



Comprehensive review

Received: 2018/05/28, Revised: 2018/06/11,
Accepted: 2018/06/12, Published: 2018/06/30

©2018 Nana Chung et al; License Journal of Exercise Nutrition and Biochemistry. This is an open access article distributed under the terms of the creative commons attribution license (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

*Corresponding author: Kiwon Lim Ph.D.

Department of Physical education, Konkuk University, Seoul, Republic of Korea.

Phone: 82-2-450-3827 / E-mail: exercise@konkuk.ac.kr

©2018 The Korean Society for Exercise Nutrition

Non-exercise activity thermogenesis (NEAT): a component of total daily energy expenditure

Nana Chung¹ / Mi-Young Park¹ / Jisu Kim¹ / Hun-Young Park¹ / Hyejung Hwang¹ / Chi-Ho Lee¹ / Jin-Soo Han¹ / Jaemoo So¹ / Jonghoon Park¹ / Kiwon Lim^{1,2*}

1. Physical Activity and Performance Institute (PAPI), Konkuk University, Seoul, Republic of Korea
2. Department of Physical Education, Konkuk University, Seoul, Republic of Korea

[Purpose] The purpose of this review is to promote awareness of non-exercise activity thermogenesis (NEAT) as a new strategy to increase energy expenditure, and to manage obesity.

[Methods] The content of this review is based on a literature search of PubMed and the Google Scholar search engine, using the search terms obesity, energy expenditure, non-exercise activity thermogenesis (NEAT), and sitting disease.

[Results] Daily energy expenditure is of great interest because most obese individuals have no exercise activity-related thermogenesis (EAT); thus their physical activity-related energy expenditure (PEE) is comprised almost entirely of NEAT. Consequently, NEAT represents the main variable component of daily total energy expenditure (TEE); this varies considerably, both within among individuals. These somewhat unplanned and unstructured low level physical activities are associated with energy expenditure in excess of the resting metabolic rate (RMR). They may therefore have the potential to stimulate greater energy expenditure over time with a higher rate of adherence.

[Conclusion] In conclusion, NEAT is a highly variable component of daily TEE and a low level of NEAT is associated with obesity. NEAT enhances lifestyle, and variations in individual and environmental factors can significantly affect daily energy expenditure. Therefore, well designed longitudinal studies that focus on personal behavioral approaches and re-engineered environments to increase NEAT should be conducted in the future.

[Keywords] Obesity, energy expenditure, non-exercise activity thermogenesis (NEAT), sitting disease.

INTRODUCTION

Obesity is a leading preventable cause of death worldwide; however obesity rates are increasing in adults and children¹. Globally, 600 million adults and 100 million children were obese in 2015². In 2013, the American Medical Association classified obesity as a disease³. Individuals are considered to be obese when their body mass index (BMI), a measurement obtained by dividing body weight by the square of height (kg/m²), exceeds 30 kg/m², with a BMI of 25–30 kg/m² defined as overweight¹. Some East Asian countries use lower values⁴. Excessive body weight increases the likelihood of various diseases, particularly type 2 diabetes mellitus, hypertension, dyslipidemia, cardiovascular diseases, obstructive sleep apnea, asthma, osteoarthritis and certain types of cancer⁵⁻⁷. As a consequence, obesity has been found to reduce life expectancy. Obesity poses serious public health and policy problems because of its prevalence, health effects and health-care costs⁸.

The etiology of obesity is multifactorial; resulting from genetic⁹⁻¹¹, epigenetic^{11,12}, physiological^{13,14}, behavioral, sociocultural¹⁵, and environmental^{16,17} factors that lead to an imbalance between energy intake and energy expenditure. A limited case are genes, endocrine disorders, medications, or psychiatric disorder¹⁸. Excessive food intake and a lack of physical activity are believed to be the two most important causes of the recent increase in obesity¹⁹. The modern lifestyle with decreasing levels of physical activity and food intake in excess of daily energy expenditure is closely related to the increase in obesity. Nevertheless, whether increased energy intake is associated with the increased prevalence of obesity remains controversial, since previous studies have shown that energy intake has remained relatively constant. For example, in Britain since the 1980's, obesity rates have doubled but energy intake appears to have actually declined²⁰. Weinsier et al. 1998²¹ also found diverging trends of decreasing energy intake and increasing body weight. Furthermore, Hill et al. 2012²² speculated that if physical activity decreased, body weight may increase if energy intake is not altered. In the absence of evidence that positively associates increased energy intake with obesity, the role of physical activity in human energy bal-

ance has come under greater scrutiny. Since a reduction in energy expenditure might be the most important factor explaining the rising obesity pandemic^{23,24}, it is important to examine the progressive decline in daily energy expenditure.

Traditional interventions to overcome a net positive energy balance have focused on encouraging moderate- to vigorous- intensity physical activity. However, this strategy has met with limited success in long-term randomized clinical trials. Therefore, a new strategy has recently emerged that focuses on increasing low level daily physical activity or NEAT (non-exercise activity thermogenesis), which includes the addition of short repetitive bouts of non-exercise physical activity. NEAT demonstrates inter-individual variation due to differences in occupation and leisure-time activities; however it is crucial to the regulation of energy expenditure. Therefore NEAT may contribute to our understanding of the causes and successful treatment of obesity.

The purpose of this review is to promote NEAT as a new strategy to increase energy expenditure and to aid the management of obesity.

METHODS

This review used literature obtained from the PubMed and Google Scholar search engines, with the following search terms: obesity, energy expenditure, non-exercise activity thermogenesis (NEAT) and sitting disease. We conducted a comprehensive literature search from inception up to 2018, without language or year of publication restrictions. We excluded studies with incomplete data, letters, editorials and case reports. The references of the studies included in the full-text review were searched for cross-references, to find the studies that could have been missed in the original search.

RESULTS and DISCUSSION

NEAT: a component of total daily energy expenditure

Three main components of energy balance determine total energy expenditure (TEE): resting metabolic rate (RMR), diet-induced thermogenesis (DIT), and physical activity-related energy expenditure (PEE)²⁵.

RMR represents the minimal amount of energy expended for homeostatic processes^{23,24}, and it accounts for approximately 60% of daily total energy expenditure (TEE) in a mainly sedentary individual²⁶. About 80% of the variance in RMR is determined by body size, with lean body mass (LBM) positively correlated with RMR. Diet-induced thermogenesis (DIT) accounts for approximately 10-15% of TEE, and it is related to digestion, absorption and the storage of food. The variance of DIT has been associated with the nutrient composition and energy content of consumed foods²⁷. Although it is theorized that energy expenditure changes in relation to alterations

in intake, the magnitude of change remains disputed^{28,29}, and it does not vary greatly between individuals³⁰. The third main determinant of TEE is physical activity-related energy expenditure (PEE) that accounts for 15% to 30% of TEE. It is defined as the additional energy expenditure above RMR and DIT, which is required for activity. PEE can be further subdivided into exercise-related activity thermogenesis (EAT) and NEAT. These vary widely, both within and among individuals. EAT is defined as planned, structured, and repetitive physical activity that has the objective of improving health (for example, sport, visiting the gym)³¹. NEAT represents the predominant component of daily activity thermogenesis with the exception of some sports-like exercise and resistance training³². NEAT is the energy expenditure that we do not typically consider and it includes the energy expended maintaining and changing posture (laying, standing, walking, stair climbing, spontaneous muscle contraction, fidgeting, cleaning), singing, and other activities of daily living. These activities do not involve moderate- to vigorous- intensity exercise and occur at a trivial or a low energy workload for minutes to hours.^{33,34} These somewhat unplanned and unstructured low grade physical activities can have a remarkable effect on metabolic rate and, as a result, stimulate greater energy expenditure over time.

Causes of decreased energy expenditure

Basal metabolic rate (BMR) is largely (~80%) accounted for by body size and DIT is a relatively small component of TEE (10-15%), so the variance in TEE between individuals of similar size can only be explained by variance in PEE (EAT and/or NEAT)^{26,29,32}. Recently, there has been a large shift towards less physically demanding work, and currently at least 30% of the world's population lacks sufficient physical activity¹. This is primarily due to an increasing reliance on cars, mechanized manufacturing and a greater prevalence of labor-saving technology in the home, combined with an inactive lifestyle³⁵. This has caused a progressive and systematic decline in energy expenditure, resulting in a sustained positive energy balance. More time is devoted to sedentary behaviors involving prolonged sitting, and this global trend is likely to continue. Obesity has emerged as a result of environmental influences with increasing inactivity. Evidence suggests that an active lifestyle conveys many important health benefits, and sedentary habits are associated with an increased risk of chronic diseases and decreased life expectancy³⁶. Recognition of the health and functional hazards of an inactive lifestyle has led to the development of public health recommendations for physical activity.

Traditional strategies to increase energy expenditure

Low energy expenditure was suggested to play a role in the development of obesity because of a lower RMR, DIT, PEE or a combination of all of these components, contributing towards a positive energy balance and subsequent weight gain³⁷. For obesity treatment to be successful, an

understanding of the physiological determinants of obesity should be carefully considered. Although our understanding of body weight regulation and energy balance has evolved, how alterations of the individual components of energy expenditure influence energy balance is debated. Nevertheless, PEE (EAT + NEAT) has the greatest variability of all the TEE components.

To address obesity and its related clinical complications, strategies that are mainly based on physical activity in the form of EAT have been implemented. To prevent and control obesity, exercise guidelines typically recommend > 30 min/day of moderate intensity exercise, at least 5 days/week³⁸⁻⁴⁰. There is sufficient exercise physiology research to support these public health guidelines, promoting at least 150 min/week of moderate-vigorous leisure-time physical activity, aimed at decreasing the risk of metabolic diseases^{41,42}. Commonly EAT accounts for a maximum of 15-30% of TEE in those who regularly participate in the recommended physical training^{25,43}, and it explains 1-2% of the variance in TEE. However, for the majority of people in modern society, EAT is believed to be negligible. Also, adherence to the recommended exercise intensity and duration remains low in obese patients, and consequently, EAT is nearer to zero^{26,44}. Even for people who participate in exercise for less than two hours a week, exercise accounts for an average energy expenditure of only ~100 kcal/day⁴⁵. Interestingly, even if adults adhere to the exercise guidelines, sitting for prolonged periods of time can compromise their metabolic health⁴⁶. Therefore, NEAT has emerged as the main component of TEE variability³⁰. This suggests that in addition to moderate-vigorous intensity physical activity, light-moderate intensity daily physical activity should also be considered as an alternative and complimentary exercise therapy regimen for obese individuals. Attention should shift to an active lifestyle intervention to reduce obesity, with NEAT considered as a substitute for the amount of time currently spent inactive, to increase the low level of energy expenditure.

NEAT as a new strategy to increase energy expenditure

If the EAT of individuals with obesity is considered to be negligible, PEE is comprised entirely of NEAT. Consequently, NEAT represents the main variable component of TEE within individuals, and it also varies substantially between individuals. Although the contribution of NEAT to daily energy expenditure is negligible, it can accumulate over time, eventually becoming an important factor in weight loss or weight gain. The variability in work, leisure-time and household activities between individuals plays a fundamental role in NEAT differences⁴⁶⁻⁵⁰. Thus, the biggest advantage of NEAT is that the requirement to comply with purposeful exercise is low, and it has a higher rate of adherence over time. This was observed in a Chinese study that compared the prevalence of recommended exercise and NEAT in 32 005 adolescents; NEAT remained high over time and seemed easier to accumulate

than exercise in adolescents, regardless of age or gender⁵¹.

Daily additive unplanned or unstructured low intensity activities are associated with energy expenditure in excess of the RMR and account for significant thermogenesis and energy expenditure^{52,53}. The metabolic equivalent (MET) is a useful measurement to represent the intensity of physical activity and it is defined as the amount of oxygen uptake while sitting at rest. An oxygen uptake of 3.5 mL/kg/min is equal to the RMR and is considered to be equivalent to 1.0 MET⁵⁴. Sitting, standing and walking increase energy expenditure above resting levels by 5-10%, 10-20% and 100-200%, respectively⁵⁵⁻⁵⁸. Ainsworth et al. 2011⁵⁹ reported that walking inside is equal to only 2.0 METs, whereas walking with children is equivalent to 4.0 METs. Likewise, daily physical activity can span a wide range of intensity levels that at times are the same as structured exercise.

In fact, energy expenditure induced by NEAT is much larger than EAT when measured over the day^{60,61}. Levine et al., 1999⁶⁰ observed that long-term weight control may be easier to maintain by focusing less on EAT and more on increasing NEAT. They recruited 10 lean and 10 mildly obese sedentary volunteers and measured their postures, activities of daily living, and fidgeting for 10 days. Obese individuals were seated on average, two hours a day longer than lean people. If obese individuals adopted the NEAT-enhanced behaviors of their lean counterparts, they could expend an additional 350 kcal/day from these numerous small low grade activities and movements. Since this is equivalent to approximately 18 kg in a year, this may be an important factor in long-term weight control. Other studies have also demonstrated that obese individuals tend to have a lower level of NEAT than non-obese individuals, with more time spent engaging in sedentary activities (for example, laying down, sitting, watching television)⁶²⁻⁶⁴. Before increasing NEAT is proposed, the number of calories which need to be expended to prevent or treat obesity should be considered. According to Levine et al. 2007⁶¹, an additional energy expenditure of 280-350 kcal/day or 2000-2500 kcal/week through NEAT is required for weight loss. However, there is lack of evidence from randomized controlled studies to support whether strategies promoting NEAT are effective for obesity treatment, and there is limited research examining the thermogenic potential of fidgeting-like activities at very low workloads. In addition individual biological and environmental factors such as gender, age, body composition, and season can result in significant variation in NEAT. These four factors will therefore be discussed in more detail. 1) Gender is biologically determined but there are gender-specific environmental cues that influence NEAT. Although the United States report similar levels of PEE in adult men and women⁶⁵, in other countries, including Canada, England, and Australia, men tend to have a higher PEE than women⁶⁶. Gender may also influence NEAT in more subtle ways. For example, society and culture may require that women to do more housework and child care. 2) Previous studies have demonstrated an age-related de-

cline in NEAT⁶⁷. Harris et al. 2007⁶⁸ showed a substantial decrease in NEAT in elderly compared to younger individuals. Indeed, elderly people showed 29% less NEAT, and this result was mainly attributable to the NEAT subcomponent of ambulation. 3) Overweight individuals tend to have lower NEAT levels than their lean counterparts^{69,70}, and this is consistent across all ages, for both genders. It is not possible to ascertain whether the effects of body composition on NEAT are independent of weight³². 4) Limited data are available regarding differences in NEAT across the seasons; however, physical activity time is twice as high during the summer compared with the winter²⁶. These data were explained by the seasonal variation in occupation-associated NEAT⁷¹⁻⁷³. For example, there is greater emphasis on agricultural work or work in the construction industry during the summer. In addition, people do more outdoor activities in the summer. Therefore, further research is required to accurately measure both locomotor and household activities, and to further our understanding of NEAT in the different stages of life, from childhood to early and late adulthood. Since movement patterns during these unique periods differ, NEAT recommendations may be specific for each age group. However, at present, the general advice should be to encourage people to limit their sitting time whilst at home, at work, and using transport. Prolonged periods of sitting throughout the day should be interrupted by frequent transitions from sitting to standing and ambulation⁷⁴.

The metabolic health consequences of NEAT

Recently studies have shown that metabolic syndrome⁷⁵⁻⁷⁷, adiposity or weight gain^{45,78-80}, poor glucose management⁸¹ and type 2 diabetes risk^{82,83} may be directly related to sitting time and/or to a low level of NEAT that may be independent of EAT. Such findings suggest that sedentary behavior that does not increase energy expenditure substantially above resting levels, may contribute to disease risk separately from EAT, affecting health via independent pathways. Therefore, EAT does not counteract the negative effects of prolonged sitting. This is supported by a Canadian study which showed a dose-response relationship between sitting time and all-cause mortality and cardiovascular disease (CVD) mortality, independent of EAT. After adjusting for cofounders, they found a progressively higher risk of all-cause mortality and CVD mortality with higher levels of sitting time. Moreover, Hagger-Johnson et al.⁸⁴ analyzed data from the United Kingdom Women's Cohort Study (1999-2002) to investigate the association between fidgeting behaviors and all-cause mortality. They found that subjects in the low fidgeting group, sitting for ≥ 7 h/day (vs. < 5 h/day) experienced a 30% increase in risk of all-cause mortality. In contrast, in a large scale cohort study, Wen et al., 2011⁸⁵, found that individuals who performed low volume physical activity, defined as 15 min/day or 90 min/week, had a 14% reduced risk of all-cause mortality and a three year increase in life expectancy. Thus, in addition to EAT through moderate- to vigorous- intensity exercise, light- to moderate- intensity

daily NEAT should also be considered as an alternative and complimentary therapy regimen to improve metabolic health. Nevertheless, research in NEAT-related fields is in the early stage, and uncertainty still exists regarding which components of energy expenditure are altered in obese patients and patients with metabolic diseases. The documented effects of the different components of energy expenditure on TEE in obese patients and patients with metabolic diseases are inconsistent. Thus, it is necessary to accumulate evidence on the positive effects of EAT on the management of obesity and metabolic diseases.

NEAT quantification

At present, several methods are used to measure energy expenditure. Indirect and direct calorimetric and non-calorimetric methods are used for providing information on body posture and activity recognition. Questionnaires can provide qualitative information, such as the type and the purpose of physical activity, which differs to that provided by objective methods. However, questionnaires are unable to accurately measure the intensity of physical activity and energy expenditure. The NEAT score calculated using a questionnaire is subjective and may not always represent the true NEAT⁸⁶⁻⁸⁸. In contrast, instruments such as accelerometers will provide more accurate information on the intensity, duration, and frequency of PEE, mitigating the recall bias of self-report questionnaire data⁸⁹. Also, accelerometers in mobile phones and stand-alone devices with consumer applications enable precise and accurate measurement of daily physical activity⁹⁰. These are attractive strategies for encouraging the measurement of physical activity and they are easy to self-monitor and user-friendly⁹¹. Nonetheless, this method has the disadvantage that physical activity can only be measured when the mobile phone is on the body, and it does not accurately reflect actual energy expenditure in situations such as walking carrying objects, and walking uphill^{88,91}. Methods of measuring energy expenditure based on the concentration of oxygen and carbon dioxide are relatively accurate, however conventional indirect calorimetry equipment configurations either prohibit movement completely (for example, hood calorimetry), or involve the application of a mouthpiece and nose clip that may prevent normal body movements, (in this case breathing may differ to normal). The doubly labeled water (DLW) method is one of the best techniques for measuring TEE under free-living conditions, however, this method can only evaluate TEE and it cannot assess variations in the TEE components⁸³. Thus, separate measurements of NEAT are limited by the need to measure activity level using an accelerometer. The human metabolic chamber (HMC), is an indirect calorimetric method that can overcome these limitations and it can be used to measure all of the components of TEE, including RMR, DIT, EAT, and NEAT. This equipment also allows the precise measurement of energy expenditure with unrestricted movement and a rapid response time. Therefore, it is con-

sidered to be a valuable method in this research field.

CONCLUSION

In conclusion, NEAT is a highly variable component of daily TEE and a low level of NEAT is associated with obesity. NEAT enhances lifestyle, and variations in individual and environmental factors can significantly affect daily energy expenditure. Therefore, well designed longitudinal studies that focus on personal behavioral approaches and re-engineered environments to increase NEAT should be conducted in the future. A greater understanding of the amounts and intensities of NEAT needed to protect against obesity and metabolic diseases in the context of gender and age differences is required. Also, the feasibility of NEAT regarding the thermogenic potential of fidgeting-like activities at very low workloads for different groups (older, younger), in different settings (workplace, domestic, transit), needs to be explored. Finally, intervention trials are required to demonstrate whether significant increases in NEAT are associated with improvements in the relevant biomarkers.

ACKNOWLEDGEMENTS

This work was supported by the Ministry of Education of the Republic of Korea and the National Research Foundation of Korea (NRF-2016S1A5B8914314).

REFERENCES

1. Obesity and overweight Fact sheet N311 2016. *WHO*.
2. GBD 2015 Obesity Collaborators, Afshin A, Forouzanfar MH, Reitsma MB, Sur P, Estep K, Lee A, Marczak L, Mokdad AH, Moradi-Lakeh M, Naghavi M, Salama JS, Vos T, Abate KH, Abbafati C, Ahmed MB, Al-Aly Z, Alkerwi A, Al-Raddadi R, Amare AT, Amberbir A, Amegah AK, Amini E, Amrock SM, Anjana RM, Ärnlöv J, Asayesh H, Banerjee A, Barac A, Baye E, Bennett DA, Beyene AS, Biadgilign S, Biryukov S, Bjertness E, Boneya DJ, Campos-Nonato I, Carrero JJ, Cecilio P, Cercy K, Ciobanu LG, Cornaby L, Damtew SA, Dandona L, Dandona R, Dharmaratne SD, Duncan BB, Eshradi B, Esteghamati A, Feigin VL, Fernandes JC, Fürst T, Gebrehiwot TT, Gold A, Gona PN, Goto A, Habtewold TD, Hadush KT, Hafezi-Nejad N, Hay SI, Horino M, Islami F, Kamal R, Kasaeian A, Katikireddi SV, Kengne AP, Kesavachandran CN, Khader YS, Khang YH, Khubchandani J, Kim D, Kim YJ, Kinfu Y, Kosen S, Ku T, Defo BK, Kumar GA, Larson HJ, Leinsalu M, Liang X, Lim SS, Liu P, Lopez AD, Lozano R, Majeed A, Malekzadeh R, Malta DC, Mazidi M, McAlinden C, McGarvey ST, Mengistu DT, Mensah GA, Mensink GBM, Mezegebe HB, Mirrakhimov EM, Mueller UO, Noubiap JJ, Obermeyer CM, Ogbo FA, Owolabi MO, Patton GC, Pourmalek F, Qorbani M, Rafay A, Rai RK, Ranabhat CL, Reinig N, Safiri S, Salomon JA, Sanabria JR, Santos IS, Sartorius B, Sawhney M, Schmidhuber J, Schutte AE, Schmidt MI, Sepanlou SG, Shamsizadeh M, Sheikhbahaei S, Shin MJ, Shiri R, Shiue I, Roba HS, Silva DAS, Silverberg JI, Singh JA, Stranges S, Swaminathan S, Tabarés-Seisdedos R, Tadese F, Tedla BA, Tegegne BS, Terkawi AS, Thakur JS, Tonelli M, Topor-Madry R, Tyrovolas S, Ukwaja KN, Uthman OA, Vaezghasemi M, Vasankari T, Vlassov VV, Vollset SE, Weiderpass E, Werdecker A, Wesana J, Westerman R, Yano Y, Yonemoto N, Yonga G, Zaidi Z, Zenebe ZM, Zipkin B, Murray CJL. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. *NEJM*. 2017;377:13–27.
3. Weinstock M. The Facts About Obesity. *H&HN. American Hospital Association*. 2013.
4. Kanazawa M, Yoshiike N, Osaka T, Numba Y, Zimmet P, Inoue S. Criteria and classification of obesity in Japan and Asia-Oceania. *World Rev Nutr Diet*. 2005;94:1–12.
5. Grundy SM. Obesity, metabolic syndrome, and cardiovascular disease. *J Clin Endocrinol Metab*. 2004;89:2595–600.
6. Haslam DW, James WP. Obesity. *Lancet (Review)*. 2005;366:1197–209.
7. Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BW, Zitman FG. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Arch Gen Psychiatry*. 2010;67:220–9.
8. Dibaise JK, Foxx-Orenstein AE. Role of the gastroenterologist in managing obesity. *Expert Rev Gastroenterol Hepatol*. 2013;7:439–51.
9. Yazdi FT, Clee SM, Meyre D. Obesity genetics in mouse and human: back and forth, and back again. *PeerJ*. 2015;3:856.
10. Herrera BM, Keildson S, Lindgren CM. Genetics and epigenetics of obesity. *Maturitas*. 2011;69:41–9.
11. Burgio E, Lopomo A, Migliore L. Obesity and diabetes: from genetics to epigenetics. *Mol Biol Rep*. 2015;42:799–818.
12. Pigeyre M, Yazdi FT, Kaur Y, Meyre D. Recent progress in genetics, epigenetics and metagenomics unveils the pathophysiology of human obesity. *Clin Sci (Lond)*. 2016;130:943–86.
13. Andersen RE, Crespo CJ, Bartlett SJ, Cheskin LJ, Pratt M. Relationship of physical activity and television watching with body weight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. *JAMA*. 1998;279:938–42.
14. Gordon-Larsen P, Adair LS, Popkin BM. Ethnic differences in physical activity and inactivity patterns and overweight status. *Obes Res*. 2002;10:141–9.
15. Cutler GJ1, Flood A, Hannan P, Neumark-Sztainer D. Multiple sociodemographic and socioenvironmental characteristics are correlated with major patterns of dietary intake in adolescents. *J Am Diet Assoc*. 2011;111:230–40.
16. Kumanyika SK. Environmental influences on childhood obesity: ethnic and cultural influences in context. *Physiol Behav*. 2008;94:61–70.
17. Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, Gortmaker SL. The global obesity pandemic: shaped by global drivers and local environments. *Lancet*. 2011;378:804–14.
18. Bleich S, Cutler D, Murray C, Adams A. Why is the developed world obese?. *Annu Rev Public Health*. 2008;29:273–95.

19. Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]. *CMAJ*. 2007;176:1–13.
20. Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *BMJ*. 1995;311:437–39.
21. Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity. *Am J Med*. 1998;105:145–50.
22. Hill JO, Wyatt HR, Peters JC. Energy balance and obesity. *Circulation*. 2012;126:126–32.
23. Ford LE. Some consequences of body size. *Am J Physiol*. 1984;247:495–507.
24. Daan S, Masman D, Strijkstra A, Verhulst S. Intraspecific allometry of basal metabolic rate: relations with body size, temperature, composition, and circadian phase in the kestrel, *Falco tinnunculus*. *J Biol Rhythms*. 1989;4:267–83.
25. Psota T, Chen KY. Measuring energy expenditure in clinical populations: rewards and challenges. *Eur J Clin Nutr*. 2013;67:436–42.
26. Levine JA, Kotz CM. NEAT--non-exercise activity thermogenesis--egocentric & geocentric environmental factors vs. biological regulation. *Acta Physiol Scand*. 2005;184:309–18.
27. Kinabo JL, Durnin JV. Thermic effect of food in man: effect of meal composition, and energy content. *Br J Nutr*. 1990;64:37–44.
28. Bouchard C. The magnitude of the energy imbalance in obesity is generally underestimated. *Int J Obes (Lond)*. 2008;32:879–80.
29. Galgani J, Ravussin E. Energy metabolism, fuel selection and body weight regulation. *Int J Obes (Lond)*. 2008;32:109–19.
30. Donahoo WT, Levine JA, Melanson EL. Variability in energy expenditure and its components. *Curr Opin Clin Nutr Metab Care*. 2004;7:599–605.
31. Caspersen CJ, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep*. 1985;100:126–31.
32. Levine JA. Non-exercise activity thermogenesis (NEAT). *Nutr Rev*. 2004;62:82–97.
33. Kumahara H, Tanaka H, Schutz Y. Daily physical activity assessment: what is the importance of upper limb movements vs whole body movements? *Int J Obes Relat Metab Disord*. 2004;28:1105–10.
34. Levine JA, Vander Weg MW, Hill JO, Klesges RC. Non-exercise activity thermogenesis. *Arterioscler Thromb Vasc Biol*. 2006;26:729–36.
35. Ness-Abramof R, Apovian CM. Diet modification for treatment and prevention of obesity. *Endocrine*. 2006;29:5–9.
36. Owen N, Sparling PB, Healy GN, Dunstan DW, Matthews CE. Sedentary Behavior: Emerging Evidence for a New Health Risk. *Mayo Clin Proc*. 2010;85:113841.
37. Roberts SB. Abnormalities of energy expenditure and the development of obesity. *Obes Res*. 1995;3:155–63.
38. Jakicic JM, Egan CM, Fabricatore AN, Gaussoin SA, Glasser SP, Hesson LA, Knowler WC, Lang W, Regensteiner JG, Ribisl PM, Ryan DH, Look AHEAD Research Group. Four-year change in cardiorespiratory fitness and influence on glycemic control in adults with type 2 diabetes in a randomized trial: the Look AHEAD Trial. *Diabetes Care*. 2013;36:1297–303.
39. Ryan D, Heaner M. Guidelines (2013) for managing overweight and obesity in adults. Preface to the full report. *Obesity (Silver Spring)*. 2014;22:1–3.
40. National Institute for Health and Clinical Excellence: Guidance. Obesity: identification, assessment and management of overweight and obesity in children, young people and adults: partial update of CG43. London: National Institute for Health and Care Excellence (UK). 2014.
41. Niebauer J, Hambrecht R, Velich T, Hauer K, Marburger C, Kalberer B, Weiss C, von Hodenberg E, Schlierf G, Schuler G, Zimmermann R, Kubler W. Attenuated progression of coronary artery disease after 6 years of multifactorial risk intervention: role of physical exercise. *Circulation*. 1997;96:2534–41.
42. Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW, King AC, Kriska A, Leon AS, Marcus BH, Jr Paffenbarger RS, Patrick K, Pollock ML, Rippe JM, Salis J, Wilmore JH. Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA*. 1995;273:402–7.
43. Segal KR, Pisunyer FX. Exercise and Obesity. *Med Clin North Am*. 1989;73:217–36.
44. Levine JA. Nonexercise activity thermogenesis (NEAT): environment and biology. *Am J Physiol Endocrinol Metab*. 2004;286:675–85.
45. Levine JA, Lanningham-Foster LM, McCrady SK, Krizan AC, Olson LR, Kane PH, Jensen MD, Clark MM: Interindividual variation in posture allocation: possible role in human obesity. *Science*. 2005;307:584–6.
46. Owen N, Healy GN, Matthews CE, Dunstan DW. Too Much Sitting: The Population-Health Science of Sedentary Behavior. *Exerc Sport Sci Rev*. 2010;8:105–13.
47. Church TS, Thomas DM, Tudor-Locke C, Katzmarzyk PT, Earnest CP, Rodarte RQ, Martin CK, Blair SN, Bouchard C. Trends over 5 decades in U.S. occupation-related physical activity and their associations with obesity. *PLoS One*. 2011; 6:19657.
48. Archer E, Shook RP, Thomas DM, Church TS, Katzmarzyk PT, Hébert JR, McIver KL, Hand GA, Lavie CJ, Blair SN. 45-Year trends in women's use of time and household management energy expenditure. *PLoS One*. 2013;8:56620.
49. Steffen LM, Arnett DK, Blackburn H, Shah G, Armstrong C, Luepker RV, Jacobs DR Jr. Population trends in leisure-time physical activity: Minnesota Heart Survey, 1980- 2000. *Med Sci Sports Exerc*. 2006;38:1716–23.
50. De Vera MA, Ratzlaff C, Doerfling P, Kopec J. Reliability and validity of an internet-based questionnaire measuring lifetime physical activity. *Am J Epidemiol*. 2010;172:1190–8.
51. Mak KK, Ho SY, Lo WS, McManus AM, Lam TH. Prevalence of exercise and non-exercise physical activity in Chinese adolescents. *Int J Behav Nutr Phys Act*. 2011;20:3.
52. Dauncey MJ. Activity and energy expenditure. *Can J Physiol Pharmacol*. 1990;68:17–27.
53. Levine JA, Schlessner SJ, Jensen MD. Energy expenditure of nonexercise activity. *Am J Clin Nutr*. 2000 72:1451–4.
54. Balducci S, Sacchetti M, Haxhi J, Orlando G, D'Errico V, Fal-

- lucca S, Menini S, Pugliese G. Physical exercise as therapy for type 2 diabetes mellitus. *Diabetes Metab Res Rev.* 2014; 30:13–23
55. Diaz EO, Prentice AM, Goldberg GR, Murgatroyd PR, Coward WA. Metabolic response to experimental overfeeding in lean and overweight healthy volunteers. *Am J Clin Nutr.* 1992;56:641–55.
 56. Norgan NG, Durnin JV. The effect of 6 weeks of overfeeding on the body weight, body composition, and energy metabolism of young men. *Am J Clin Nutr.* 1980;33:978–88.
 57. Bouten CV, Westerterp KR, Verduin M, Janssen JD. Assessment of energy expenditure for physical activity using a triaxial accelerometer. *Med Sci Sports Exerc.* 1994;26:1516–23
 58. Goldberg GR, Prentice AM, Davies HL, Murgatroyd PR. Residual effect of graded levels of exercise on metabolic rate. *Eur J Clin Nutr.* 1990;44:99–105.
 59. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Bassett DR, Tudor-Locke C, Greer JL, Vezina J, Whitt-Glover MC, Leon AS. 2011 Compendium of Physical Activities: a second update of codes and MET values. *Med Sci Sports Exerc.* 2011;43:1575–81.
 60. Levine JA, Eberhardt NL, Jensen MD. Role of nonexercise activity thermogenesis in resistance to fat gain in humans. *Science.* 1999;283:212–4.
 61. Levine JA. Nonexercise activity thermogenesis – liberating the life-force. *J Intern Med.* 2007;262:273–87.
 62. Elbelt U, Schuetz T, Hoffmann I, Pirlich M, Strasburger CJ, Lochs H. Differences of energy expenditure and physical activity patterns in subjects with various degrees of obesity. *Clin Nutr.* 2010;29:766–72.
 63. DeLany JP, Kelley DE, Hames KC, Jakicic JM, Goodpaster BH. High energy expenditure masks low physical activity in obesity. *Int J Obes (Lond).* 2013;37:1006–11.
 64. Johannsen DL, Welk GJ, Sharp RL, Flakoll PJ. Differences in daily energy expenditure in lean and obese women: the role of posture allocation. *Obesity (Silver Spring).* 2008;16:34–9.
 65. Yusuf HR, Croft JB, Giles WH, Anda RF, Casper ML, Caspersen CJ, Jones DA. Leisure-time physical activity among older adults. United States, 1990. *Arch Intern Med.* 1996;156:1321–6.
 66. Caspersen CJ, Merritt RK. Physical activity trends among 26 states, 1986–1990. *Med Sci Sports Exerc.* 1995 27:713–20.
 67. Westerterp KR. Daily physical activity and ageing. *Curr Opin Clin Nutr Metab Care.* 2000;3:485–8.
 68. Harris AM, Lanningham-Foster LM, McCrady SK, Levine JA. Nonexercise movement in elderly compared with young people. *Am J Physiol Endocrinol Metab.* 2007;292:1207–12.
 69. Pratt M, Macera CA, Blanton C. Levels of physical activity and inactivity in children and adults in the United States: current evidence and research issues. *Med Sci Sports Exerc.* 1999;31:526–33.
 70. Livingstone B. Epidemiology of childhood obesity in Europe. *Eur J Pediatr.* 2000;159:14–34.
 71. Katzmarzyk PT, Craig CL, Bouchard C. Original article underweight, overweight and obesity: relationships with mortality in the 13-year follow-up of the Canada Fitness Survey. *J Clin Epidemiol.* 2001;54:916–20.
 72. Singh J, Prentice AM, Diaz E, Coward WA, Ashford J, Sawyer M, Whitehead RG. Energy expenditure of Gambian women during peak agricultural activity measured by the doubly-labelled water method. *Br J Nutr.* 1989; 62:315–29.
 73. Pastore G, Branca F, Demissie T, Ferro-Luzzi A. Seasonal energy stress in an Ethiopian rural community: an analysis of the impact at the household level. *Eur J Clin Nutr.* 1993;47:851–62.
 74. Villablanca PA, Alegria JR, Mookadam F, Holmes DR Jr, Wright RS, Levine JA. Nonexercise activity thermogenesis in obesity management. *Mayo Clin Proc.* 2015;90:509–19.
 75. Dunstan DW, Salmon J, Owen N, Armstrong T, Zimmet PZ, Welborn TA, Cameron AJ, Dwyer T, Jolley D, Shaw JE; AusDiab Steering Committee. Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia.* 2005;48:2254–61.
 76. Bertrais S, Beyeme-Ondoua JP, Czernichow S, Galan P, Hercberg S, Oppert JM. Sedentary behaviors, physical activity, and metabolic syndrome in middle-aged French subjects. *Obes Res.* 2005;13:936–44.
 77. Gao X, Nelson ME, Tucker KL. Television viewing is associated with prevalence of metabolic syndrome in Hispanic elders. *Diabetes Care.* 2007;30:694–700.
 78. Owen N, Leslie E, Salmon J, Fotheringham MJ. Environmental determinants of physical activity and sedentary behavior. *Exerc Sport Sci Rev.* 2000;28:153–8.
 79. Brown WJ, Williams L, Ford JH, Ball K, Dobson AJ. Identifying the energy gap: magnitude and determinants of 5-year weight gain in midage women. *Obes Res.* 2005;13:1431–41.
 80. Cameron AJ, Welborn TA, Zimmet PZ, Dunstan DW, Owen N, Salmon J, Dalton M, Jolley D, Shaw JE. Overweight and obesity in Australia: the 1999–2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Med J Aust.* 2003;178:427–32.
 81. Margeirsdottir HD, Larsen JR, Brunborg C, Sandvik L, Dahl-Jørgensen K, the Norwegian Study Group for Childhood Diabetes. Strong association between time watching TV and blood glucose control in children and adolescents with type 1 diabetes. *Diabetes Care.* 2007;30:1567–70.
 82. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and TV watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med.* 2001; 161:1542–8.
 83. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA.* 2003; 289:1785–91.
 84. Hagger-Johnson G, Gow AJ, Burley V, Greenwood D, Cade JE. Sitting Time, Fidgeting, and All-Cause Mortality in the UK Women's Cohort Study. *Am J Prev Med.* 2016;50:154–60.
 85. Wen CP, Wai JP, Tsai MK, Yang YC, Cheng TY, Lee MC, Chan HT, Tsao CK, Tsai SP, Wu X. Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study. *Lancet.* 2011;378:1244–53
 86. Colbert LH, Matthews CE, Havighurst TC, Kim K, Schoeller DA. Comparative validity of physical activity measures in older adults. *Med Sci Sports Exerc.* 2011;43:867–76.
 87. Colbert LH, Schoeller DA. Expending our physical activity (measurement) budget wisely. *J Appl Physiol.* 2011;111:606–7.
 88. Levine JA. Measurement of energy expenditure. *Public*

- Health Nutr.* 2005;8:1123–32.
89. Manohar CU, McCrady SK, Fujiki Y, Pavlidis IT, Levine JA. Evaluation of the accuracy of a triaxial accelerometer embedded into a cell phone platform for measuring physical activity. *J Obes Weight Loss Ther.* 2012;1:3309.
90. Bravata DM, Smith-Spangler C, Sundaram V, Gienger AL, Lin N, Lewis R, Stave CD, Olkin I, Sirard JR. Using pedometers to increase physical activity and improve health: a systematic review. *JAMA.* 2007;298:2296-304.
91. Pedisic Z, Bauman A. Accelerometer-based measures in physical activity surveillance: current practices and issues. *Br J Sports Med.* 2015;49:219-23.