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Behavioral Contributions to the Pathogenesis of Type 2 Diabetes

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Abstract

Behavioral Contributions to the pathogenesis of prediabetes and Type 2 diabetes (T2D) include lifestyle behaviors including dietary intake, exercise, sedentariness, sleep, and stress. The purpose of this paper is to review evidence for the metabolic pathways by which the behavior is linked to T2D. Evidence for interventions which change each of the lifestyle behaviors is discussed. The article will close with a brief discussion on how new technologies may provide opportunities to better understand relationships between moment-to-moment fluctuations in behaviors and diabetes pathogenesis, as well as provide opportunities to personalize and adapt interventions to achieve successful behavior change and maintenance of that change. Especially promising are new technologies which assist in tracking lifestyle behaviors along with clinical and metabolic outcomes.

Keywords

Type 2 diabetes; behavior; pathogenesis; technology

Introduction

Type 2 diabetes (T2D) has reached epidemic proportions in the United States [1] and globally [2]. According to the Centers for Disease Control, 11.8% of men and 10.8% of women over 20 years of age had T2D in the United States in 2010. Compared to non-Hispanic white adults, the risk of diagnosed diabetes was 18% higher among Asian Americans, 66% higher among Hispanics, and 77% higher among non-Hispanic Blacks [3].

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Conflict of Interest

Donna Spruijt-Metz, Lauren Cook, Gillian A. O'Reilly, Kathleen A. Page, and Charlene Quinn declare that they have no conflict of interest.

Compliance with Ethics Guidelines

Human and Animal Rights and Informed Consent

This article does not contain any studies with human or animal subjects performed by any of the authors.

Not only is T2D a disease of the present, it is perhaps *the* disease of the future. On a global scale, it is estimated that diabetes (type 1 and 2 taken together) will see a 54% increase by 2030 [4]. In the United States, the lifetime risk for developing diabetes for children born in the year 2000 has been estimated at 32.8% for males and 38.5% for females. Lifetime risk is considerably higher for Hispanic and non-Hispanic black populations [5].

In conjunction with the epidemic of T2D, the prevalence of prediabetes has skyrocketed. Prediabetes, also referred to as impaired glucose tolerance (IGT) or impaired fasting glucose (IFG), depending on the test used for diagnosis, entails blood sugar levels that are higher than normal, but not high enough to be diagnosed with diabetes. Data from the National Health and Nutrition Examination Surveys (NHANES) showed that in 2010, 36.2% of US adults aged 20 years and older had prediabetes [6]. Children are also experiencing increasing levels of prediabetes. NHANES data from 2005–2006 showed that 16.1% of children between 12–19 years of age had prediabetes, with Hispanic, non-Hispanic Black, obese and older children showing considerably higher prevalence [7]. One study conducted in a representative sample of US adolescents aged 12–19 in 2007–2008 found that 23% had either prediabetes or diabetes [8]. Without making lasting positive changes in obesity-related behaviors, 15% to 30% of people with prediabetes will develop T2D within 5 years [9].

Poor health-related behaviors during pregnancy can have important long-term effects on the development of prediabetes and diabetes in the child. For example, mounting evidence indicates that poor maternal diet [10] and diabetes during pregnancy are contributing in a feed-forward fashion to the epidemics of obesity and T2D.[11] The effects of diabetes during pregnancy can be thought of a vicious cycle. Offspring exposed to poor maternal diet or maternal diabetes *in utero* are at higher risk of developing obesity and type 2 diabetes early in life. This leads to a greater number of women of child-bearing age who develop diabetes before or during pregnancy, which in turn perpetuates the cycle.

Behavioral contributions to prediabetes and T2D

Prediabetes and T2D are profoundly impacted by lifestyle behaviors [12]including dietary intake, exercise, sedentariness, sleep, and stress.. Many of these behaviors, such as sugar consumption, contribute to poor glucose regulation, regardless of obesity levels (see for instance [13]). This review will address five lifestyle behaviors that have strong evidence base for a causal link with T2D: diet, physical activity, sedentary behavior, sleep and stress. Each section will provide the evidence for the link between the behavior and T2D, the metabolic pathways by which the behavior is linked to T2D, and some information on changing that behavior. The article will close with a brief discussion on how new technologies may provide opportunities to better understand relationships between moment-to-moment fluctuations in behaviors and diabetes pathogenesis, as well as provide opportunities to personalize and adapt interventions to achieve successful behavior change and maintenance of that change.

Dietary intake and pathogenesis of T2D

A comprehensive review of all dietary intakes that are related to T2D is far beyond the scope of this brief review, therefore we focus on two major ‘culprits’ that have been

identified in current literature. Fiber consumption (in fruit, vegetables and whole grains) has been shown to be protective against prediabetes and diabetes. Sugar intake has been directly related to increased risk for T2D, and we focus on consumption of sugar sweetened beverages here.

Fiber and High-Fiber Foods

A study of 2909 healthy adults found an inverse association between dietary fiber measured by food frequency screener and both fasting insulin and 2-hour postprandial insulin. [14] Similarly, a crossover trial with individuals with T2D found that following a high-fiber diet resulted in a 10% decrease in 24-hour plasma glucose and insulin concentrations after six weeks, compared to a diet with moderate fiber composition. [15] These findings are echoed in studies of individual food items, and several large cohort studies have demonstrated a decreased relative risk of T2D with higher intake of whole grains, reported by food frequency screeners. [16–18] Fruit and vegetable (FV) intake has also been demonstrated to contribute to a decreased risk for T2D. [19, 20] Recently, studies have focused on the specific FV consumed, indicating that a greater variety of FV [19] and greater amounts of green leafy vegetables [21] are protective against diabetes risk.

The mechanism through which fibrous foods act to reduce T2D risk is not completely understood. [22, 23] Two prominent pathways through which fiber acts are via slowing gastric emptying and macronutrient absorption, [24] and by increasing satiety following meals to limit additional caloric intake. [25] Furthermore, polyphenols found in plant foods may also mitigate diabetes risk by altering sucrose breakdown and inhibiting glucose and fructose transport. [26]

Sugar-Sweetened Beverages

Sugar-sweetened beverage (SSB) consumption has dramatically risen in the last half century, [27] which is concerning given implication of SSB intake on the risk of T2D. A large cohort study revealed over 80% increase in T2D risk for women who drank one SSB per day, relative to women who had less than one drink per month; an association which was still significant after controlling for BMI.[28] A meta-analysis similarly found a 26% increase in diabetes risk comparing the aforementioned intake groups. [29]

Surprisingly, there is limited data on the impact of total sugars on T2D risk, and these studies do not show strong evidence of a relationship (of three studies identified in a recent review, two found no association, and one found an unexpected inverse association).[30] This may be due to the significant presence of high-fructose corn syrup (HFCS) in SSB. While glucose enters cells by a transport mechanism (Glut-4) that is insulin dependent in most tissues, and helps to provide “satiety” signals to the brain, fructose provides no such signal. Fructose enters cells via a Glut-5 transporter that does not depend on insulin. This transporter is absent from pancreatic beta cells and the brain, suggesting that the entry of fructose into these tissues is quite limited. Therefore, glucose provides “satiety” signals to the brain while fructose cannot provide these signals. In this manner, fructose alters regulatory pathways contributing to obesity and co-morbidities. [31]

Several major pathways through which SSB may alter T2D risk have been studied, including one where SSB consumption is posited to limit satiety feedback mechanisms, leading to weight gain.[31, 32] Also, the easily absorbable nature of carbohydrates present in SSBs increases postprandial insulin response and ultimately insulin resistance.[33] Furthermore, the high concentration of fructose consumed in SSBs promotes hepatic *de novo* lipogenesis, which can also contribute to the development of insulin resistance.[34]

Changing Dietary Behaviors

Educational interventions have long been the most popular strategy for improving dietary behaviors, yet new modifications to this approach show promise to improve reach and impact. Using new technologies such as smart phones to deliver real-time, personalized interventions may be especially transformative because of the opportunities for accessible self-monitoring and adaptive feedback.[35, 36] The evidence base on this approach is rapidly growing, and preliminary studies show that technology-based interventions are equally, [37] or even more, effective at changing dietary habits than traditional in-person or paper formats. [38, 39]

Garden- and cooking-based nutrition interventions with children have also grown in popularity over recent years [40–42], and although there is a theoretical benefit to these approaches (increased exposure to unfamiliar vegetables, focus on origins of foods, hands on participation), there is little evidence to date that these interventions are more effective than tradition models.

Taxation has recently been explored as an option to limit SSB, and economic principals suggest that a higher cost of these drinks would limit purchases, yet we cannot know the definitive impact of policy until implemented. [43] A major limitation to this approach is the lack of public support for a SSB tax, [44] and more advanced strategies will be needed for policy to be successful in limiting SSB intake.

Physical Activity

Physical activity has been shown to have a protective effect on T2D risk, and lack of physical activity has been shown to increase T2D risk. Indirect demonstration of this relationship is provided by the fact that shifts from traditional to modern lifestyles over the past century, characterized by a decrease in habitual physical activity, have been accompanied by a rise in the prevalence of T2D.[45] Beyond these more broad associations, large cross-sectional and retrospective studies have shown that the prevalence of T2D among people who regularly exercise is lower than among people who do not regularly exercise.[46] Results from several large prospective studies including the Women's Health Study, the Nurses' Health Study, and the Health Professionals Follow-up Study have shown that in both men and women, regular moderate exercise (such as walking) reduces the risk for T2D by as much as 34%.[46] Habitual lack of physical activity appears to have a detrimental impact on glucose tolerance, [45] as studies of non-diabetic populations have shown that individuals who are overall less physically active have higher post-challenge blood glucose levels than individuals who are more physically active.[47–50] Results from physical activity interventions for the prevention of T2D provide perhaps the most direct

evidence of this association, demonstrating that increased physical activity significantly reduces the risk for or delays the onset of T2D, even among individuals with impaired glucose tolerance, compared to control conditions.[51, 52]

The mechanisms underlying the protective effects of physical activity on T2D are not fully understood. However, compliance with recommended physical activity guidelines is known to help prevent weight gain, [46] an important risk factor for T2D.[53] The benefits of physical activity may not only be due to weight regulation, since evidence indicates that risk for T2D is reduced with increased physical activity despite adiposity levels.[46, 54] This may be explained by the short and long-term metabolic benefits conferred by physical activity. Studies have also shown that bouts of exercise have an impact of lowering blood glucose levels which extends to the post-exercise period.[55] Physical activity is known to enhance insulin sensitivity, thereby improving glucose tolerance.[46, 56]

Changing physical activity behavior

Recent studies have indicated that most of the U.S. population does not meet the recommendation for 150 minutes per week of moderate-intensity [57] or 75 minutes per week of vigorous-intensity [57] physical activity.[58, 59] Yet, health risks associated with low levels of physical activity can be mitigated with as little as 30 minutes of moderate-intensity activity per day.[46] It is therefore evident that physical activity is one modifiable health behavior that can delay or protect against the onset of T2D. However, physical activity behavior change adoption and maintenance are not easy to promote, as evidenced by inconsistent success [60] and lack of scalability [61] typical of many physical activity interventions. There are factors that can be targeted that have been shown to improve physical activity compliance. For instance, increased social support from friends and family has been shown to improve enjoyment of [62] and adherence to [63] physical activity programs among women. Additionally, interventions that target motivational readiness for physical activity behavior change have been shown to improve behavior change adoption. [64] [65] There is also evidence that tailored interventions can improve behavior change. [66] The task of increasing physical activity has proven difficult, particularly in developed societies where both work and play are dominated by opportunities for sedentary activities. Novel interventions that have shown promise include active gaming [67], socially networked games [68], and mobile health interventions [69] that combine real-time monitoring through wearable sensors and personalized, adaptive feedback through mobile phones [36].

Sedentary Behavior

Sedentary behavior pertains to activities that do no raise energy expenditure above resting levels (defined as 1.0–1.5 METs).[70, 71] Sedentary behavior does not merely represent a lack of physical activity, but is instead a distinct set of behaviors that carries its own risks for chronic diseases, including T2D.[72–74] A meta-analysis of twenty-three studies that examined the relationship between sedentary behavior and T2D concluded that, compared to active individuals, sedentary individuals have a 1.31–1.45 increase in risk for developing the disease.[75] Sedentary behavior, such as TV watching, is known to have a detrimental impact on metabolic health. This is indicated by studies that have found an association

between sedentary behaviors and abnormal glucose metabolism.[50] Additionally, single bouts of prolonged sedentary behavior have been shown to lead to decreased insulin sensitivity.[76, 77] Moreover, sedentary behavior may be related to risk for T2D independent of physical activity levels. Recent evidence indicates that even if individuals meet recommended physical activity guidelines, excessive amounts of time spent in sedentary behavior can have detrimental effects on metabolic health.[71, 73] Studies have found detrimental dose-response relationships between number of hours of TV watched per week and 2-hour plasma glucose as well as fasting plasma glucose levels in healthy adults, [74] even among those who reported at least 150 minutes/week of moderate-to-vigorous physical activity.[71] This evidence indicates that sedentary behavior is a distinct risk factor for T2D.

Recent studies of “inactivity physiology” have demonstrated that the mechanisms underlying the association between sedentary behavior and T2D risk may differ from those for physical activity.[73] Muscle contraction is integral to clearing glucose from the blood, which may explain why sedentary behavior (the absence of muscle movement) is associated with increases in fasting and 2-h plasma glucose levels that have been observed after bouts of sedentary behavior.[78] Additionally, results from animal studies have suggested that the impact of sedentary time on lipoprotein lipase may be the underlying mechanism for the influence of sedentary behavior on metabolic health.[74] The activity of lipoprotein lipase, an enzyme that is critical for the hydrolysis of triglycerides in lipoproteins, has been shown to decrease a short time after inactivity.[79] The effects of this loss of lipoprotein lipase activity has been suggested to cascade to a detrimental impact on glucose homeostasis.[74]

Changing sedentary behavior

Similar to physical activity, there is a lack of consensus in the literature regarding how to successfully change sedentary behavior and maintain that change over time.[80] Interventions that have aimed to decrease sedentary behavior by increasing physical activity have had mixed results. For example, an intervention by Sevick et al., 2000 compared cognitive-behavior modification techniques for physical activity behavior change to a structured exercise program and found that participants in both conditions maintained increased physical activity at 24 months after the intervention.[81] This suggests that both structured exercise programs and programs that employ behavior modification strategies can decrease sedentary behavior and increase physical activity.[81] However, a study conducted by Lee et al., 2003, that aimed to change physical activity in sedentary adults found that sedentary behaviors persisted despite increased physical activity.[82] Furthermore, there is a lack of interventions for adults to decrease sedentary behaviors common in the workplace, at home and during transportation.[83] Literature is emerging on the metabolic benefits of taking breaks in sedentary time[80], also in people with early stage T2D.[84]

Sleep

Poor sleeping habits have been investigated as contributors to T2D risk. Evidence from these studies indicates that both short-duration (<6 hours) and long-duration (>9 hours) sleep are associated with an increase in the risk for T2D.[85–87] In addition to sleep duration, frequent difficulty initiating sleep and maintaining sleep have been shown to be risks for

late-onset T2D.[85] The association between sleep habits and diabetes may be due to the adverse impact on blood glucose regulation and insulin after decreased sleep duration that has been found in laboratory sleep studies.[88–90] Additionally, epidemiologic studies have demonstrated that poor sleep duration and quality predict high $HbA1_c$, which is a marker for overall glycemic control.[88] Although sleep duration and quality can be determined by both behavioral and medical factors, behavioral causes of poor sleep appear to be modifiable targets for behavioral interventions for T2D prevention.

The slow-wave sleep phase may have a particularly important role for glycemic control. The onset of the slow-wave sleep phase occurs simultaneously with hormonal changes that affect glucose metabolism, suggesting that this phase of sleep may play a role in maintaining normal glucose regulation.[91] Sleep quality may be linked to T2D risk through the neuroendocrine regulation of appetite.[92, 90] Poor sleep quality leads to changes in circulating leptin and ghrelin levels, hormones that play a part in regulating hunger and satiety. Changes in the circulating levels of these hormones are associated with increased hunger and appetite during waking hours, which can in the long-term lead to weight gain and impaired glycemic control.[92, 90]

Changing poor sleeping behaviors

Evidence from intervention studies indicates that behavioral changes can improve sleep issues. A meta-analysis of 23 studies found that behavioral intervention strategies, including cognitive-behavioral treatments and relaxation therapy, have positive impacts on sleep onset, maintenance, and quality.[93] Additionally, there is evidence that improved sleep outcomes are maintained over a longer period of time after behavioral treatments that target stimulus control, sleep restriction, and sleep hygiene, compared to pharmacological treatments.[94, 95] Exercise has also been shown to improve sleep outcomes in advanced age individuals.[96] This evidence indicates that sleep is a modifiable behavioral risk factor for T2D.

Stress

Stress appears to be an important consideration for T2D risk, evidenced by a prospective cohort study indicating that perceived permanent stress resulted in a 45% increase in risk for T2D (relative to those who reported no stress), even after adjusting for typical risk factors including socio-economic status.[97] However, other reports, such as a review and meta-analysis indicating no link between work-related stress and T2D, [98] appear to provide a contrary evaluation. The discrepancy in these data could be attributable to the distinction between actual and perceived stress, with the literature suggesting that individual-level factors related to stress perception, such as coping skills, may be more important predictors than actual circumstances. For example, an assessment of burnout (measured by exhaustion, fatigue and weariness) and T2D indicates that burnout is associated with an 84% increased risk for T2D.[99] Also, a review of psychosocial predictors for T2D identified depression, general emotional stress, anxiety, sleeping problems and hostility as key risk factors.[100]

Stressful conditions stimulate the hypothalamic-pituitary-adrenal (HPA) axis to produce cortisol (the primary hormone responsible for the physiologic stress response), which

induces hepatic insulin resistance and decreased insulin secretion.[101, 102] Chronic overstress may also function to negatively impact HPA axis regulation.[103] HPA axis dysregulation is problematic in that it is strongly implicated in the development of T2D, likely through resultant increased visceral adiposity.[102] Furthermore, stress may also increase T2D risk by altering food intake behaviors, especially by increasing cravings for and consumption of foods higher in fat and sugar, subsequently increasing postprandial insulin response and obesity risk.[104]

Changing the Stress Response

Physical activity has repeatedly been shown to reduce stress [105], however as evidenced above, changing people's physical activity levels can be challenging. One of the most popular intervention mechanisms to reduce stress is through meditation and mindfulness coaching. Mindfulness-based stress reduction (MBSR) works by encouraging participants to be more attentive to their thoughts and experiences, leading to a greater sense of control and stronger coping.[106] A review and meta-analysis of MBSR interventions indicates that this approach is helpful for a wide range of conditions.[106] To our knowledge there have not been any MBSR interventions for diabetes prevention, but programs targeting obesity and eating behaviors have been shown to be effective to reduce food cravings and have a limited positive effect on body weight.[107–109] These approaches may be improved through the use of mobile technology to deliver intervention programs, [110] which would not only improve program reach, but also provide the opportunity to intervene at specific targeted times.

Conclusion

Preventing diabetes and preventing the progression to diabetes: Why is this one so hard to conquer?

T2D pathogenesis and behavior is a marathon, not a sprint. Early clinical trials documented the benefit of some lifestyle behaviors in preventing or delaying diabetes [111, 112]. Nonetheless, there has been minimal overall, long-term gain in improvements in obesity and diabetes-related health behaviors. The initiation and maintenance of lifestyle behavior changes is extremely difficult, even for the highly motivated. Even when interventions succeed in controlled clinical trials, maintenance of behavior change is the exception rather than the norm, with the vast majority of participants relapsing, regaining weight, and/or returning to old dietary and exercise habits [113].

The behaviors related to T2D are also interrelated. For instance, poor sleep alters circulating leptin and ghrelin levels [114], which in turn can lead to higher caloric intake. Poor sleep is exacerbated by increased stress [115], which is related to increased secretion of cortisol and interleukin-6, elevated insulin concentrations, development of central obesity, insulin resistance, and the metabolic syndrome [116]. Stress is furthermore independently related to intake of calorically dense foods. There is evidence for several mechanisms that might explain this relationship, including evidence that sweet or highly palatable, energy dense foods inhibit the stressor-induced glucocorticoid effects on the brain [117]. Conversely, increasing physical activity can alleviate stress, partially through promoting production of

neurohormones such as norepinephrine, which is associated with improved cognitive function and elevated mood [118]. There is evidence that physical activity also helps to improve sleep. Several underlying mechanisms for this effect have been suggested [119]. Better understanding of pathogenesis and behavioral interrelationships may lead us to consider whether behavior interventions should target single behaviors or multiple, interrelated behaviors [120]. However, past the need to change behaviors, there is a need to develop good, lifelong habits. Short-term behavior changes are often followed by relapse [121], and a major goal of future interventions should be sustained behavior change, i.e. maintenance of healthy behaviors throughout life.

Mobile health interventions as well as mobile health tracking technology may provide opportunities to better understand the relationship of behaviors to diabetes pathogenesis and clinical measures (blood glucose, cholesterol, blood pressure). Behaviors, as well as health indicators, fluctuate throughout the day, across days and weeks and years. How are these fluctuations interrelated, and how do they relate to health outcomes? Understanding these relationships will improve interventions and contribute to behavior change and maintenance. To accomplish this, a strong, empirical theoretical basis for understanding health-related behaviors is required. The current body of literature on behavioral theory enumerates influences on behavior on many levels, including metabolic, demographic, psychological, social, environmental, socio-economic, and policy-level [122–124]. However, these theories do not take into account the moment-to-moment variability in behaviors and the real-time influences that drive behavior changes. New technologies, including wearable and deployable sensors, global positioning systems and smartphone technologies can provide rich, contextualized and temporally dense data that will help us to develop empirically grounded momentary models of health-related behaviors. [125–127] The combination of momentary models of behavior and wearable, wireless technologies will enable a new generation of interventions that can be tailored to changing behavior ‘on the fly’.[128] New sensor technologies are enabling ongoing tracking of calories, sleep, stress, blood glucose, exercise, lipid levels and many other T2D related behaviors and conditions that can be used for self-monitoring, to help health care providers track their patients, and to provide real-time and personalized interventions. To help people make lasting behavioral changes, interventions will need to be compelling, engaging, and seamlessly integrated into daily life.

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References

1. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. JAMA. 2001; 286(10):1195–200. [PubMed: 11559264]
2. Chen L, Magliano DJ, Zimmet PZ. The worldwide epidemiology of type 2 diabetes mellitus - present and future perspectives. Nature Reviews Endocrinology. 2011; 8(4):228–36.

3. Centers for Disease Control and Prevention. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2011.
4. Shaw J, Sicree R, Zimmet P. Global estimates of the prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract. 2010; 87(1):4–14. [PubMed: 19896746]
5. Narayan KM, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. Lifetime risk for diabetes mellitus in the United States. Jama. 2003; 290(14):1884–90. [PubMed: 14532317]
6. Bullard KM, Saydah SH, Imperatore G, Cowie CC, Gregg EW, Geiss LS, et al. Secular Changes in US Prediabetes Prevalence Defined by Hemoglobin A1c and Fasting Plasma Glucose National Health and Nutrition Examination Surveys, 1999–2010. Diabetes Care. 2013
7. Li C, Ford ES, Zhao G, Mokdad AH. Prevalence of pre-diabetes and its association with clustering of cardiometabolic risk factors and hyperinsulinemia among US adolescents national health and nutrition examination survey 2005–2006. Diabetes Care. 2009; 32(2):342–7. [PubMed: 18957533]
8. May AL, Kuklina EV, Yoon PW. Prevalence of cardiovascular disease risk factors among US adolescents, 1999–2008. Pediatrics. 2012; 129(6):1035–41. [PubMed: 22614778]
9. Center for Disease Control. National Diabetes Prevention Program. 2012. http://www.cdc.gov/diabetes/prevention/factsheet.htm
10. Virtanen S, Uusitalo L, Kenward M, Nevalainen J, Uusitalo U, Kronberg–Kippilä C, et al. Maternal food consumption during pregnancy and risk of advanced β–cell autoimmunity in the offspring. Pediatric diabetes. 2011; 12(2):95–9. [PubMed: 21352426]
11. Dabelea D, Crume T. Maternal environment and the transgenerational cycle of obesity and diabetes. Diabetes. 2011; 60(7):1849–55. [PubMed: 21709280]
12. Wing RR, Goldstein MG, Acton KJ, Birch LL, Jakicic JM, Sallis JF, et al. Behavioral Science Research in Diabetes: Lifestyle changes related to obesity, eating behavior, and physical activity. Diabetes Care. 2001; 24(1):117–23.10.2337/diacare.24.1.117 [PubMed: 11194216]
13•. Malik VS, Popkin BM, Bray GA, Després J-P, Hu FB. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. Circulation. 2010; 121(11):1356–64. This article provides evidence for links between sugar-sweetened beverages and disease. [PubMed: 20308626]
14. Ludwig DS, Pereira MA, Kroenke CH, Hilner JE, Van Horn L, Slattery ML, et al. Dietary fiber, weight gain, and cardiovascular disease risk factors in young adults. JAMA. 1999; 282(16):1539–46. [PubMed: 10546693]
15. Chandalia M, Garg A, Lutjohann D, von Bergmann K, Grundy SM, Brinkley LJ. Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. N Engl J Med. 2000; 342(19): 1392–8.10.1056/NEJM200005113421903 [PubMed: 10805824]
16. Liu S, Serdula M, Janket SJ, Cook NR, Sesso HD, Willett WC, et al. A prospective study of fruit and vegetable intake and the risk of type 2 diabetes in women. Diabetes Care. 2004; 27(12):2993–6. 27/12/2993 [pii]. [PubMed: 15562224]
17. Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, et al. Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. Am J Clin Nutr. 2002; 76(3):535–40. [PubMed: 12197996]
18. Montonen J, Knekt P, Järvinen R, Aromaa A, Reunanen A. Whole-grain and fiber intake and the incidence of type 2 diabetes. Am J Clin Nutr. 2003; 77(3):622–9. [PubMed: 12600852]
19•. Cooper AJ, Sharp SJ, Lentjes MA, Luben RN, Khaw KT, Wareham NJ, et al. A prospective study of the association between quantity and variety of fruit and vegetable intake and incident type 2 diabetes. Diabetes Care. 2012; 35(6):1293–300. dc11-2388 [pii]. This group is doing interesting work understanding links between fruit & vegetable consumption and diabetes. 10.2337/dc11-2388 [PubMed: 22474042]
20. Ford ES, Mokdad AH. Fruit and vegetable consumption and diabetes mellitus incidence among U.S. adults. Prev Med. 2001; 32(1):33–9.10.1006/pmed.2000.0772 [PubMed: 11162324]
21. Cooper AJ, Forouhi NG, Ye Z, Buijsse B, Arriola L, Balkau B, et al. Fruit and vegetable intake and type 2 diabetes: EPIC-InterAct prospective study and meta-analysis. Eur J Clin Nutr. 2012; 66(10):1082–92. ejcn201285 [pii]. 10.1038/ejcn.2012.85 [PubMed: 22854878]

22. Weickert MO, Pfeiffer AF. Metabolic effects of dietary fiber consumption and prevention of diabetes. J Nutr. 2008; 138(3):439–42. [PubMed: 18287346]
23. Lattimer JM, Haub MD. Effects of dietary fiber and its components on metabolic health. Nutrients. 2010; 2(12):1266–89. nutrients-02-01266 [pii]. 10.3390/nu2121266 [PubMed: 22254008]
24. Ray TK, Mansell KM, Knight LC, Malmud LS, Owen OE, Boden G. Long-term effects of dietary fiber on glucose tolerance and gastric emptying in noninsulin-dependent diabetic patients. Am J Clin Nutr. 1983; 37(3):376–81. [PubMed: 6299089]
25. Bolton RP, Heaton KW, Burroughs LF. The role of dietary fiber in satiety, glucose, and insulin: studies with fruit and fruit juice. Am J Clin Nutr. 1981; 34(2):211–7. [PubMed: 6259919]
26. Williamson G. Possible effects of dietary polyphenols on sugar absorption and digestion. Mol Nutr Food Res. 2013; 57(1):48–57.10.1002/mnfr.201200511 [PubMed: 23180627]
27. Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001. Am J Prev Med. 2004; 27(3):205–10.10.1016/j.amepre.2004.05.005 [PubMed: 15450632]
28. Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA. 2004; 292(8):927–34. [PubMed: 15328324]
29. Malik VS, Popkin BM, Bray GA, Després JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 2010; 33(11): 2477–83.10.2337/dc10-1079 [PubMed: 20693348]
30. Sonestedt E, Overby NC, Laaksonen DE, Birgisdottir BE. Does high sugar consumption exacerbate cardiometabolic risk factors and increase the risk of type 2 diabetes and cardiovascular disease? Food Nutr Res. 2012:56.10.3402/fnr.v56i0.19104
31. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. Am J Clin Nutr. 2004; 79(4):537–43. [PubMed: 15051594]
32. Hu FB, Malik VS. Sugar-sweetened beverages and risk of obesity and type 2 diabetes: epidemiologic evidence. Physiol Behav. 2010; 100(1):47–54.10.1016/j.physbeh.2010.01.036 [PubMed: 20138901]
33. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. JAMA. 2002; 287(18):2414–23. [PubMed: 11988062]
34. Basciano H, Federico L, Adeli K. Fructose, insulin resistance, and metabolic dyslipidemia. Nutr Metab (Lond). 2005; 2(1):5.10.1186/1743-7075-2-5 [PubMed: 15723702]
35. Hebden L, Cook A, van der Ploeg HP, Allman-Farinelli M. Development of smartphone applications for nutrition and physical activity behavior change. JMIR Res Protoc. 2012; 1(2):e9.10.2196/resprot.2205 [PubMed: 23611892]
36. Spring B, Duncan JM, Janke EA, Kozak AT, McFadden HG, DeMott A, et al. Integrating technology into standard weight loss treatment: a randomized controlled trial. JAMA internal medicine. 2013; 173(2):105. [PubMed: 23229890]
37. Neuenschwander LM, Abbott A, Mobley AR. Comparison of a web-based vs in-person nutrition education program for low-income adults. J Acad Nutr Diet. 2013; 113(1):120–6.10.1016/j.jand.2012.07.034 [PubMed: 23092741]
38. Atienza AA, King AC, Oliveira BM, Ahn DK, Gardner CD. Using hand-held computer technologies to improve dietary intake. Am J Prev Med. 2008; 34(6):514–8.10.1016/j.amepre.2008.01.034 [PubMed: 18471588]
39. Beasley JM, Riley WT, Davis A, Singh J. Evaluation of a PDA-based dietary assessment and intervention program: a randomized controlled trial. J Am Coll Nutr. 2008; 27(2):280–6. [PubMed: 18689560]
40. Davis JN, Ventura EE, Cook LT, Gyllenhammer LE, Gatto NM. LA Sprouts: a gardening, nutrition, and cooking intervention for Latino youth improves diet and reduces obesity. J Am Diet Assoc. 2011; 111(8):1224–30. S0002-8223(11)00581-5 [pii]. 10.1016/j.jada.2011.05.009 [PubMed: 21802571]
41. Robinson-O'Brien R, Story M, Heim S. Impact of garden-based youth nutrition intervention programs: a review. J Am Diet Assoc. 2009; 109(2):273–80. S0002-8223(08)02044-0 [pii]. 10.1016/j.jada.2008.10.051 [PubMed: 19167954]

42. Caraher M, Seeley A, Wu M, Lloyd S. When chefs adopt a school? An evaluation of a cooking intervention in English primary schools. Appetite. 2013; 62:50–9.10.1016/j.appet.2012.11.007 [PubMed: 23201301]
43. Brownell KD, Frieden TR. Ounces of prevention--the public policy case for taxes on sugared beverages. N Engl J Med. 2009; 360(18):1805–8.10.1056/NEJMp0902392 [PubMed: 19357400]
44. Barry CL, Niederdeppe J, Gollust SE. Taxes on sugar-sweetened beverages: results from a 2011 national public opinion survey. Am J Prev Med. 2013; 44(2):158–63.10.1016/j.amepre.2012.09.065 [PubMed: 23332333]
45. Albright A, Franz M, Hornsby G, Kriska A, Marrero D, Ullrich I, et al. American College of Sports Medicine position stand. Exercise and type 2 diabetes. Med Sci Sports Exerc. 2000; 32(7):1345–60. [PubMed: 10912903]
46. Bassuk SS, Manson JE. Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. Journal of Applied Physiology. 2005; 99(3):1193–204. [PubMed: 16103522]
47. Lindgarde F, Saltin B. Daily physical activity, work capacity and glucose tolerance in lean and obese normoglycaemic middle-aged men. Diabetologia. 1981; 20(2):134–8. [PubMed: 7009291]
48. Wang JT, Ho LT, Tang KT, Wang LM, Chen YD, Reaven GM. Effect of habitual physical activity on age-related glucose intolerance. Journal of the American Geriatrics Society. 1989; 37(3):203. [PubMed: 2645353]
49. Cederholm JAN, Wibell L. Glucose Tolerance and Physical Activity in a Health Survey of Middle–aged Subjects. Acta Medica Scandinavica. 1985; 217(4):373–8. [PubMed: 4013828]
50. Dunstan DW, Salmon J, Owen N, Armstrong T, Zimmet PZ, Welborn TA, et al. Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. Diabetes Care. 2004; 27(11):2603–9. [PubMed: 15504993]
51. Eriksson KF, Lindggindgwen N, Armstrong T, Zimmet PZ, Welborn TA, et al. Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. Dia.
52. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care. 1997; 20(4):537–44. [PubMed: 9096977]
53. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. New England Journal of Medicine. 2001; 345(11):790–7. [PubMed: 11556298]
54. Berman LJ, Weigensberg MJ, Spruijt-Metz D. Physical activity is related to insulin sensitivity in children and adolescents, independent of adiposity: a review of the literature. Diabetes Metab Res Rev. 2012; 28(5):395–408.10.1002/dmrr.2292 [PubMed: 22389103]
55. Hubinger A, Franzen A, Gries FA. Hormonal and metabolic response to physical exercise in hyperinsulinemic and non-hyperinsulinemic type 2 diabetics. Diabetes research (Edinburgh, Scotland). 1987; 4(2):57–61.
56. Winter G. Handbook of exercise in diabetes. British Journal of Sports Medicine. 2007; 41(3):182.
57. USDHHS. USDHHS. 2008 Physical Activity Guidelines for Americans. Washington DC: USDHHS; 2008.
58. Bickmore, T.; Gruber, A.; Intille, S., editors. Just-in-time automated counseling for physical activity promotion; AMIA... Annual Symposium proceedings/AMIA Symposium. AMIA Symposium; 2008.
59. Brownson RC, Baker EA, Housemann RA, Brennan LK, Bacak SJ. Environmental and policy determinants of physical activity in the United States. American journal of public health. 2001; 91(12):1995–2003. [PubMed: 11726382]
60. Cobiac LJ, Vos T, Barendregt JJ. Cost-effectiveness of interventions to promote physical activity: a modelling study. PLoS Med. 2009; 6(7):e1000110.10.1371/journal.pmed.1000110 [PubMed: 19597537]
61. Estabrooks PA, Gyurcsik NC. Evaluating the impact of behavioral interventions that target physical activity: issues of generalizability and public health. Psychology of sport and exercise. 2003; 4(1):41–55.

62. Heck TA, Kimiecik JC. What is exercise enjoyment? A qualitative investigation of adult exercise maintainers. Wellness Perspectives. 1993; 10(1):3–21.
63. Nies MA, Vollman M, Cook T. Facilitators, barriers, and strategies for exercise in European American women in the community. Public Health Nursing. 1998; 15(4):263–72. [PubMed: 9682619]
64. Marcus BH, Emmons KM, Simkin-Silverman LR, Linnan LA, Taylor ER, Bock BC, et al. Evaluation of motivationally tailored vs. standard self-help physical activity interventions at the workplace. American Journal of Health Promotion. 1998; 12(4):246–53. [PubMed: 10178617]
65. Marcus BH, Banspach SW, Lefebvre RC, Rossi JS, Carleton RA, Abrams DB. Using the stages of change model to increase the adoption of physical activity among community participants. American Journal of Health Promotion. 1992; 6(6):424–9. [PubMed: 10146803]
66. Krebs P, Prochaska JO, Rossi JS. A meta-analysis of computer-tailored interventions for health behavior change. Preventive medicine. 2010; 51(3):214–21. [PubMed: 20558196]
67. Warburton DE. The Health Benefits of Active Gaming: Separating the Myths from the Virtual Reality. Current Cardiovascular Risk Reports. 2013:1–5.
68. Gotsis M, Wang H, Spruijt-Metz D, Jordan-Marsh M, Valente TW. Wellness partners: design and evaluation of a web-based physical activity diary with social gaming features for adults. JMIR RESEARCH PROTOCOLS. 2013; 2(1):e10. [PubMed: 23611986]
69. Kumar S, Nilsen WJ, Abernethy A, Atienza A, Patrick K, Pavel M, et al. Mobile Health Technology Evaluation: The mHealth Evidence Workshop. Am J Prev Med. 2013; 45(2):228–36. http://dx.doi.org/10.1016/j.amepre.2013.03.017. [PubMed: 23867031]
70. Pate RR, O'Neill JR, Lobelo F. The evolving definition of "sedentary". Exercise and sport sciences reviews. 2008; 36(4):173–8. [PubMed: 18815485]
71. Owen N, Healy GvN, Matthews CE, Dunstan DW. Too much sitting: the population-health science of sedentary behavior. Exercise and sport sciences reviews. 2010; 38(3):105. [PubMed: 20577058]
72. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. Diabetes. 2007; 56(11):2655–67.10.2337/db07-0882 [PubMed: 17827399]
73. Hamilton MT, Healy GN, Dunstan DW, Zderic TW, Owen N. Too little exercise and too much sitting: inactivity physiology and the need for new recommendations on sedentary behavior. Current cardiovascular risk reports. 2008; 2(4):292–8. [PubMed: 22905272]
74. Dunstan DW, Salmon J, Healy GN, Shaw JE, Jolley D, Zimmet PZ, et al. Association of television viewing with fasting and 2-h postchallenge plasma glucose levels in adults without diagnosed diabetes. Diabetes Care. 2007; 30(3):516–22. [PubMed: 17327314]
75. Ezzati M, Lopez AD, Rodgers A, Murray CJL. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors. OMS. 2004
76. Farrell SW, Cheng YJ, Blair SN. Prevalence of the metabolic syndrome across cardiorespiratory fitness levels in women. Obesity research. 2004; 12(5):824–30. [PubMed: 15166303]
77. Katzmarzyk PT, Church TS, Blair SN. Cardiorespiratory fitness attenuates the effects of the metabolic syndrome on all-cause and cardiovascular disease mortality in men. Archives of Internal Medicine. 2004; 164(10):1092. [PubMed: 15159266]
78. Kronenberg F, Pereira MA, Schmitz MKH, Arnett DK, Evenson KR, Crapo RO, et al. Influence of leisure time physical activity and television watching on atherosclerosis risk factors in the NHLBI Family Heart Study. Atherosclerosis. 2000; 153(2):433–43. [PubMed: 11164433]
79. Bey L, Hamilton MT. Suppression of skeletal muscle lipoprotein lipase activity during physical inactivity: a molecular reason to maintain daily low–intensity activity. The Journal of physiology. 2003; 551(2):673–82. [PubMed: 12815182]
80. Healy GN, Dunstan DW, Salmon J, Cerin E, Shaw JE, Zimmet PZ, et al. Breaks in Sedentary Time Beneficial associations with metabolic risk. Diabetes Care. 2008; 31(4):661–6. [PubMed: 18252901]
81. Sevick MA, Dunn AL, Morrow MS, Marcus BH, Chen GJ, Blair SN. Cost-effectiveness of lifestyle and structured exercise interventions in sedentary adults: results of project ACTIVE. American journal of preventive medicine. 2000; 19(1):1–8. [PubMed: 10865157]

82. Lee J, Mustafa T, McDowall SG, Mendelsohn FA, Brennan M, Lew RA, et al. Structure-activity study of LVV-hemorphin-7: angiotensin AT4 receptor ligand and inhibitor of insulin-regulated aminopeptidase. Journal of Pharmacology & Experimental Therapeutics. 2003; 305(1):205–11. [PubMed: 12649370]
83. Owen N, Sugiyama T, Eakin EE, Gardiner PA, Tremblay MS, Sallis JF. Adults' sedentary behavior: determinants and interventions. American journal of preventive medicine. 2011; 41(2): 189–96. [PubMed: 21767727]
84. Cooper A, Sebire S, Montgomery A, Peters T, Sharp D, Jackson N, et al. Sedentary time, breaks in sedentary time and metabolic variables in people with newly diagnosed type 2 diabetes. Diabetologia. 2012; 55(3):589–99. [PubMed: 22167127]
85. Chaput JP, Despres JP, Bouchard C, Tremblay A. Association of sleep duration with type 2 diabetes and impaired glucose tolerance. Diabetologia. 2007; 50(11):2298–304. [PubMed: 17717644]
86. Ayas NT, White DP, Al-Delaimy WK, Manson JE, Stampfer MJ, Speizer FE, et al. A prospective study of self-reported sleep duration and incident diabetes in women. Diabetes Care. 2003; 26(2): 380–4. [PubMed: 12547866]
87. Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. Diabetes Care. 2006; 29(3):657–61. [PubMed: 16505522]
88. Knutson KL, Ryden AM, Mander BA, Van Cauter E. Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. Archives of Internal Medicine. 2006; 166(16):1768. [PubMed: 16983057]
89. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. The Lancet. 1999; 354(9188):1435–9.
90. Spiegel K, Knutson K, Leproult R, Tasali E, Van Cauter E. Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. Journal of Applied Physiology. 2005; 99(5):2008–19. [PubMed: 16227462]
91. Tasali E, Leproult R, Ehrmann DA, Van Cauter E. Slow-wave sleep and the risk of type 2 diabetes in humans. Proceedings of the National Academy of Sciences. 2008; 105(3):1044–9.
92. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and Quality of Sleep and Incidence of Type 2 Diabetes A systematic review and meta-analysis. Diabetes Care. 2010; 33(2):414–20. [PubMed: 19910503]
93. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. Health Psychology. 2006; 25(1):3. [PubMed: 16448292]
94. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia. JAMA: the journal of the American Medical Association. 1999; 281(11):991–9. [PubMed: 10086433]
95. McClusky HY, Milby JB, Switzer PK, Williams V, Wooten V. Efficacy of behavioral versus triazolam treatment in persistent sleep-onset insomnia. Am J Psychiatry. 1991; 148(1):121–6. [PubMed: 1888345]
96. Tanaka H, Taira K, Arakawa M, Toguti H, Urasaki C, Yamamoto Y, et al. Effects of short nap and exercise on elderly people having difficulty in sleeping. Psychiatry and clinical neurosciences. 2001; 55(3):173–4. [PubMed: 11422829]
97. Novak M, Björck L, Giang KW, Heden-Ståhl C, Wilhelmsen L, Rosengren A. Perceived stress and incidence of Type 2 diabetes: a 35-year follow-up study of middle-aged Swedish men. Diabet Med. 2013; 30(1):e8–16.10.1111/dme.12037 [PubMed: 23075206]
98. Cosgrove MP, Sargeant LA, Caleyachetty R, Griffin SJ. Work-related stress and Type 2 diabetes: systematic review and meta-analysis. Occup Med (Lond). 2012; 62(3):167–73.10.1093/occmed/kqs002 [PubMed: 22333189]
99. Melamed S, Shirom A, Toker S, Shapira I. Burnout and risk of type 2 diabetes: a prospective study of apparently healthy employed persons. Psychosom Med. 2006; 68(6):863–9.10.1097/01.psy.0000242860.24009.f0 [PubMed: 17132837]

100. Pouwer F, Kupper N, Adriaanse MC. Does emotional stress cause type 2 diabetes mellitus? A review from the European Depression in Diabetes (EDID) Research Consortium. Discov Med. 2010; 9(45):112–8. [PubMed: 20193636]

101. Björntorp P, Holm G, Rosmond R. Hypothalamic arousal, insulin resistance and Type 2 diabetes mellitus. Diabet Med. 1999; 16(5):373–83. [PubMed: 10342336]

102. Rosmond R. Stress induced disturbances of the HPA axis: a pathway to Type 2 diabetes? Med Sci Monit. 2003; 9(2):RA35-9. [PubMed: 12601304]

103. McEwen BS. Protective and damaging effects of stress mediators. N Engl J Med. 1998; 338(3): 171–9.10.1056/NEJM199801153380307 [PubMed: 9428819]

104. Scott KA, Melhorn SJ, Sakai RR. Effects of Chronic Social Stress on Obesity. Curr Obes Rep. 2012; 1(1):16–25.10.1007/s13679-011-0006-3 [PubMed: 22943039]

105. Hamer M, Endrighi R, Poole L. Physical activity, stress reduction, and mood: insight into immunological mechanisms. Methods Mol Biol. 2012; 934:89–102.10.1007/978-1-62703-071-7_5 [PubMed: 22933142]

106. Grossman P, Niemann L, Schmidt S, Walach H. Mindfulness-based stress reduction and health benefits. A meta-analysis. J Psychosom Res. 2004; 57(1):35–43.10.1016/S0022-3999(03)00573-7 [PubMed: 15256293]

107. Alberts HJ, Mulkens S, Smeets M, Thewissen R. Coping with food cravings. Investigating the potential of a mindfulness-based intervention. Appetite. 2010; 55(1):160–3.10.1016/j.appet.2010.05.044 [PubMed: 20493913]

108. Dalen J, Smith BW, Shelley BM, Sloan AL, Leahigh L, Begay D. Pilot study: Mindful Eating and Living (MEAL): weight, eating behavior, and psychological outcomes associated with a mindfulness-based intervention for people with obesity. Complement Ther Med. 2010; 18(6): 260–4.10.1016/j.ctim.2010.09.008 [PubMed: 21130363]

109. Daubenmier J, Kristeller J, Hecht FM, Maninger N, Kuwata M, Jhaveri K, et al. Mindfulness Intervention for Stress Eating to Reduce Cortisol and Abdominal Fat among Overweight and Obese Women: An Exploratory Randomized Controlled Study. J Obes. 2011; 2011:651936.10.1155/2011/651936 [PubMed: 21977314]

110. Hoch DB, Watson AJ, Linton DA, Bello HE, Senelly M, Milik MT, et al. The feasibility and impact of delivering a mind-body intervention in a virtual world. PLoS One. 2012; 7(3):e33843.10.1371/journal.pone.0033843 [PubMed: 22470483]

111. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin.[see comment]. N Engl J Med. 2002; 346(6):393–403. [PubMed: 11832527]

112. Knowler WC, Fowler SE, Hamman RF, Christophi CA, Hoffman HJ, Brenneman AT, et al. 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. Lancet. 2009; 374(9702):1677–86.10.1016/s0140-6736(09)61457-4 [PubMed: 19878986]

113. Ory MG, Lee Smith M, Mier N, Wernicke MM. The science of sustaining health behavior change: the Health Maintenance Consortium. American Journal of Health Behavior. 2010; 34(6): 647–59. [PubMed: 20604691]

114. Taheri S, Lin L, Austin D, Young T, Mignot E. Short Sleep Duration is Associated with Reduced Leptin, Elevated Ghrelin, and Increased Body Mass Index. PLOS MEDICINE. 2004; 1(3):210.

115. Van Reeth O, Weibel L, Spiegel K, Leproult R, Dugovic C, Maccari S. Physiology of Sleep (Review)–Interactions between stress and sleep: from basic research to clinical situations. Sleep Medicine Reviews. 2000; 4(2):201–19.

116. Pervanidou P, Chrousos GP. Metabolic consequences of stress during childhood and adolescence. Metabolism. 2012; 61(5):611–9. [PubMed: 22146091]

117. Dallman MF, Akana SF, Laugero KD, Gomez F, Manalo S, Bell ME, et al. A spoonful of sugar: feedback signals of energy stores and corticosterone regulate responses to chronic stress. Physiol Behav. 2003; 79(1):3–12.10.1016/s0031-9384(03)00100-8 [PubMed: 12818705]

118. Dishman RK, Renner KJ, White-Welkley JE, Burke K, Bunnell BN. Treadmill exercise training augments brain norepinephrine response to familiar and novel stress. Brain Res Bull. 2000; 52(5):337–42. [PubMed: 10922511]

119. Driver HS, Taylor SR. Exercise and sleep. Sleep Medicine Reviews. 2000; 4(4):387–402. http://dx.doi.org/10.1053/smrv.2000.0110. [PubMed: 12531177]

120••. Spring B, Schneider K, McFadden HG, Vaughn J, Kozak AT, Smith M, et al. Multiple Behavior Changes in Diet and Activity: A Randomized Controlled Trial Using Mobile Technology. Arch Intern Med. 2012; 172(10):789–96. This study gives an outsanding example of how mobile technologies and new research designs can provide effective interventions to improve diabetes-related behaviors. 10.1001/archinternmed.2012.1044 [PubMed: 22636824]

121•. Nilsen WJ, Haverkos L, Nebeling L, Taylor MV. Maintenance of long-term behavior change. American Journal of Health Behavior. 2010; 34(6):643–6. This article explains why maintenance of behavior change is so difficult. [PubMed: 20604690]

122. Bronfenbrenner, U.; Morris, PA. The Bioecological Model of Human Development. In: Lerner, RM.; Damon, W., editors. Handbook of child psychology (6th ed.): Vol 1, Theoretical models of human development. Hoboken, NJ, US: John Wiley & Sons Inc; 2006. p. 793-828.

123. Glanz, K.; Rimer, BK.; Viswanath, K. Health behavior and health education: theory, research, and practice. Jossey-Bass Inc Pub; 2008.

124. Michie S, van Stralen MM, West R. The behaviour change wheel: A new method for characterising and designing behaviour change interventions. Implement Sci. 2011; 6:42. [PubMed: 21513547]

125. Hekler EB, Klasnja P, Traver V, Hendriks M. Realizing Effective Behavioral Management of Health: The Metamorphosis of Behavioral Science Methods. IEEE PULSE. in press.

126. Saranummi N, Spruijt-Metz D, Intille SS, Korhonen I, Nilsen WJ, Pavel M. Moving the science of behavior change into the 21st Century. IEEE PULSE. in press.

127. Spring B, Gotsis M, Paiva A, Spruijt-Metz D. Technology Enabled Continuous Observation: Creative Disruptor of Behavior Change Intervention. IEEE PULSE. in press.

128. Collins LM, Murphy SA, Bierman KL. A conceptual framework for adaptive preventive interventions. Prev Sci. 2004; 5(3):185–96. [PubMed: 15470938]