similar fashion to the weight-maintenance baseline. All of the subjects gained weight in a fashion compatible with life-long weight gain; the average weight gain was 3.6 ± 1.6 kg, and the average fat gain was 2.8 ± 1.7 kg; $P < 0.0001$. With overfeeding, daily walking distance decreased by 1.5 miles/day (P = 0.0005) (Fig. 4*B*). The number of walking bouts and total daily walking time was constant with weight gain (Table 2; Fig. 4*C*). The decrease in walking distance with overfeeding occurred because the bouts of walking became significantly shortened through a decrease in free-living walking velocity (P = 0.0007; Table 2). The greater the decrease in the velocity with overfeeding, the greater the decrease in walking distance (Fig. 5; r = 0.81; $P < 0.0001$). The magnitudes of the decreases in walking associated with overfeeding were similar for the lean and obese subjects (Table 2).

To us, these findings suggest that obesity lies on a continuum with respect to these adaptations to overfeeding (Fig. 4*A*). We suggest that walking is under mechanistic control because of the remarkable intra-individual consistency that the components of walking exhibited before and after overfeeding (Fig. 4*C*). Thus, with overfeeding and experimental weight gain, free-living walking distance decreases.

We also examined the hypothesis that nonambulatory movement differed between the lean and obese subjects. Seated accelerometer output per day was 787 ± 327 vs. 1,041 ± 381 accelerometer units (AU)/day. Lying accelerometer output was 808 ± 344 vs. 688 ± 147 AU/day. When corrected for the number of minutes allocated to these postures, for the lean seated accelerometer, output was 1.84 ± 0.51 AU/min and for the obese subjects, 1.78 ± 0.52 AU/min. Lying accelerometer output was 1.57 ± 0.74 AU/min for the lean and 1.40 ± 0.24 AU/min for the obese subjects. None of these differences was significantly different. There was no relationship between walking velocity and BMI.

Finally, to address the concern that the decrement in walking distance that we saw with overfeeding was not a result of the repeated measures, eight of our subjects (three women and five men; 35 ± 6 years, BMI 27 ± 5 kg/m^2) agreed to wear PAMS for a third 10-day period once they had lost the excess body fat (baseline 27.4 ± 13.8 kg body fat; repeat baseline 27.5 ± 13.4 kg). Their baseline walking distance was 8.7 ± 3.7 miles/day, and on the repeat measurement, 9.0 ± 3.7 miles/day; this was not significantly different. There was a good correlation for the two baselines (albeit separated by ~6 months) (r = 0.95, $P < 0.001$). Thus, it is highly unlikely that the decrement of

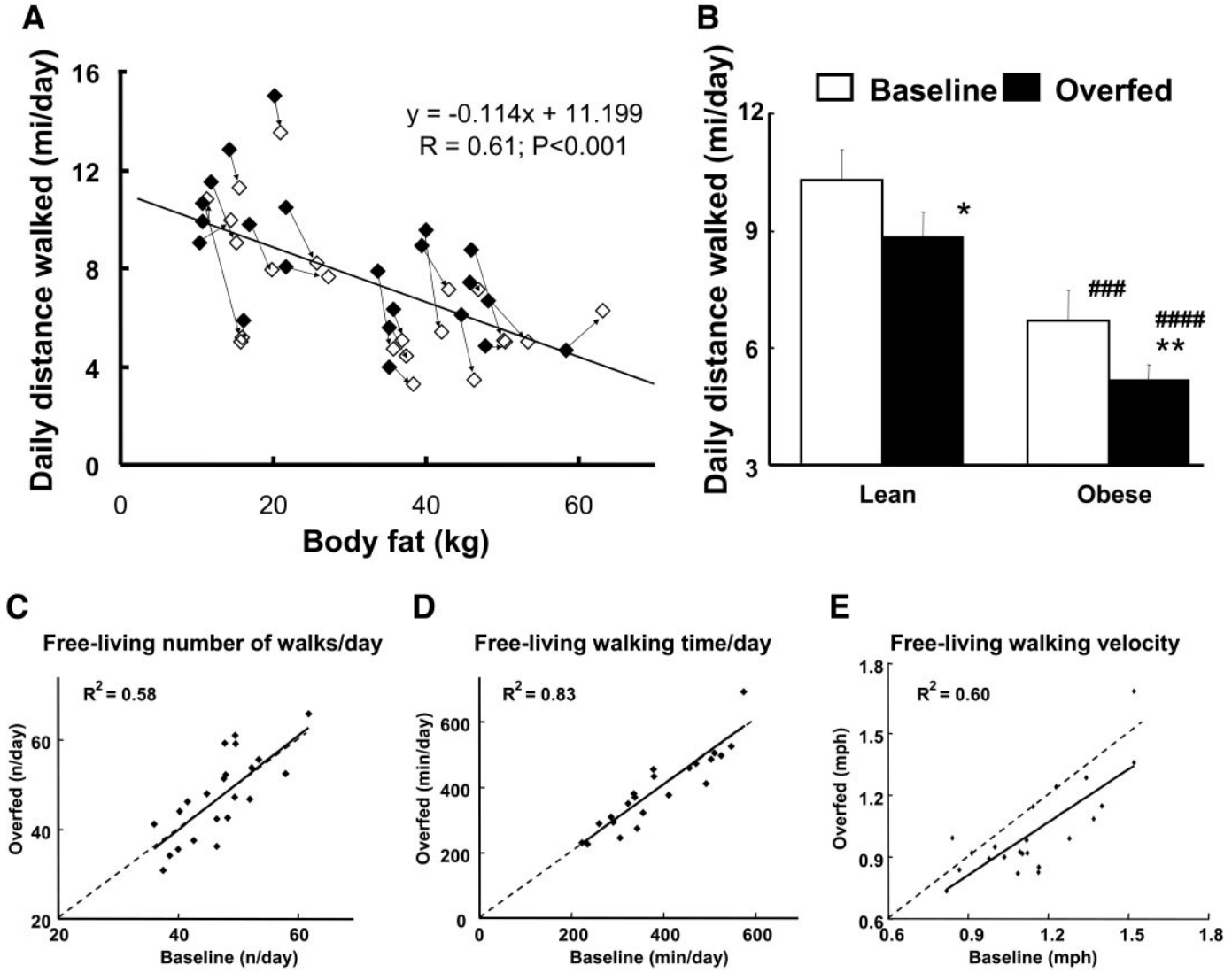

FIG. 4. *A*: Body fat and daily walking distance in 22 free-living people. Body fat was determined by dual X-ray absorptiometry. The baseline (♦) equation of the regression line was $y = -0.086x + 10.5$. The post–weight gain (◇) regression equation is $y = -0.083 + 9.8$ ($r = -0.55$); there were no significant differences for the slopes or intercepts. The arrows represent the changes for individuals. *B*: Daily walking distance in free-living lean and obese subjects before and after overfeeding. Free-living daily distance walked during baseline weight-maintenance feeding and after 8 weeks of overfeeding by 1,000 kcal/day. Data are means ± SE. Statistical differences are indicated; obese subjects compared with lean, $^{###}P = 0.0009$, $^{####}P = 0.0003$; overfed compared with baseline, $^{*}P = 0.04$, $^{**}P = 0.002$. Data by sex for women and men, respectively, for daily walking distance at baseline: 8.5 ± 3.0 compared with 8.2 ± 2.7 miles, and with overfeeding: 6.9 ± 3.0 compared with 6.8 ± 2.5 miles. *C–E*: The components of free-living walking during baseline weight-maintenance feeding and after 8 weeks of overfeeding by 1,000 kcal/day in 22 subjects determined using PAMS. The broken lines are the lines of identity. *C*: Number of walking bouts per day. *D*: Time spent walking each day. *E*: Free-living walking velocity.

walking distance that we saw with overfeeding can be explained by repeat measures.

DISCUSSION

Low levels of physical activity have been associated with obesity and type 2 diabetes (10,16–21). To address the hypothesis that walking distance is decreased in obese individuals compared with lean sedentary individuals, we compared all of the bouts of walking that lean and obese people took. We found that free-living walking comprises many (~47) short-duration (<15 min), low-velocity (~1 mph) walking bouts. Obesity is associated with decreased walking by 3.5 miles per day. It could be argued that this might reflect greater body weight rather than a mechanist effect of weight gain. To address the hypothesis that weight gain is associated with decreased walking, we overfed lean and obese individuals and reassessed their walking. Walking distance decreased with overfeeding similarly in the lean and the obese subjects. The data suggest that obesity falls on a continuum that is mechanistically associated with diminished ambulation.

There has been much written about the link between progressive urbanization, mechanization, and obesity (22–24), and implicit in these analyses is the idea that obesity and general levels of inactivity and walking (25) concur. However, the direct link between obesity and free-living walking is lacking generally because of the difficulty in gathering data on free-living walking in lean and obese subjects and because of the difficulty in gaining such information with experimental weight gain. This is partially because readily available technologies such as pedometers are inaccurate and imprecise (26) and partially because overfeeding lean and obese subjects is technically challenging. The data presented here were gathered with

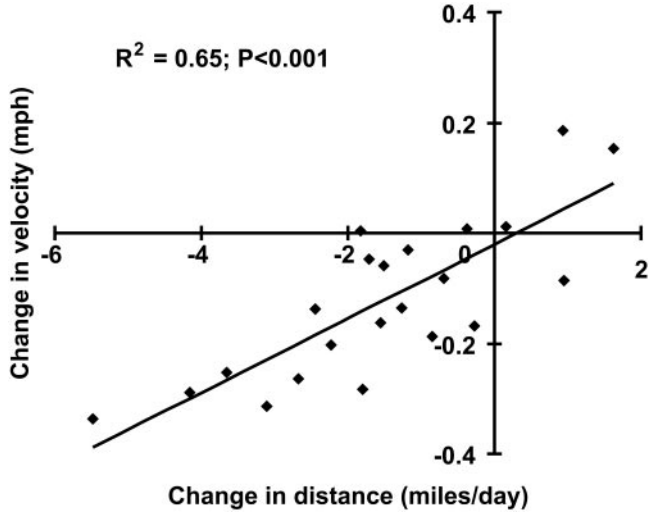

FIG. 5. Changes in free-living walking distance versus changes in walking velocity in 22 free-living lean and obese subjects before and after overfeeding.

precise and accurate equipment that enabled us to address our hypotheses (7). The data demonstrate that walking is diminished in obesity and that with progressive weight gain, walking decreases. Furthermore, these data also suggest that walking is intricately programmed and regulated such that after 2 months of overfeeding, the number of bouts of walking and their duration is fixed, whereas walking velocity is dynamically modulated to impact the total distance walked. Hence, because there was such remarkable intra-individual preservation of the components of free-living walking, this implies that walking is under detailed and exquisite mechanistic control.

The concept that there is central "wiring" of time allocation with respect to locomotion has widespread precedent throughout biology (e.g., primates [27], other mammals [28]; birds [29], and fish [30]); also, homozygous Clock mutant mice (31) show temporally disrupted activity patterns. Thus, for the hypothetical person predisposed to fat gain, the more protracted the excess of energy intake, the greater the fat gain and the greater the decrease in walking. Although these observations are beyond the scope of these clinical studies, others (32) and ourselves (33) have been interested in orexin as a potential mediator of movement and ambulation whereby obesity-prone rats have decreased sensitivity to central orexin administration with weight gain compared with lean rats (34). We speculate that with weight gain, there is decreased sensitivity to central neurotransmitters that drive walking.

There were limitations to our study. We recognize, for example, that the sample size was small. However, we were adequately powered to address our hypotheses because our measurements were highly intensive, accurate, and precise. The period of weight gain was only 2 months, so it could be argued that if the weight was sustained for longer, the decline in walking may not have been sustained with overfeeding. However, the fact that the obese individuals show the effects of sustained weight gain and have diminished walking argues against this. We respect that we did not follow our subjects when free-living and so could not directly assess free-living walking; however, potential errors associated with PAMS prediction of free-living walking would have been systematic and would not have been biasing with respect to addressing our hypotheses. Finally, we recognize that recruiting biases could potentially affect these data, especially since informed written consent was obtained. However, this is true of all human studies, especially those where behavior can impact the data. Having noted this, however, it is difficult to believe that recruitment biases explain the unexpected sequential declines in walking distance that we demonstrated and the regression relationships that we depict in Figs. 4 and 5.

The implications of these data regarding energy are potentially intriguing. Previous data demonstrated that NEAT increases with overfeeding (35). Because NEAT is the product of the time engaged in nonexercise activities and their energetic equivalents and because walking is the predominant component of NEAT, the data presented here imply that as NEAT increases with overfeeding, walking should become more energy wasteful. This will become the focus of future experiments and analyses, but we conjecture that the modulation of energy efficiency is a primary mechanism for dissipating excess energy consumption.

We suggest that obesity is associated with a walking deficit of ~2 h of walking per day that occurs because walking bouts are shorter. This information is useful for nationwide programs that promote walking (36) and may promote studies that target walking components in obesity. It would be beneficial to decipher the mechanism that underlies this. For example, if a gene variant was predictive of a tendency to have shortened daily walking, such individuals could be targeted with early obesity prevention or treated using focused interventions. The question posed, particularly from the animal data (34), is whether ambulation is so preprogrammed that obesity is inevitable. In humans, this cannot be so because obesity was rare a century ago when the majority of the population lived in agricultural environments and were more active. Interestingly, failing to walk to work (37) alone accounts for the majority of the 100 kcal/day of negative energy balance that Hill described as being responsible for the emergence of obesity in the U.S. (22). To increase walking by 2 h/day, we need to consider integrating greater walking into work and leisure time; a simple solution might be to convert 1 h of work time plus 1 h of leisure time from chair-based to walk-based activities. Thus, targeted prevention and intervention coupled with an activity-permissive society should enable the walking deficit associated with obesity to be resolved.

Obesity and overweight affect more than one-half of the U.S. population, and obesity has emerged as a worldwide epidemic. The role of the sedentary, seated lifestyle in the presence of abundant food in the genesis of obesity is not disputed (21). Obesity was uncommon a century ago, when the environmental cues to walk prevailed (22). By identifying walking as pivotal in weight gain, we hope to add credence to an argument for a future that, like the past, is ambulatory.

ACKNOWLEDGMENTS

This work has received support from the National Institutes for Health and the Mayo Foundation.

We thank the volunteers, dietitians, food technicians, nursing staff, Mass Spectrometer Core at the GCRC, and Prof. M. Clark for assistance with psychological assessments and counseling of subjects.

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