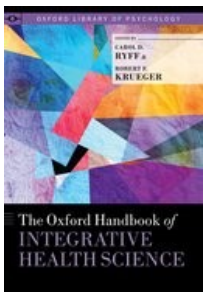

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CHAPTER

18 Biopsychosocial Pathways to Prediabetes and Diabetes

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Abstract

Type 2 diabetes is a significant problem **worldwide**, accounting for substantial morbidity and premature mortality. Understanding why some people remain healthy while others progress to insulin resistance and diabetes requires an integrative approach. Models of diabetes have focused on excess weight and physical inactivity as key targets for reducing diabetes risk. Obesity is a risk factor for developing Type 2 diabetes, yet most obese persons do not develop diabetes, suggesting moderation by other influences. This chapter highlights Midlife in the United States studies that employed multidomain assessments of the interplay between established risk factors and psychosocial influences on diabetes. The chapter reviews evidence that depression, anger, perceived weight discrimination, and neuroticism exacerbate the association between obesity and diabetes. The conclusion that psychosocial processes are major risk factors underscores the need for assessing mental health and promoting psychological well-being to complement traditional prevention efforts.

Keywords: diabetes, health, obesity, type 2 diabetes, MIDUS, risk factors, depression, anger, neuroticism, psychosocial factors

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Introduction

Diabetes is a public health epidemic of striking proportions: It affects an estimated 9.3% (29.1 million people) of the US population, and 37% have milder forms of hyperglycemia that typically transition to overt diabetes (CDC, 2014). Diabetes accounts for substantial morbidity, such as kidney failure, blindness, and lower limb amputations. Cardiovascular disease is the most significant complication of diabetes; 65% of the deaths among people with diabetes are due to heart disease and stroke (2014). Individuals with diabetes have a 50% higher risk of premature death; in the United States, the total annual cost of diabetes was estimated in 2014 at $245 billion dollars. Risk for diabetes increases dramatically as a function of age, affecting more than 26% of US adults 65 years and older.

Type 2 diabetes is the most common form of diabetes and the focus of this chapter. We start by introducing the biggest risk factor for diabetes—obesity—and review Midlife in the United States (MIDUS) studies that have demonstrated synergies between obesity and psychosocial vulnerabilities in influencing glucoregulation. Specifically, we review evidence that depression, anger, perceived weight discrimination, and neuroticism amplify the extent to which obesity increases diabetes risk. Then, we review emerging evidence for the role of psychosocial resources vis-à-vis a powerful nonmodifiable risk factor, such as a family history of diabetes. The role of socioeconomic status (SES) and race differences in diabetes risk has been well documented, and we review information from MIDUS focused on identifying underlying pathways and subgroup differences in these associations.

These studies have capitalized on the richness of multidomain assessments in MIDUS and leveraged its
p. 252 ongoing longitudinal sample (MIDUS 1 and 2; about 10 years between assessments) to integrate ↳ separate strands of biomedical, health disparities, and psychosocial research. The availability of comprehensive assessments of glucoregulation in MIDUS is particularly noteworthy; it has allowed examination of preclinical hyperglycemic states, such as insulin resistance, that often transition to diabetes and are thus of key importance as early warning states. Tracking the predisease development of Type 2 diabetes via multiple markers such as fasting glucose, fasting insulin, and glycosylated hemoglobin (HbA_{1c}) is an important approach for examining the documented high interindividual variability in the preclinical progression to diabetes (Fonseca, 2009).

Obesity

Obesity is a major public health threat in the United States: More than one third of adults were obese in 2009–2010, up from 5–6% only three decades ago (Ogden, Carroll, Kit, & Flegal, 2012). Contemporary environment plays a central role in this increase and is frequently described as obesogenic because it facilitates obesity via an abundant availability of energy-dense food and declines in physical activity. Central obesity, or the distribution of body fat around the abdominal area, has been identified as a key culprit in promoting diabetogenic substances and increasing diabetes risk (DeFronzo, 2004).

Convincing evidence suggests that physical activity and weight loss are two critical cornerstones of successful diabetes prevention. The Diabetes Prevention Program, a landmark multicenter randomized control trial, aimed to investigate whether diabetes can be delayed or prevented in at-risk people (Knowler et al., 2002). At the beginning of the program, all participants were overweight and met criteria for prediabetes. Results showed that even modest weight loss decreased diabetes risk: The incidence of diabetes was reduced by 58% with lifestyle intervention; importantly, 10 years after beginning the study, the incidence of diabetes for individuals in the lifestyle intervention group remained reduced at 34% (Diabetes Prevention Program Research Group, Knowler, Fowler, et al., 2009).

The public health crises of obesity have stimulated interest in the concept of energy balance, or the relationship between "energy in" (calories obtained through food and drink) and "energy out" (calories expended for daily energy requirements). Much research has focused on the triggers of eating behavior and identified factors that extend the concept of energy balance by describing pathways to energy imbalance. Negative energy balance is a sufficient, but not necessary, condition for initiating an eating episode (Del Parigi, 2010), and given the overlap between the physiological systems that regulate food intake and the ones that mediate the stress response, the association between stress and eating has garnered a lot of attention (Tannenbaum, Anisman, & Abizaid, 2010).

The sympathetic–adrenomedullary axis (SAM) and hypothalamic–pituitary–adrenal (HPA) axis are activated following a stressful event to prepare the organism for *fight or flight*, as mounting an effective defensive response depends on available energy. In essence, it is a metabolic shift from an anabolic state to a catabolic or energy-available state, resulting in increased circulating levels of glucose and free fatty acids. Adrenal catecholamines and the HPA axis play a major role in this release of glucose into the bloodstream from the tissue stores of glycogen, thereby suppressing hunger (Gold & Chrousos, 2002). Stress eaters, however, do not necessarily show reduced food intake during stress (Gold & Chrousos, 2002), but instead consume the same amount (or more) (Oliver, Wardle, & Gibson, 2000; van Strien & Ouwens, 2003). Intake of "comfort foods," or foods high in sugar and fat, is thought to alleviate the experience of stress and arousal by promoting the activation of brain circuits involved in reward-seeking behavior (Dallman et al., 2003), thereby further reinforcing feeding behavior (Dallman, 2010).

Obesity and Stress Eating

We used data from the MIDUS 2 Biomarker subsample to examine the associations among stress eating, obesity, and glucoregulation (Tsenkova, Boylan, & Ryff, 2013). MIDUS respondents were asked to indicate how they "usually experience a stressful event," two options of which were "I eat more of my favorite foods to make myself feel better" and "I eat more than I usually do." Responses were summed so that higher scores indicated greater use of food in response to stress. We found that people who reported eating in response to stress had significantly worse preclinical glucoregulation and higher risk for diabetes, with the effects occurring primarily through increased central adiposity. The findings have significant implications: Self-reported stress levels have been increasing over time (S. Cohen & Janicki-Deverts, 2012), and a recent national survey documented that 39% of people overeat in response to stress (American Psychological
p. 253 Association, 2012). Thus, stress eating is a worthy target for intervention ↳ and prevention work regarding obesity and chronic disease. Such work requires understanding the root causes of overeating and identifying novel routes for achieving lasting weight loss and preventing diabetes.

Psychosocial Moderation of Traditional Risk Factors: Obesity

While there is little doubt that the epidemic of Type 2 diabetes has been fueled by obesity, significant variability exists in the association between obesity and diabetes. For example, even though more than 80% of people with Type 2 diabetes are obese, most obese people *never* develop diabetes (Attie, 2004), suggesting that other factors moderate the influence of obesity to increase susceptibility to, or confer protection from, disease. This heterogeneity was highlighted by a recent consensus report from the American Diabetes Association, the Endocrine Society, and the European Association for the Study of Diabetes, which identified major questions to guide the field, improve primary prevention and intervention, and benefit overall patient care (Eckel et al., 2011). The first question was, "Why do not all patients with obesity develop type 2 diabetes?" (p. 1424). MIDUS has provided a valuable forum for focusing on individual differences and delineating the biopsychosocial pathways to Type 2 diabetes. While much work remains, our initial studies with MIDUS have identified different psychosocial vulnerabilities (depression, anger, perceived weight discrimination, and neuroticism) that amplify the obesity-conferred risk for insulin resistance, prediabetes, and Type 2 diabetes.

Depression

Depression has emerged as a key risk factor for Type 2 diabetes. Depressed adults have a 37% to 60% higher risk of developing diabetes than adults without depression (Knol et al., 2006; Mezuk, Eaton, Albrecht, & Golden, 2008). Depressive symptoms have been linked to metabolic abnormalities that precede the development of diabetes (Kan et al., 2013) as well as poor prognosis (Lustman et al., 2000; Sullivan et al., 2012). Behavioral and physiological mechanisms have been proposed to explain the increased risk of diabetes among depressed adults. For example, depression is associated with unhealthy behaviors that may increase one's risk of obesity (Golden et al., 2008; Nouwen et al., 2011). Depressed individuals also show abnormalities in the HPA axis, particularly in regulation of cortisol, which contributes to dysregulated glucose metabolism (Joseph et al., 2015; Rosmond & Bjorntorp, 2000; Stetler & Miller, 2011). The co-occurrence of depression with other metabolic risk factors (e.g., high triglycerides, hypertension, and abdominal obesity) has been labeled "metabolic depression" (Liu, Carvalho, & McIntyre, 2014; Vogelzangs et al., 2011), and the combination of depression and metabolic dysregulation might be a fundamental risk factor associated with increased diabetes risk (Schmitz et al., 2016).

We used data from MIDUS 1 (Survey) and MIDUS 2 (Survey and Biomarker) to examine prospectively the association of central obesity and depression with glucose metabolism 10 years later among adults without diabetes at baseline (Tsenkova & Karlamangla, 2016). A diagnosis of past year depressive episode was defined as a period of at least 2 weeks of either depressed mood or anhedonia most of the day, nearly every day, and a series of at least four symptoms typically found to accompany depression, such as problems with eating, sleeping, energy, concentration, feelings of self-worth, and suicidal thoughts or actions (Kessler, Mickelson, Walters, Zhao, & Hamilton, 2004). We found, not surprisingly, strong evidence that a larger waist-to-hip ratio at baseline predicted higher diabetes incidence and higher levels of glucose dysregulation 10 years later. We also found strong evidence of moderation effects, wherein depression *amplified* the association of waist-to-hip ratio with incident diabetes (Figure 18.1). This relationship did not depend on one's age or gender and persisted net of adjustments for demographics, SES, and health status, thereby sharpening the focus on the distinctive contribution of depression and central obesity to glycemic control. Our findings suggest that identifying and addressing depression could prove to be an effective approach to preventing diabetes in individuals who are already at a higher risk due to central obesity.

Anger

Anger has been consistently implicated in the etiology of cardiovascular disease (al'Absi & Bongard, 2006; Haukkala, Konttinen, Laatikainen, Kawachi, & Uutela, 2010; Koh, Choe, & An, 2003; Merjonen et al., 2008; Mittleman et al., 1995; Williams et al., 2000) and metabolic syndrome (Boylan & Ryff, 2015; B. E. Cohen, Panguluri, Na, & Whooley, 2010; Gremigni, 2006; Nelson, Palmer, & Pedersen, 2004; Niaura et al., 2000; Raikkonen, Matthews, & Kuller, 2002). Despite the shared risk factors between cardiovascular disease,
p. 254 metabolic syndrome, and Type 2 diabetes, only two prospective ↳ studies have linked anger to diabetes incidence. In the Atherosclerosis Risk in Communities Study, a prospective epidemiologic study conducted in four US communities, people in the highest category of trait anger had a 34% increased risk of diabetes relative to those in the lowest category (Golden et al., 2006). Similarly, in the Multi-Ethnic Study of Atherosclerosis, a population-based sample of ethnically diverse men and women, individuals with high trait anger at baseline had a 50% increased risk of developing diabetes (Abraham et al., 2015).

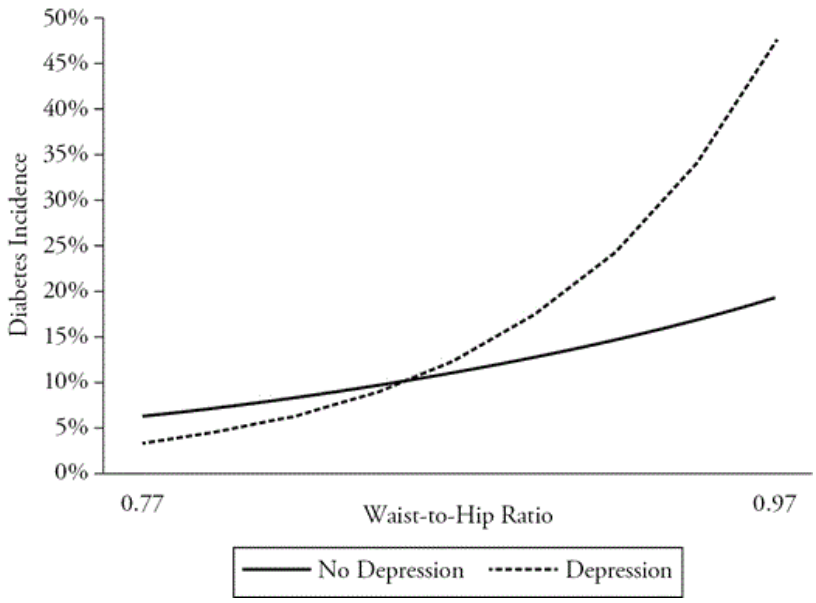

Figure 18.1 Model-predicted diabetes incidence risk in MIDUS 2 as a function of baseline depression (yes/no) and waist-to-hip ratio.

From Tsenkova, V. K., & Karlamangla, A. (2016). Depression amplifies the influence of central obesity on 10-year incidence of diabetes: Findings from MIDUS. *PLos One, 11*(10), e0164802.

We used data from the MIDUS 2 Biomarker subsample to examine whether anger and obesity were associated with nondiabetic glucoregulation (Tsenkova, Carr, Coe, & Ryff, 2014). Anger expression was operationalized with the Spielberger's State Trait Anger Expression questionnaire (Spielberger, 1996), a theoretically informed measure for characterizing anger expression. Two distinctive modes of anger expression were captured: *anger-out* (expressing angry feelings in aggressive behavior directed toward other people or objects) and *anger-in* (suppressing angry feelings and holding them in). We showed that anger expression was independently, and in combination with body mass index (BMI) and waist-to-hip ratio, associated with worse glucoregulation. This effect was evident across multiple measures of anger, weight distribution, and glucose metabolism, further supporting the idea that negative psychological states are particularly pernicious in the presence of obesity.

Perceived Weight Discrimination

Perceived weight discrimination is a social stressor that has only recently been linked to health outcomes. (See Chapter 20 for a discussion of linkages between perceived weight discrimination and psychosocial outcomes.) Obesity is considered one of the most enduring stigmas, due in part to the perception that it is a result of characterological flaws such as laziness or gluttony (Puhl & Brownell, 2001; Puhl & Heuer, 2009). Theoretical work on the origins of weight stigma has identified perceived controllability of the cause of obesity as particularly important (Crandall & Martinez, 1997; Rodin, Price, Sanchez, & McElliot, 1989). Recent MIDUS studies showed that people who reported experiencing weight discrimination had increased allostatic loads and mortality rates compared to people who did not report weight discrimination (Sutin, Stephan, & Terracciano, 2015; Vadiveloo & Mattei, 2017).

We used data from the MIDUS 2 Biomarker subsample to integrate the separate strands of previous biomedical research on obesity and psychosocial research on stigma. Perceived discrimination due to weight is a targeted measure of obesity-related stress whose association to glucoregulation was unknown. We investigated whether the impact of obesity on glycemic control was amplified by experiences of perceived discrimination that one attributes to body weight (Tsenkova, Carr, Schoeller, & Ryff, 2011). Our focus on
p. 255
perceived ↳ discrimination as an individual difference variable highlighted the fact that some obese people see themselves as treated unfairly by others on the basis of their weight, while other obese people do not. Thus, the central hypothesis was that obese people who *also* perceived daily weight discrimination were more likely to have dysregulated glycemic control than obese people who did not report perceived discrimination due to weight. We found that the physical burden of carrying excess weight was significantly exacerbated by perceptions of discriminatory treatment due to weight, consistent with our work on anger and depression (Tsenkova, Carr, et al., 2014; Tsenkova & Karlamangla, 2016). This study provides further evidence that viewing biological and psychosocial factors as complementary influences is important: Policies and interventions that tackle both the discriminatory social environment faced by obese persons and their adaptations to these environments may be consequential for minimizing diabetes risk.

Neuroticism

Neuroticism refers to relatively stable tendencies to respond with negative emotions to threat, frustration, and loss (Costa & McCrae, 1992; Lahey, 2009) and reflects nervousness, moodiness, and temperamental style (Goldberg, 1993). Neuroticism has been linked to depression (Fanous, Gardner, Prescott, Cancro, & Kendler, 2002; Roberts & Kendler, 1999; Wouts et al., 2011) and anxiety (Bienvenu et al., 2001), as well as metabolic syndrome (Phillips et al., 2010; Sutin et al., 2010), cardiovascular disease (Suls & Bunde, 2005), and diabetes (Goodwin, Cox, & Clara, 2006).

We used data from the MIDUS 2 Biomarker subsample to examine whether neuroticism was associated with poor glucose regulation and, further, whether it amplified the harmful impact of high BMI on glucose metabolism (Tsenkova, Carr, Coe, & Ryff, 2012). Neuroticism was assessed using measures of the Big Five personality inventory via the self-administered portions of MIDUS 2. Respondents were asked to rate how well the following four terms described them: moody, worrying, nervous, and calm (reverse coded). A summary score was created. We found that neuroticism acted synergistically with BMI to promote insulin resistance in nondiabetic people, suggesting that neuroticism is another psychological vulnerability that amplifies the strength of the association between obesity and glucoregulatory measures.

The MIDUS study has provided an exciting opportunity to examine the interplay between established risk factors and psychosocial moderators on glucoregulation. We have documented that psychosocial vulnerabilities such as depression, anger, perceived weight discrimination, and neuroticism amplify the pernicious influence of obesity on dysregulated glucose metabolism. Taken together, these studies suggest

that targeting psychosocial vulnerabilities in addition to established risk factors such as obesity might be another route to preventing or delaying diabetes in people at risk.

Psychosocial Moderation of Traditional Risk Factors: Family History

Family history of diabetes is a key risk factor for diabetes that represents genomic information and the complex interplay between genes, shared environments and behaviors, and epigenetic effects (Franks, 2010). It predicts metabolic abnormalities and two- to six-fold higher risk for Type 2 diabetes (Hariri et al., 2006; Hilding et al., 2006; Valdez, Yoon, Liu, & Khoury, 2007; Velasco Mondragon et al., 2010). While the clustering of obesity and physical inactivity in families may account for some of the risk associated with family history of diabetes, only a small percentage of the variance has been explained by lifestyle, anthropomorphic, and genetic risk factors (InterAct Consortium et al., 2013). Moreover, the association between family history and diabetes risk in the offspring appears to be modifiable; physical inactivity and obesity, the main targets of diabetes prevention efforts, moderate the effects of family history of diabetes on glucoregulation (Chen, Rennie, & Dosman, 2010; Goodrich et al., 2012; van Dam, Boer, Feskens, & Seidell, 2001; Velasco Mondragon et al., 2010; Wikner, Gigante, Hellenius, de Faire, & Leander, 2013).

It is unknown whether psychological resources might also modulate this association, perhaps serving as buffering influences vis-à-vis established risk factors. Emerging research on the role of positive affect and well-being in glucoregulation has shown that higher levels of these factors is associated with better glycemic control and lower diabetes risk (Boehm, Trudel-Fitzgerald, Kivimaki, & Kubzansky, 2015; Nefs et al., 2012; Okely & Gale, 2016; Rasmussen et al., 2013; Richman et al., 2005; Tsenkova, Dienberg Love, Singer,
p. 256 & Ryff, 2008) as well as lower mortality among people with diabetes (Moskowitz, Epel, & Acree, 2008), ↳ independent from the influences of depression and negative affect.

We used the MIDUS 2 Biomarker subsample to examine whether positive affect mitigates the influence of parental history of diabetes on diabetes risk (Tsenkova, Karlamangla, & Ryff, 2016). Positive affect was assessed during the MIDUS 2 Survey project with six items and probed according to the following stem: "During the past 30 days, how much of the time did you feel: cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, and full of life?" We found that a parental history of diabetes predicted an almost three-fold increase in relative risk for diabetes. Consistent with other studies, this effect was only slightly attenuated by including likely mediators, highlighting the fact that while parental diabetes is a strong predictor of diabetes risk, we have a limited understanding of mediating processes and subgroup differences. Importantly, we found that positive affect buffered the impact of parental history of diabetes: Each one standard deviation unit increment in positive affect was associated with a 34% reduced risk of diabetes among people who have a parental history of diabetes, an effect size comparable in magnitude to 31% reduced diabetes incidence in the Diabetes Prevention Program with "gold standard" pharmacological treatment (Knowler et al., 2002; Tsenkova et al., 2016). Our work underscores the potential role of protective influences in a multifactorial context and suggests that interventions targeting diabetes could be strengthened by promoting psychological well-being, which is modifiable, including in contexts of depression (Ruini & Ryff, forthcoming).

Psychosocial Moderation of Traditional Risk Factors: Socioeconomic Status and Race

Persons with fewer socioeconomic resources and racial/ethnic minority groups bear a disproportionate share of the diabetes burden. While the prevalence and incidence of diabetes have increased markedly in the last three decades, low SES groups and minorities have experienced the most dramatic increases and most severe consequences from diabetes (Bardenheier et al., 2013; Demakakos, Marmot, & Steptoe, 2012; Geiss et al., 2006; Johnson & Schoeni, 2011; Kanjilal et al., 2006; Kavanagh et al., 2010; Kumari, Head, & Marmot, 2004; Narayan, Boyle, Geiss, Saaddine, & Thompson, 2006; Tamayo, Christian, & Rathmann, 2010). For example, the age-adjusted prevalence of diabetes among black adults is approximately twice that for non-Hispanic whites (CDC, 2014). Therefore, better understanding of and addressing social inequalities in diabetes have assumed increasing importance.

We used longitudinal data from MIDUS 1 (Survey) and MIDUS 2 (Survey and Biomarker) to explore life course processes linking early life SES disadvantage and glucoregulation (Tsenkova, Pudrovska, & Karlamangla, 2014). The overarching goal was to quantify the relationship between childhood SES disadvantage and adult glucoregulation and, further, examine whether key risk factors (central obesity, depressive symptoms, and physical inactivity) were components of the pathway between early life SES and glucoregulation in later life. We found that childhood SES disadvantage predicted higher waist circumference and less physical activity net of adult SES, suggesting that early childhood may be a critical period for tracking long-term effects of disadvantage on biobehavioral risk factors for diabetes. We also found support for a pathway model, with abdominal obesity emerging as a particularly strong mediator of the effect of early life SES disadvantage on glucoregulation. Thus, the effects of childhood SES disadvantage on adult glucoregulation are complex, including effects of a critical period of childhood SES on waist circumference and physical activity, as well as indirect effects through adult SES and waist circumference, physical activity, and depressive symptoms. These findings deepen our understanding of the ways that early life socioeconomic factors shape life course trajectories of SES disadvantage and unhealthy behaviors in adulthood that ultimately influence health.

Despite well-documented disparities in Type 2 diabetes, the majority of blacks and lower SES people do not have diabetes, highlighting the need for integrative studies that focus on the heterogeneity in diabetes risk. Emerging research has shown that the relationship between psychosocial vulnerabilities such as depressive symptoms, anger, and hostility and glucoregulation may be more prevalent among black women (Everson-Rose et al., 2004; Georgiades et al., 2009; Suarez, 2006; Surwit et al., 2002). We used data from the MIDUS 2 Biomarker subsample, including participants from the MIDUS 2 Milwaukee, Wisconsin, oversample of African Americans, to investigate whether anxiety was associated with glucose metabolism in people
p. 257 without diabetes (Tsenkova, Albert, Georgiades, & Ryff, 2012). Consistent with prior work, we found ↳ that black women were the only subgroup vulnerable to the effects of anxiety on glucose metabolism, underscoring the significance of targeting mental health in groups who are substantially less likely to receive mental health specialist care (Gum, King-Kallimanis, & Kohn, 2009; Kessler et al., 1994), as well as developing interventions that target anxiety along with established risk factors (e.g., weight and physical activity) for diabetes risk reduction in vulnerable groups.

We have summarized evidence that a dynamic interplay of biopsychosocial factors is associated with diabetes risk. Future work will further benefit from a life course approach to examining mechanisms and individual differences in this relationship. We need to understand both the intergenerational transmission among families, as well as the lingering influence of childhood experiences even among upward mobile individuals. Much important work remains, particularly in identifying resilience factors that mitigate diabetes risk among vulnerable adults. Understanding the social inequalities in diabetes necessitates

enriching the formulation with comprehensive assessments of disease status, socioeconomic circumstances over the life course, biobehavioral mediators, and psychosocial moderators.

Conclusion and Future Directions

The increasing prevalence of Type 2 diabetes is a public health crisis associated with considerable economic costs and human suffering, particularly among low SES groups and minorities. Many of the antecedent risk factors are well documented, but still leave considerable sources of variation unexplained, necessitating more research to better understand those at-risk people who do not fit the predicted gradient. MIDUS provided an invaluable resource for examining the reasons for individual differences in the relationship between risk factors and glucoregulation. We have shown that psychosocial factors can be a significant reason for vulnerability. Depressive symptoms, neuroticism, anger, weight discrimination can exacerbate diabetes risk among people already at higher risk due to being overweight or obese. Patients and providers can benefit from recognizing psychosocial factors are associated with diabetes risk. Their impact is accentuated in people already at some risk due to obesity or a sedentary life style. Conversely, there is some hope in the evidence that a powerful, nonmodifiable risk factor such as a familial history of diabetes can be offset by positive affect. This finding underscores the importance of acquiring a better understanding of resilience among people with preexisting risk factors. It illustrates the value of an individual differences approach that includes targeted psychosocial vulnerabilities and resources and risk factors from multiple domains.

The new MIDUS 3 Survey and Biomarker data will allow us to examine temporal changes in glucoregulation status, including the transition from prediabetes in MIDUS 2 to symptomatic diabetes in MIDUS 3. This opportunity to quantify change across comprehensive assessments of glucoregulation as a function of the dynamic interplay among sociodemographic factors, psychosocial functioning, and behavior is unprecedented. It will enrich our understanding of the biopsychosocial pathways to diabetes. Ultimately, prevention of diabetes will depend on understanding its preclinical progression, including how glucoregulation is contoured by antecedent factors that emanate from one's socioeconomic standing, psychosocial well-being, and health behaviors. Elucidating the risk and protective nexus from different domains will be key to understanding a multifactorial disease such as diabetes and better inform theory-based, person-centered interventions aimed at reducing risk. While the clinical importance of targeting physical activity and weight loss in people at risk for diabetes is clear, integrative approaches that also consider psychosocial vulnerabilities and promote psychological well-being will prove to be an equally powerful complement for preventing the progression to diabetes.

References

Abraham, S., Shah, N. G., Diez Roux, A., Hill-Briggs, F., Seeman, T., Szklo, M., . . . Golden, S. H. (2015). Trait anger but not anxiety predicts incident type 2 diabetes: The Multi-Ethnic Study of Atherosclerosis (MESA). *Psychoneuroendocrinology*, *60*, 105–113. doi:10.1016/j.psyneuen.2015.06.007
Google Scholar WorldCat

al'Absi, M., & Bongard, S. (2006). Neuroendocrine and behavioral mechanisms mediating the relationship between anger expression and cardiovascular risk: Assessment considerations and improvements. *Journal of Behavioral Medicine*, *29*(6), 573–591. doi:10.1007/s10865-006-9077-0
Google Scholar WorldCat

American Psychological Association. (2012, January 11). *Stress in America: Our health at risk* [Press release]. Washington, DC: Author.
Google Scholar Google Preview WorldCat COPAC

Attie, A. D. (2004). Genetic and genomic studies of Type 2 diabetes in mice. *International Congress Series*, *1262*, 220–223.
Google Scholar WorldCat

Bardenheier, B. H., Bullard, K. M., Caspersen, C. J., Cheng, Y. J., Gregg, E. W., & Geiss, L. S. (2013). A novel use of structural equation models to examine factors associated with prediabetes among adults aged 50 years and older: National Health and Nutrition Examination Survey 2001–2006. *Diabetes Care*, *36*(9), 2655–2662. doi:10.2337/dc12-2608
Google Scholar WorldCat

p. 258 Bienvenu, O. J., Nestadt, G., Samuels, J. F., Costa, P. T., Howard, W. T., & Eaton, W. W. (2001). Phobic, panic, and major depressive disorders and the five-factor model of personality. *Journal of Nervous and Mental Disease*, *189*(3), 154–161.
Google Scholar WorldCat

Boehm, J. K., Trudel-Fitzgerald, C., Kivimaki, M., & Kubzansky, L. D. (2015). The prospective association between positive psychological well-being and diabetes. *Health Psychology*, *34*(10), 1013–1021. doi:10.1037/hea0000200
Google Scholar WorldCat

Boylan, J. M., & Ryff, C. D. (2015). High anger expression exacerbates the relationship between age and metabolic syndrome. *Journals of Gerontology, Series B, Psychological Sciences and Social Sciences*, *70*(1), 77–82. doi:10.1093/geronb/gbt092
Google Scholar WorldCat

Centers for Disease Control and Prevention. (2014). *National diabetes statistics report: Estimates of diabetes and its burden in the United States, 2014*. Atlanta, GA: Author.
Google Scholar Google Preview WorldCat COPAC

Chen, Y., Rennie, D. C., & Dosman, J. A. (2010). Synergy of BMI and family history on diabetes: The Humboldt Study. *Public Health Nutrition*, *13*(4), 461–465. doi:10.1017/S1368980009991285
Google Scholar WorldCat

Cohen, B. E., Panguluri, P., Na, B., & Whooley, M. A. (2010). Psychological risk factors and the metabolic syndrome in patients with coronary heart disease: Findings from the Heart and Soul Study. *Psychiatry Research*, *175*(1–2), 133–137. doi:10.1016/j.psychres.2009.02.004
Google Scholar WorldCat

Cohen, S., & Janicki-Deverts, D. (2012). Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006, and 2009. *Journal of Applied Social Psychology*, *42*, 1320–1334.
Google Scholar WorldCat

Costa, P. T., & McCrae, R. R. (1992). Four ways five factors are basic. *Personality and Individual Differences*, *13*, 653–665.

Google Scholar WorldCat

Crandall, C. S., & Martinez, R. . (1997). Culture, ideology, and antifat attitudes. *Personality and Social Psychology Bulletin*, *22*, 1165–1176.
Google Scholar WorldCat

Dallman, M. F. (2010). Stress-induced obesity and the emotional nervous system. *Trends in Endocrinology & Metabolism*, *21*(3), 159–165. doi:10.1016/j.tem.2009.10.004
Google Scholar WorldCat

Dallman, M. F., Pecoraro, N., Akana, S. F., La Fleur, S. E., Gomez, F., Houshyar, H., . . . Manalo, S. (2003). Chronic stress and obesity: A new view of "comfort food." *Proceedings of the National Academy of Sciences of the United States of America*, *100*(20), 11696–11701. doi:10.1073/pnas.1934666100
Google Scholar WorldCat

DeFronzo, R. A. (2004). Pathogenesis of Type 2 diabetes mellitus. *Medical Clinics of North America*, *88*(4), 787–835, ix. doi:10.1016/j.mcna.2004.04.013
Google Scholar WorldCat

Del Parigi, A. (2010). Neuroanatomical correlates of hunger and satiety in lean and obese individuals. In L. Dube, A. Bechara, A. Dagher, A. Drewnowski, J. LeBel, P. James, & R. Yada (Eds.), *Obesity prevention* (Vol. 1, pp. 253–271). New York: Academic Press.
Google Scholar Google Preview WorldCat COPAC

Demakakos, P., Marmot, M., & Steptoe, A. (2012). Socioeconomic position and the incidence of Type 2 diabetes: The ELSA study. *European Journal of Epidemiology*, *27*(5), 367–378. doi:10.1007/s10654-012-9688-4
Google Scholar WorldCat

Diabetes Prevention Program Research Group, Knowler, W. C., Fowler, S. E., Hamman, R. F., Christophi, C. A., Hoffman, H. J., . . . Nathan, D. M. (2009). 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet*, *374*(9702), 1677–1686. doi:10.1016/S0140-6736(09)61457-4
Google Scholar WorldCat

Eckel, R. H., Kahn, S. E., Ferrannini, E., Goldfine, A. B., Nathan, D. M., Schwartz, M. W., . . . Smith, S. R. (2011). Obesity and type 2 diabetes: what can be unified and what needs to be individualized? *Diabetes Care*, *34*(6), 1424–1430. doi:10.2337/dc11-0447
Google Scholar WorldCat

Everson-Rose, S. A., Meyer, P. M., Powell, L. H., Pandey, D., Torrens, J. I., Kravitz, H. M., . . . Matthews, K. A. (2004). Depressive symptoms, insulin resistance, and risk of diabetes in women at midlife. *Diabetes Care*, *27*(12), 2856–2862.
Google Scholar WorldCat

Fanous, A., Gardner, C. O., Prescott, C. A., Cancro, R., & Kendler, K. S. (2002). Neuroticism, major depression and gender: A population-based twin study. *Psychological Medicine*, *32*(4), 719–728.
Google Scholar WorldCat

Fonseca, V. A. (2009). Defining and characterizing the progression of type 2 diabetes. *Diabetes Care*, *32* (Suppl. 2), S151–S156. doi:10.2337/dc09-S301
Google Scholar WorldCat

Franks, P. W. (2010). *Diabetes* family history: A metabolic storm you should not sit out. *Diabetes*, *59*(11), 2732–2734. doi:10.2337/db10-0768

Geiss, L. S., Pan, L., Cadwell, B., Gregg, E. W., Benjamin, S. M., & Engelgau, M. M. (2006). Changes in incidence of diabetes in US adults, 1997–2003. *American Journal of Preventive Medicine*, *30*(5), 371–377. doi:10.1016/j.amepre.2005.12.009
Google Scholar WorldCat

Georgiades, A., Lane, J. D., Boyle, S. H., Brummett, B. H., Barefoot, J. C., Kuhn, C. M., . . . Surwit, R. S. (2009). Hostility and fasting glucose in African American women. *Psychosomatic Medicine*, *71*(6), 642–645. doi:10.1097/PSY.0b013e3181acee3a
Google Scholar WorldCat

Gold, P. W., & Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: High versus low CRH/NE states. *Molecular Psychiatry*, *7*(3), 254–275. doi:10.1038/sj.mp.4001032
Google Scholar WorldCat

Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist*, *48*(1), 26–34.
Google Scholar WorldCat

Golden, S. H., Lazo, M., Carnethon, M., Bertoni, A. G., Schreiner, P. J., Diez Roux, A. V., . . . Lyketsos, C. (2008). Examining a bidirectional association between depressive symptoms and diabetes. *Journal of the American Medical Association*, *299*(23), 2751–2759. doi:10.1001/jama.299.23.2751
Google Scholar WorldCat

Golden, S. H., Williams, J. E., Ford, D. E., Yeh, H. C., Sanford, C. P., Nieto, F. J., & Brancati, F. L. (2006). Anger temperament is modestly associated with the risk of Type 2 diabetes mellitus: The Atherosclerosis Risk in Communities Study. *Psychoneuroendocrinology*, *31*(3), 325–332. doi:10.1016/j.psyneuen.2005.08.008
Google Scholar WorldCat

Goodrich, K. M., Crowley, S. K., Lee, D. C., Sui, X. S., Hooker, S. P., & Blair, S. N. (2012). Associations of cardiorespiratory fitness and parental history of diabetes with risk of Type 2 diabetes. *Diabetes Research and Clinical Practice*, *95*(3), 425–431. doi:10.1016/j.diabres.2011.10.045
Google Scholar WorldCat

Goodwin, R. D., Cox, B. J., & Clara, I. (2006). Neuroticism and physical disorders among adults in the community: Results from the National Comorbidity Survey. *Journal of Behavioral Medicine*, *29*(3), 229–238. doi:10.1007/s10865-006-9048-5
Google Scholar WorldCat

Gremigni, P. (2006). Cynical hostility and the metabolic syndrome: A case-control study. *Monaldi Archives for Chest Disease*, *66*(3), 224–229.
Google Scholar WorldCat

Gum, A. M., King-Kallimanis, B., & Kohn, R. (2009). Prevalence of mood, anxiety, and substance-abuse disorders for older Americans in the national comorbidity survey-replication. *American Journal of Geriatric Psychiatry*, *17*(9), 769–781. doi:10.1097/JGP.0b013e3181ad4f5a
Google Scholar WorldCat

p. 259 Hariri, S., Yoon, P. W., Qureshi, N., Valdez, R., Scheuner, M. T., & Khoury, M. J. (2006). Family history of type 2 ↳ diabetes: a population-based screening tool for prevention? *Genetics in Medicine*, *8*(2), 102–108. doi:10.109701.gim.0000200949.52795.df

Haukkala, A., Konttinen, H., Laatikainen, T., Kawachi, I., & Uutela, A. (2010). Hostility, anger control, and anger expression as predictors of cardiovascular disease. *Psychosomatic Medicine*, *72*(6), 556–562. doi:10.1097/PSY.0b013e3181dbab87
Google Scholar WorldCat

Hilding, A., Eriksson, A. K., Agardh, E. E., Grill, V., Ahlbom, A., Efendic, S., & Ostenson, C. G. (2006). The impact of family history of diabetes and lifestyle factors on abnormal glucose regulation in middle-aged Swedish men and women. *Diabetologia*, *49*(11), 2589–2598. doi:10.1007/s00125-006-0402-5
Google Scholar WorldCat

InterAct Consortium, Scott, R. A., Langenberg, C., Sharp, S. J., Franks, P. W., Rolandsson, O., . . . Wareham, N. J. (2013). The link between family history and risk of type 2 diabetes is not explained by anthropometric, lifestyle or genetic risk factors: The EPIC-InterAct study. *Diabetologia*, *56*(1), 60–69. doi:10.1007/s00125-012-2715-x

Google Scholar WorldCat

Johnson, R. C., & Schoeni, R. F. (2011). Early-life origins of adult disease: National longitudinal population-based study of the United States. *American Journal of Public Health*, *101*(12), 2317–2324. doi:10.2105/AJPH.2011.300252
Google Scholar WorldCat

Joseph, J. J., Wang, X., Spanakis, E., Seeman, T., Wand, G., Needham, B., & Golden, S. H. (2015). Diurnal salivary cortisol, glycemia and insulin resistance: The multi-ethnic study of atherosclerosis. *Psychoneuroendocrinology*, *62*, 327–335. doi:10.1016/j.psyneuen.2015.08.021
Google Scholar WorldCat

Kan, C., Silva, N., Golden, S. H., Rajala, U., Timonen, M., Stahl, D., & Ismail, K. (2013). A systematic review and meta-analysis of the association between depression and insulin resistance. *Diabetes Care*, *36*(2), 480–489. doi:10.2337/dc12-1442
Google Scholar WorldCat

Kanjilal, S., Gregg, E. W., Cheng, Y. J., Zhang, P., Nelson, D. E., Mensah, G., & Beckles, G. L. (2006). Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971–2002. *Archives of Internal Medicine*, *166*(21), 2348–2355. doi:10.1001/archinte.166.21.2348
Google Scholar WorldCat

Kavanagh, A., Bentley, R. J., Turrell, G., Shaw, J., Dunstan, D., & Subramanian, S. V. (2010). Socioeconomic position, gender, health behaviours and biomarkers of cardiovascular disease and diabetes. *Social Science and Medicine*, *71*(6), 1150–1160. doi:10.1016/j.socscimed.2010.05.038
Google Scholar WorldCat

Kessler, R. C., McGonagle, K. A., Zhao, S., Nelson, C. B., Hughes, M., Eshleman, S., . . . Kendler, K. S. (1994). Lifetime and 12-month prevalence of *DSM-III-R* psychiatric disorders in the United States. Results from the National Comorbidity Survey. *Archives of General Psychiatry*, *51*(1), 8–19.

Kessler, R. C., Mickelson, K. D., Walters, E. E., Zhao, S., & Hamilton, L. (2004). Age and depression in the MIDUS survey. In O. G. Brim, C. D. Ryff, & R. C. Kessler (Eds.), *How healthy are we? A national study of well-being at midlife* (pp. 227–251). Chicago: University of Chicago Press.
Google Scholar Google Preview WorldCat COPAC

Knol, M. J., Twisk, J. W., Beekman, A. T., Heine, R. J., Snoek, F. J., & Pouwer, F. (2006). Depression as a risk factor for the onset of Type 2 diabetes mellitus. *A meta-analysis*. *Diabetologia*, *49*(5), 837–845. doi:10.1007/s00125-006-0159-x
Google Scholar WorldCat

Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., Walker, E. A., . . . Diabetes Prevention Program Research Group. (2002). Reduction in the incidence of Type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine*, *346*(6), 393–403. doi:10.1056/NEJMoa012512
Google Scholar WorldCat

Koh, K. B., Choe, K. O., & An, S. K. (2003). Anger and coronary calcification in individuals with and without risk factors of coronary artery disease. *Yonsei Medical Journal*, *44*(5), 793–799.
Google Scholar WorldCat

Kumari, M., Head, J., & Marmot, M. (2004). Prospective study of social and other risk factors for incidence of Type 2 diabetes in the Whitehall II study. *Archives of Internal Medicine*, *164*(17), 1873–1880.
Google Scholar WorldCat

Lahey, B. B. (2009). Public health significance of neuroticism. *American Psychologist*, *64*(4), 241–256.
Google Scholar WorldCat

Liu, C. S., Carvalho, A. F., & McIntyre, R. S. (2014). Towards a “metabolic” subtype of major depressive disorder: Shared pathophysiological mechanisms may contribute to cognitive dysfunction. *CNS & Neurological Disorders: Drug Targets*, *13*(10), 1693–1707.
Google Scholar WorldCat

Lustman, P. J., Anderson, R. J., Freedland, K. E., de Groot, M., Carney, R. M., & Clouse, R. E. (2000). Depression and poor glycemic control: A meta-analytic review of the literature. *Diabetes Care*, *23*(7), 934–942.
Google Scholar WorldCat

Merjonen, P., Pulkki-Raback, L., Puttonen, S., Keskivaara, P., Juonala, M., Telama, R., . . . Keltikangas-Jarvinen, L. (2008). Anger is associated with subclinical atherosclerosis in low SES but not in higher SES men and women. The Cardiovascular Risk in Young Finns Study. *Journal of Behavioral Medicine*, *31*(1), 35–44. doi:10.1007/s10865-007-9131-6
Google Scholar WorldCat

Mezuk, B., Eaton, W. W., Albrecht, S., & Golden, S. H. (2008). Depression and Type 2 diabetes over the lifespan: A meta-analysis. *Diabetes Care*, *31*(12), 2383–2390. doi:10.2337/dc08-0985
Google Scholar WorldCat

Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., . . . Muller, J. E. (1995). Triggering of acute myocardial infarction onset by episodes of anger. Determinants of Myocardial Infarction Onset Study Investigators. *Circulation*, *92*(7), 1720–1725.
Google Scholar WorldCat

Moskowitz, J. T., Epel, E. S., & Acree, M. (2008). Positive affect uniquely predicts lower risk of mortality in people with diabetes. *Health Psychology*, *27*(1, Suppl.), S73–S82. doi:10.1037/0278-6133.27.1.S73
Google Scholar WorldCat

Narayan, K. M., Boyle, J. P., Geiss, L. S., Saaddine, J. B., & Thompson, T. J. (2006). Impact of recent increase in incidence on future diabetes burden: US, 2005–2050. *Diabetes Care*, *29*(9), 2114–2116. doi:10.2337/dc06-1136
Google Scholar WorldCat

Nefs, G., Pouwer, F., Denollet, J., Kramer, H., Wijnands-van Gent, C. J., & Pop, V. J. (2012). Suboptimal glycemic control in Type 2 diabetes: A key role for anhedonia? *Journal of Psychiatric Research*, *46*(4), 549–554. doi:10.1016/j.jpsychires.2012.01.013
Google Scholar WorldCat

Nelson, T. L., Palmer, R. F., & Pedersen, N. L. (2004). The metabolic syndrome mediates the relationship between cynical hostility and cardiovascular disease. *Experimental Aging Research*, *30*(2), 163–177. doi:10.1080/03610730490275148
Google Scholar WorldCat

Niaura, R., Banks, S. M., Ward, K. D., Stoney, C. M., Spiro, A., 3rd, Aldwin, C. M., . . . Weiss, S. T. (2000). Hostility and the metabolic syndrome in older males: The normative aging study. *Psychosomatic Medicine*, *62*(1), 7–16.
Google Scholar WorldCat

Nouwen, A., Nefs, G., Caramlau, I., Connock, M., Winkley, K., Lloyd, C. E., . . . European Depression in Diabetes Research, C. (2011). Prevalence of depression in individuals with impaired glucose metabolism or undiagnosed diabetes: A systematic review and meta-analysis of the European Depression in Diabetes (EDID) Research Consortium. *Diabetes Care*, *34*(3), 752–762. doi:10.2337/dc10-1414
Google Scholar WorldCat

p. 260 Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). Prevalence of obesity in the United States, 2009–2010. *NCHS Data Brief,* (82), 1–8.
Google Scholar WorldCat

Okely, J. A., & Gale, C. R. (2016). Well-being and chronic disease incidence: The English Longitudinal Study of Ageing. *Psychosomatic Medicine*, *78*(3), 335–344. doi:10.1097/PSY.0000000000000279

Google Scholar WorldCat

Oliver, G., Wardle, J., & Gibson, E. L. (2000). Stress and food choice: A laboratory study. *Psychosomatic Medicine*, *62*(6), 853–865.
Google Scholar WorldCat

Phillips, A. C., Batty, G. D., Weiss, A., Deary, I., Gale, C. R., Thomas, G. N., & Carroll, D. (2010). Neuroticism, cognitive ability, and the metabolic syndrome: The Vietnam Experience Study. *Journal of Psychosomatic Research*, *69*(2), 193–201. doi:10.1016/j.jpsychores.2010.01.016
Google Scholar WorldCat

Puhl, R., & Brownell, K. D. (2001). Bias, discrimination, and obesity. *Obesity Research*, *9*(12), 788–805. doi:10.1038/oby.2001.108
Google Scholar WorldCat

Puhl, R. M., & Heuer, C. A. (2009). The stigma of obesity: A review and update. *Obesity (Silver Spring)*, *17*(5), 941–964. doi:10.1038/oby.2008.636
Google Scholar WorldCat

Raikkonen, K., Matthews, K. A., & Kuller, L. H. (2002). The relationship between psychological risk attributes and the metabolic syndrome in healthy women: Antecedent or consequence? *Metabolism*, *51*(12), 1573–1577. doi:10.1053/meta.2002.36301
Google Scholar WorldCat

Rasmussen, N. H., Smith, S. A., Maxson, J. A., Bernard, M. E., Cha, S. S., Agerter, D. C., & Shah, N. D. (2013). Association of HbA_{1c} with emotion regulation, intolerance of uncertainty, and purpose in life in type 2 diabetes mellitus. *Primary Care Diabetes*, *7*(3), 213–221. doi:10.1016/j.pcd.2013.04.006

Richman, L. S., Kubzansky, L., Maselko, J., Kawachi, I., Choo, P., & Bauer, M. (2005). Positive emotion and health: Going beyond the negative. *Health Psychology*, *24*(4), 422–429. doi:10.1037/0278-6133.24.4.422
Google Scholar WorldCat

Roberts, S. B., & Kendler, K. S. (1999). Neuroticism and self-esteem as indices of the vulnerability to major depression in women. *Psychological Medicine*, *29*(5), 1101–1109.
Google Scholar WorldCat

Rodin, M., Price, J., Sanchez, F., & McElliot, S. (1989). Derogation, exclusion, and unfair treatment of persons with social flaws: controllability of stigma and the attribution of prejudice. *Personality and Social Psychology Bulletin*, *15*, 439–451.
Google Scholar WorldCat

Rosmond, R., & Bjorntorp, P. (2000). The hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, Type 2 diabetes and stroke. *Journal of Internal Medicine*, *247*(2), 188–197.
Google Scholar WorldCat

Ruini, C., & Ryff, C. D. (2016). Using eudaimonic well-being to improve lives. In A. Wood & J. Johnson (Eds.), *Wiley Handbook of Positive Clinical Psychology* (pp. 153–166). New York: Wiley.
Google Scholar Google Preview WorldCat COPAC

Schmitz, N., Deschenes, S. S., Burns, R. J., Smith, K. J., Lesage, A., Strychar, I., . . . Wang, J. L. (2016). Depression and risk of type 2 diabetes: The potential role of metabolic factors. *Molecular Psychiatry*, *21*(12), 1726–1732. doi:10.1038/mp.2016.7
Google Scholar WorldCat

Spielberger, C. D. (1996). *State-Trait Anger Expression Inventory: Professional manual*. Odessa, FL: Psychological Assessment Resource.
Google Scholar Google Preview WorldCat COPAC

Stetler, C., & Miller, G. E. (2011). Depression and hypothalamic-pituitary-adrenal activation: A quantitative summary of four

decades of research. *Psychosomatic Medicine*, *73*(2), 114–126. doi:10.1097/PSY.0b013e31820ad12b
Google Scholar WorldCat

Suarez, E. C. (2006). Sex differences in the relation of depressive symptoms, hostility, and anger expression to indices of glucose metabolism in nondiabetic adults. *Health Psychology*, *25*(4), 484–492. doi:10.1037/0278-6133.25.4.484
Google Scholar WorldCat

Sullivan, M. D., O'Connor, P., Feeney, P., Hire, D., Simmons, D. L., Raisch, D. W., . . . Katon, W. J. (2012). Depression predicts all-cause mortality: Epidemiological evaluation from the ACCORD HRQL substudy. *Diabetes Care*, *35*(8), 1708–1715. doi:10.2337/dc11-1791
Google Scholar WorldCat

Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin*, *131*(2), 260–300. doi:10.1037/0033-2909.131.2.260
Google Scholar WorldCat

Surwit, R. S., Williams, R. B., Siegler, I. C., Lane, J. D., Helms, M., Applegate, K. L., . . . Barefoot, J. C. (2002). Hostility, race, and glucose metabolism in nondiabetic individuals. *Diabetes Care*, *25*(5), 835–839.
Google Scholar WorldCat

Sutin, A. R., Costa, P. T., Jr., Uda, M., Ferrucci, L., Schlessinger, D., & Terracciano, A. (2010). Personality and metabolic syndrome. *Age (Dordrecht)*, *32*(4), 513–519. doi:10.1007/s11357-010-9153-9
Google Scholar WorldCat

Sutin, A. R., Stephan, Y., & Terracciano, A. (2015). Weight discrimination and risk of mortality. *Psychological Science*, *26*(11), 1803–1811. doi:10.1177/0956797615601103
Google Scholar WorldCat

Tamayo, T., Christian, H., & Rathmann, W. (2010). Impact of early psychosocial factors (childhood socioeconomic factors and adversities) on future risk of Type 2 diabetes, metabolic disturbances and obesity: A systematic review. *BMC Public Health*, *10*, 525. doi:10.1186/1471-2458-10-525
Google Scholar WorldCat

Tannenbaum, B., Anisman, H., & Abizaid, A. (2010). Neuroendocrine stress response and its impact on eating behavior and body weight. In L. Dube, A. Bechara, A. Dagher, A. Drewnowski, J. LeBel, P. James, & R. Yada (Eds.), *Obesity Prevention* (pp. 261–271). New York: Academic Press.
Google Scholar Google Preview WorldCat COPAC

Tsenkova, V., Boylan, J. M., & Ryff, C. (2013). Stress eating and health. Findings from MIDUS, a national study of US adults. *Appetite*, *69*, 151–155. doi:10.1016/j.appet.2013.05.020
Google Scholar WorldCat

Tsenkova, V., Pudrovska, T., & Karlamangla, A. (2014). Childhood socioeconomic disadvantage and prediabetes and diabetes in later life: A study of biopsychosocial pathways. *Psychosomatic Medicine*, *76*(8), 622–628. doi:10.1097/PSY.0000000000000106
Google Scholar WorldCat

Tsenkova, V. K., Albert, M. A., Georgiades, A., & Ryff, C. D. (2012). Trait anxiety and glucose metabolism in people without diabetes: Vulnerabilities among black women. *Diabetic Medicine*, *29*(6), 803–806. doi:10.1111/j.1464-5491.2011.3534.x
Google Scholar WorldCat

Tsenkova, V. K., Carr, D., Coe, C. L., & Ryff, C. D. (2012). Synergistic effect of neuroticism and body mass index on glucose metabolism in nondiabetic adults. *Psychotherapy and Psychosomatics*, *81*(5), 327–328. doi:10.1159/000337413
Google Scholar WorldCat

Tsenkova, V. K., Carr, D., Coe, C. L., & Ryff, C. D. (2014). Anger, adiposity, and glucose control in nondiabetic adults: Findings from

MIDUS II. *Journal of Behavioral Medicine*, *37*(1), 37–46. doi:10.1007/s10865-012-9460-y
Google Scholar WorldCat

Tsenkova, V. K., Carr, D., Schoeller, D. A., & Ryff, C. D. (2011). Perceived weight discrimination amplifies the link between central adiposity and nondiabetic glycemic control (HbA_{1c}). *Annals of Behavioral Medicine*, *41*(2), 243–251. doi:10.1007/s12160-010-9238-9

Tsenkova, V. K., Dienberg Love, G., Singer, B. H., & Ryff, C. D. (2008). Coping and positive affect predict ↳ longitudinal change in glycosylated hemoglobin. *Health Psychology*, *27*(2, Suppl.), S163–S171. doi:10.1037/0278-6133.27.2(Suppl.).S163

Tsenkova, V. K., & Karlamangla, A. (2016). Depression amplifies the influence of central obesity on 10-year incidence of diabetes: Findings from MIDUS. *PLoS One*, *11*(10), e0164802. doi:10.1371/journal.pone.0164802
Google Scholar WorldCat

Tsenkova, V. K., Karlamangla, A. S., & Ryff, C. D. (2016). Parental history of diabetes, positive affect, and diabetes risk in adults: Findings from MIDUS. *Annals of Behavioral Medicine*, *50*(6), 836–843. doi:10.1007/s12160-016-9810-z
Google Scholar WorldCat

Vadiveloo, M., & Mattei, J. (2017). Perceived weight discrimination and 10-year risk of allostatic load among US adults. *Annals of Behavioral Medicine*, *51*(1), 94–104. doi:10.1007/s12160-016-9831-7
Google Scholar WorldCat

Valdez, R., Yoon, P. W., Liu, T., & Khoury, M. J. (2007). Family history and prevalence of diabetes in the US population: 6-year results from the National Health and Nutrition Examination Survey (NHANES, 1999–2004). *Diabetes*, *30*(10), 2517–2522. doi:10.2337/db07-0720x
Google Scholar WorldCat

van Dam, R. M., Boer, J. M., Feskens, E. J., & Seidell, J. C. (2001). Parental history of diabetes modifies the association between abdominal adiposity and hyperglycemia. *Diabetes Care*, *24*(8), 1454–1459.
Google Scholar WorldCat

van Strien, T., & Ouwens, M. A. (2003). Counterregulation in female obese emotional eaters: Schachter, Goldman, and Gordon's (1968) test of psychosomatic theory revisited. *Eating Behaviors*, *3*(4), 329–340.
Google Scholar WorldCat

Velasco Mondragon, H. E., Charlton, R. W., Peart, T., Burguete-Garcia, A. I., Hernandez-Avila, M., & Hsueh, W. C. (2010). Diabetes risk assessment in Mexicans and Mexican Americans: Effects of parental history of diabetes are modified by adiposity level. *Diabetes Care*, *33*(10), 2260–2265. doi:10.2337/dc10-0992
Google Scholar WorldCat

Vogelzangs, N., Beekman, A. T., Boelhouwer, I. G., Bandinelli, S., Milaneschi, Y., Ferrucci, L., & Penninx, B. W. (2011). Metabolic depression: A chronic depressive subtype? Findings from the InCHIANTI study of older persons. *Journal of Clinical Psychiatry*, *72*(5), 598–604. doi:10.4088/JCP.10m06559
Google Scholar WorldCat

Wikner, C., Gigante, B., Hellenius, M. L., de Faire, U., & Leander, K. (2013). The risk of type 2 diabetes in men is synergistically affected by parental history of diabetes and overweight. *PLoS One*, *8*(4), e61763. doi:10.1371/journal.pone.0061763
Google Scholar WorldCat

Williams, J. E., Paton, C. C., Siegler, I. C., Eigenbrodt, M. L., Nieto, F. J., & Tyroler, H. A. (2000). Anger proneness predicts coronary heart disease risk: Prospective analysis from the atherosclerosis risk in communities (ARIC) study. *Circulation*, *101*(17), 2034–2039.
Google Scholar WorldCat

Wouts, L., Janzing, J. G., Lampe, I. K., Franke, B., de Vegt, F., Tendolkar, I., . . . Oude Voshaar, R. C. (2011). The interaction between cerebrovascular disease and neuroticism in late-life depression: A cross-sectional study. *International Journal of Geriatric Psychiatry*, *26*(7), 702–710. doi:10.1002/gps.2584 ↳
Google Scholar WorldCat