



Review Article

Strategies for Reducing the Risk of Diabetes Associated with Depression

Melissa Scollan-Koliopoulos^{1*}, Donna Naturale²

¹Dr. Susan L. Davis, R.N. & Richard J. Henley College of Nursing, Department of Online Graduate Studies, Sacred Heart University, USA

²School of Nursing and Health Services. Department of Nursing and Public Health, Caldwell University, USA

*Corresponding author: Melissa Scollan-Koliopoulos, Dr. Susan L. Davis, R.N. & Richard J. Henley College of Nursing, Sacred Heart University, USA

Citation: Scollan-Koliopoulos M and Naturale D (2024) Strategies for Reducing the Risk of Diabetes Associated with Depression. Int J Nurs Health Care Res 7:1596. DOI: <https://doi.org/10.29011/2688-9501.101596>

Received Date: 18 October, 2024; Accepted Date: 25 October, 2024; Published Date: 28 October, 2024

Abstract

Objectives: The purpose of this review is to create awareness of the association between depression and the risk for developing diabetes. **Methods:** Major nursing, health and science databases (PROQUEST, EBSCO, CINHAL, Google scholar) were searched to locate articles that can explain the etiology of the association between depression and diabetes and to determine what interventions may be available for nurses to utilize reduce the risk of diabetes.

Results: Interventions that result in lowered glucose levels include lifestyle interventions such as exercise, stress management, and psychotherapy to reduce depression that can impact the development of diabetes by improving depression levels. **Conclusion:** When nurses identify that a patient is depressed there is an opportunity to to deliver psychoeducation regarding interventions that serve the purpose of reducing depression levels and may simultaneously prevent diabetes through lifestyle interventions.

Key words: Depression; Diabetes; Prediabetes Diabetes Prevention; Lifestyle

Introduction

Diabetes mellitus has long been associated with comorbid depression. Once referred to as “sweet sorrow” [1], diabetes was defined by the Mid-1600’s physician Thomas Willis who attributed diabetes as a disposition of “*sadness or long sorrow and other depressions*” [1]. The association between diabetes and mental illness has been recognized for over 350 years [1]. Previously thought to be a linear association between diabetes and depression depicted as “*Both psychological and humoral mechanisms could play a role in this apparent increased risk of depression among diabetic patients. The clear psychosocial demands and threats of this chronic illness are the most obvious likely precipitants of psychiatric illness (pp 441)*” [2]. Since the time of Sir Willis,

the knowledge regarding the relationship between diabetes and depression has evolved to be more reciprocal than linear with emerging evidence that depression itself is an actual predictor to the onset of diabetes [3][4]. Identifying depression by being aware of the symptoms is an opportunity to discuss the risk for diabetes and address lifestyle factors that could positively impact the trajectory of both depression and the risk of diabetes. The purpose of this paper is to raise awareness of the relationship between depression and the risk of diabetes and lifestyle-based prevention.

Background

People with diabetes are two-to-three times more likely to have depression than people without diabetes. With one in five adults estimated to be affected by diabetes, the association may even be underestimated given that only 25-50% of people with diabetes and co-morbid depression receive an initial diagnosis and treatment for

their depression [5]. The relative risk of developing diabetes if one is depressed is 1.5-2.0 times greater than in those unaffected by diabetes [6]. The risk of developing diabetes in those depressed is reduced in a stepwise fashion indicating the importance of reinforcing modifiable health lifestyle factors in those who are depressed [6]. Depression increases the risk of type 2 diabetes using Mendelian causal models (odds ratio 1.26) with 36.5% of the effect being mediated by body mass index [3]. Recent genetic studies have revealed that immune function, tau protein formation, and cellular aging have overlapping architecture in both depression and diabetes [7]. The age of diagnosis of diabetes is younger in those with higher depressive symptoms [8] with depression predating diabetes onset by 8-10 years most of the cases studied [9]. In one epidemiologic study, hazard ratio analyses showed an association between depression and an increased risk of diabetes, stratified by depressive symptoms (17% increase), diagnosis (20% increase), and antidepressant use (19% increase) [10].

Defining Characteristics of Depression

The American Psychiatric Association defines depression as symptoms that last for at least two weeks that represent a previous change in one's functioning that is not otherwise explained by a medical condition that mimics depression, such as thyroid problems, vitamin deficiencies, or a brain tumor. The symptoms include: **1)** feeling sad or having a depressed mood; **2)** loss of interest or pleasure in activities once enjoyed; **3)** changes in appetite-weight loss or gain unrelated to dieting; **4)** trouble sleeping or sleeping too much; **5)** loss of energy or increased fatigue; **6)** increased purposeless physical activity (e.g., inability to sit still, pacing, handwringing) or slowed movements or speech (severe enough to be observed by others); **7)** feeling worthless or guilty; **8)** difficulty thinking, concentrating, or making decision; **9)** thoughts of suicide or death [11]. Asking patients the first two questions regarding sadness or loss of interest is predictive enough of a diagnosis [12].

Defining Characteristics of Diabetes

The World Health Organization defines diabetes mellitus as a chronic, metabolic disease, characterized by elevations of blood glucose leading over time to serious damage to the heart, blood vessels, eyes, kidneys, and nerves [13]. Type 2 diabetes is preventable with policies and practices across whole populations that contribute to good health such as regular exercise, healthy eating, smoking avoidance, blood pressure, and lipid controlling behaviors [13]. The World Health Organization defines overweight and obesity as abnormal or excessive fat accumulation presenting a health risk. A Body Mass Index (BMI) over 25 is considered overweight and a BMI over 30 is considered obesity with a global burden of disease [13]. The type 2 diabetes syndrome is largely

hereditary like depression, with a 26% risk of prediabetes if one's parent had diabetes [14]. Observational studies have even shown that in the context of the predisposition for hereditary-based diabetes, positive affect is shown to be associated with a reduced incidence of diabetes development [15]. Approximately, 12 million people in the United States are at risk for diabetes and go unscreened [16]. Screening patients for a fasting glucose or glycosylated haemoglobin value can help detect pre-diabetes or diabetes [16]. Screening could be offered at the time people are identified as being depressed and offered screening with the basis being to offer lifestyle interventions such as exercise, such as brisk walking shown to treat both depression [17] and hyperglycaemia [18].

Pathophysiologic Relationship between Depression and Diabetes

Neuroendocrine explanations for a reciprocal association between diabetes and depression include hypothalamic-pituitary-adrenocortical axis dysfunction is hypothesized to be similar in both conditions, as evidenced by abnormal cortisol production observed in both diabetes and depression [19]. Depression is the cumulative result of interactions in the central and peripheral nervous system, endocrine system, genetics, and environmental factors [20]. The cause of major depression is largely explained by genetics, with 30-40% variance, with the remainder by a combination of genetic and environmental interactions. Psychosocial factors, monoaminergic function, stress hormones, such as serotonin, norepinephrine, dopamine, glutamate, and gamma-aminobutyric acid, and circadian rhythms are felt to play a role in predicting a familial pattern in depression [21]. Other findings associating depression onset with inflammation from stress include elevated C-reactive protein, tumor necrosis factor alpha, and/or interleukin 6 elevations [22].

The relationship between depression and diabetes is explainable by the hypothalamus-pituitary axis model of stress hormones and its influence on blood glucose control [19]. Norepinephrine and epinephrine regulate our stress and fear responses, play a role in the emotion of excitement, with regulation of heart rate and blood pressure and metabolic rate [23]. Blood glucose changes are messaged by the catecholamines facilitating the liver's release of glucose from glycogen stores, via the counterregulatory glucagon [24]. Other factors that do not have clear interactions relevant to depression may also play a role in Monoamine Oxidase Inhibitor (MAOI) neurotransmitter function. Insulin growth factor I works with insulin to deliver glucose to muscle and brain tissue [24]. Additional factors include, brain derived neurotrophic factors and vascular endothelial growth factors and fibroblast growth factors that help with neurogenesis and maintaining a healthy brain structure. Whereas Muscarinic acetylcholine --M3

and M5-- antagonists play a role in metabolic dysregulation, dyslipidemia and diabetes as seen in the atypical antipsychotics [25]. Neurotransmitters play a role in glucose homeostasis [25] with Stress hormones play a role in the development of depression and are hypothesized with the onset of diabetes.

In populations with diagnosed diabetes, mental health screening and treatment are shown to improve diabetes management, disease outcomes, and quality-of-life [26]. Glucose metabolism in the brain in unipolar depression is reduced in many regions of the frontotemporal parts of the brain and this is correlated with the degree of hypometabolism and illness severity and is normalized with serotonin reuptake inhibitor treatment [27]. Given the pervasiveness and complexity of depression in those with comorbid diabetes, patient education providers have the potential to improve outcomes by integrating best practices from diabetes education and mental health counselling fields.

Modifying Risk Factors Associated with Diabetes and Depression

Generally, obesity is recognized as modifiable and a leading risk factor for the development of diabetes [28]. Diabetes can be prevented through lifestyle interventions that reduce BMI despite a co-morbidity of depression [29]. There is an opportunity for clinicians to deliver targeted psychoeducation to individuals with depression to reduce the prospective risk of obesity and diabetes. Currently, the National Diabetes Prevention Program includes education on stress reduction (<https://www.cdc.gov/diabetes/prevention/lcp-details.html>). Stress reduction is known to contribute modifiable risk factors for diabetes [30].

Proactive measures to identify and reduce modifiable risk factors for the development of diabetes in those affected by depression are meaningful. In those affected by depression, targeted interventions to reduce obesity could prevent or delay the onset of diabetes and would improve health outcomes and reduce the burden on public health. Depression is a potentially a modifiable risk factor when it is remissible through psychotherapy and pharmacologic treatment [31]. However, the etiology of the co-morbidity remains unconfirmed, and it is unknown if depression management or reversal itself can reduce the risk of developing diabetes. A modifiable risk factor for diabetes that commonly affects those with depression is obesity [28]. It is even possible that factors such as anhedonia from depression contribute to less motivation to exercise. Nonetheless, we know that lifestyle changes that reduce obesity, such as exercise and increased movement positively impact depression with a reduction in severity [32]. Exercise has been proposed as a prescriptive treatment for both diabetes and depression prevention and treatment [33]. Clinicians who care for individuals with depression have an opportunity to address risk factors for the development of diabetes, such as obesity.

Prediabetes is a syndrome of rising blood glucose above the normal range and is largely predicted by being overweight. In fact, the Norfolk Diabetes Prevention Study (NDPS), the largest diabetes prevention study in the world following the United States Diabetes Prevention program study, showed that just a few 2-3 kilograms of weight loss reduces the risk of diabetes by 40-47 per cent [34].

Sampson et al. (2020) further showed that hyperglycemia could be reduced be it from diabetes or non-diabetes-related causes [34]. This is relevant to those on medications to manage depression and mood disorders because many have been shown to cause hyperglycemia independent of risk for developing diabetes [35]. Simple interventions, such as referral to Weight Watchers™ by general practitioners was shown to reduce diabetes onset by 1/3 in one study [36].

Nurses are in a position that could be deemed nonthreatening to the patient allowing for consciousness-raising regarding modifiable risk factors that pose harm to one's wellbeing. A patient may respond to psychoeducation efforts to address risk by body mass index, habitually poor dietary habits and/or lack of exercise within the context of routine depression care because there is a pre-existing authentic and trusting relationship that is presumed to be "stigma-free" [37]. Mental health professionals play a significant adjunctive psychotherapeutic role for obese individuals when dealing with mood disorders, including psychological evaluation for disordered eating, body dysmorphic states, the reality of weight loss, psychological symptoms that emerge during dieting, interventions for pre and post bariatric surgery, cognitive behavioral techniques, stimulus control, self-monitoring of lifestyle changes, goal-setting, restructuring of negative and self-defeating thoughts, and insight oriented therapy for dealing with eating-conflicts, the meaning of weight loss, and prejudice [37].

Since the Diabetes Prevention Program (DPP) was first implemented by the Center for Disease Control, mental health providers have successfully diffused and implemented translatable forms of the DPP into mental healthcare settings to prevent obesity and diabetes [38]. Generally, interdisciplinary-led weight loss interventions in those with serious mental illness is effective [39]. In addition, those with serious mental illness receive fewer primary care services [40]. Potential benefits of integrating diabetes prevention education during episodic mental health care visits would include an opportunity for diagnosis, dietary and physical activity behavior changes, weight loss, and accurate perceived risk for diabetes.

The landmark randomized controlled trial, the Diabetes Prevention Program (DPP), conducted across the United States, showed that type 2 diabetes could be prevented through intensive diet and physical activity and/or pharmacotherapy with metformin. The

program was based on a randomized controlled national trial that implemented a lifestyle intervention to increase physical activity and improve eating habits [29]. A secondary analysis assessed depressive symptoms and antidepressant medication use in DPP participants. The DPP concluded that an intensive lifestyle intervention did not make depression worse in participants and showed a positive psychological impact of intensive efforts to prevent diabetes. Those in the DPP who lost some weight and were more active had small but significant reductions in depression [29]. The DPP did not show a risk of diabetes development in those who were on antidepressants. This may mean the relationship between depression and eventual diabetes development is not due to antidepressants and/or the side effect of weight gain or fatigue. Perhaps the relationship between depression and diabetes is more intrinsically endocrinologic in nature [41].

A prospective collaborative depression treatment program study showed that the 9-year follow-up of the Improving Mood-Promoting Access to Collaborative Treatment (IMPACT) randomized controlled trial showed that depression treatment alone does not prevent the onset of diabetes despite reducing depression symptoms (adjusted cox proportional hazards model HR=1.18, 95% CI: 0.61-2.29, $p=.616$) [42]. This indicates that although the relationship of depression is a robust predictor of diabetes prevalence, routine treatment for depression alone without an established intervention to specifically reduce glucose levels is not likely to prevent diabetes [42].

Most of the diabetes prevention interventions targeting people with diabetes have occurred in primary care. For example, Yates, et al. (2019) found that the burden of depressive symptoms in primary care reduced effects of a physical activity intervention for diabetes prevention [32]. When it comes to tailored lifestyle interventions, the choice of physical activity walking is low cost, accessible, and requires the least supervision. Physical activity is demonstrated to improve motor cortex neuroplasticity in depressed individuals above and beyond the effects of antidepressants ($r=-0.835$, $p<0.001$) [43].

When it comes to diet and depression, the RAINBOW Randomized Clinical Trial showed benefits in reduction of body mass index and depression scores were modest after a year-long intervention. The intervention was a translation of the Diabetes Prevention Program which, included targeting a weight loss of 5-10% through healthy dietary changes by reducing calories by 500-1,000 a day and adding 150 minutes of moderate intensity activity per week (ie. walking) [44]. The study included antidepressant medications, which may have made it difficult to detect the effects of the lifestyle intervention due to weight gain caused by some medications [44]. The comparison was to usual care in those who raise concern

over antidepressant weight gain, receiving some sort of dietary counselling.

A meta-analysis of lifestyle interventions in those with severe mental illness revealed a reduction of body mass index by .63 kg/m² or 2.2 kg with a reduction in waist circumference. Those receiving interventions had a 50% likelihood for losing weight compared to the control groups [45].

The Diabetes Prevention program's 5-7% weight loss outcome was specific to individuals who were over 200lbs at the start of the trial [29]. The delay in onset of diabetes with a 5-7% weight loss in the Diabetes Prevention Program was 4 years, with 1/3 of participants less likely to develop diabetes a decade later. Accordingly, efforts to prevent diabetes through modest weight loss in those with depression will have meaningful health effects. One five-year follow-up study with 432 participants in the FINRISK study (2002) on the perceived risks of diabetes and cardiovascular disease did not predict body mass index, physical activity, and blood glucose levels unless self-efficacy and outcome beliefs were entered into the structural equation model [46].

In another study by Imai, Furukawa, & Hayashai, (2017), risk perception, self-efficacy, and trust with food habits and physical activity of individuals with diabetes showed that risk perception could worsen depression and food habits. Whereas self-efficacy and trust were more predictive of improved food habits, exercise and helped depression levels [47].

Many antidepressants demonstrate glucose lowering capabilities, but it is often discussed in the context of medication adjustments to prevent hypoglycemia when glucose lowering agents are concurrently used. For example, buspirone [48], fluoxetine, citalopram, mirtazapine [49], escitalopram [50], and sertraline [51] may reduce glucose levels while paroxetine and vilazodone increase glucose levels [52]. Tricyclic antidepressants, less often used for depression than they are for cyclic vomiting syndrome and fibromyalgia and sleep issues, are implicated in weight gain and hyperglycemia [53]. Often the antidepressants may increase weight, and this may offset indirect glucose lowering effects, so close monitoring is necessary to understand the individuals need to for glucose control.

Although sleep disorders do not explain the co-occurring relationship between depression and diabetes, it is a common comorbidity of both conditions [54]. Other lifestyle considerations to improve depression and diabetes control include treating insomnia, since 77% of people with diabetes have insomnia and when treating it with an antidepressant, trazadone, glucose control improves [55].

Risk-reducing lifestyle behaviors to prevent or delay the onset of diabetes in high-risk populations is efficacious and effective across settings and delivery methods, including group formats, technology-based, and by various forms of implementation including, through community members [56]. What is less known is the degree to which individuals who are affected by depression recognize they are at risk for diabetes and the factors that may impede their ability to act. Multiple lifestyle factors have been associated with depression despite, the fact that depression is an independent risk factor for diabetes and that lifestyle is a contributing risk factor to diabetes [57]. Lifestyle factors that influence depression include, diet, physical activity, sleep, alcohol consumption, and smoking [57]. In addition, depression is known to adversely affect cognitions, thinking and motivation (the necessary prerequisites to behavior change [58].

Conclusion

Depression is a risk factor for diabetes. When identifying people with depression nurses can address the concurrent risk of diabetes and increase screening potential. Nurses are trusted providers to whom patients are likely to disclose stigmatizing conditions. Because nurses work across the continuum of mental health and physical medical conditions, they are ideal candidates to take the lead with addressing the comorbid risk of depression and prediabetes. Given that so many factors are interrelated when it comes to lifestyle change, depression, obesity, and diabetes, it would be important for nurses to intervene to simultaneously address the risk of diabetes in those who are depressed.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Conflict of Interest

The authors declare that they have no competing interests.

References

1. Karamanou M, protogerou A, Tsoucala G, Androustos G, Poulakou-Rebelakou E (2016) Milestones in the history of diabetes mellitus: The main contributors. *World Journal of Diabetes*. 7: 1-7
2. Kahn R, Weir GC (1994) *Joslin's Diabetes Mellitus*. 13th ed.
3. Maina JG, Balkhiyarova Z, Nouwen A, Pupko I, Ulrich A, Boissel M, et al. (2023) Bidirectional mendelian randomization and multiphenotype GWAS show causality and shared pathophysiology between depression and type 2 diabetes. *Diabetes Care* 46: 1707-1714.
4. Wicke FS, Otten D, Schulz A, Wild PS, Lackner KJ, Munzel T et al. (2024) Current and past depression as risk factors for incident type 2 diabetes mellitus and prediabetes in men and women: Evidence from a longitudinal community cohort. *Diabetology & Metabolic Syndrome*. 16: 34
5. U.S. Department of Health & Human Services, Center for Disease Control, (2021).
6. Van Sloten TT, Sedaghat S, Carnethon MR, Launer LJ, Stehouwer CDA (2020) Cerebral microvascular complications of type 2 diabetes: stroke, cognitive dysfunction, and depression. *The Lancet, Diabetes & Endocrinology*. 8: P 325-336.
7. Liu D, McIntyre RS, Li R, Yang M, Xue Y, Cao B, et al. (2021) Genetic association between major depressive disorder and type 2 diabetes mellitus: Shared pathways and protein networks. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*. 111.
8. Barker M, Davies MJ, Zaccardi F, Brady EM, Hall AP, et al. (2023) Age at diagnosis of type 2 diabetes and depressive symptoms, diabetes-specific distress, and self-compassion. *Diabetes Care*. 46: 579-586
9. Deleskog A, Ijung R, Forsell Y, Nevriana A, Almas A, et al. (2019) Severity of depression, anxious distress and the risk of type 2 diabetes -a population-based cohort study in Sweden. *BMC Public Health*. 19:1174.
10. Graham E, DeSchenes SS, Rosella LC, Schmitz N (2021) Measure of depression and incident type 2 diabetes in a community sample. *Annals of Epidemiology*. 55: 4-9.
11. American Psychiatric Association (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th ed).
12. Levis B, Sun Y, He C, Wu Y, Kirishnan K, et al. (2020) Accuracy of the PHQ-2 alone and combined with the HQ-9 for screening to detect major depression. *JAMA Network*. 323: 2290-2300.
13. World Health Organization (2021). *Diabetes*.
14. Wagner R, Thorand B, Osterhoff MA, Müller G, Böhm A, Mesisinger C, et al. (2013) Family history of diabetes is associated with higher risk for prediabetes: a multicenter analysis from the German Center for Diabetes Research. *Diabetologia*. 56: 2176-2180.
15. Tsenkova V, Karlamangla A, Ryff C (2016) Parental history of diabetes positive affect, and diabetes risk in adults: Findings from MIDUS. *Annals of Behavioral Medicine*. 50: 836-843.
16. Ali MK, Imperatore G, Benoit SR, O'Brien MJ, Holiday CS, et al. (2023) Impact of changes in diabetes screening guidelines on testing eligibility and potential yield among adults without diagnosed diabetes in the United States. *Diabetes Research & Clinical Practice*. 197: 110572.
17. Noetel M, Sanders T, Gallardo-Gomez D, Taylor P, et al. (2024) Effect of exercise for depression: systematic review and network meta-analysis of randomized controlled trials. *BMJ*. 384: e075847.
18. Zahalka SJ, Abushamat LA, Scalzo RL, Reusch JEB, Feingold KR, Blackman AB, et al. (2023) The role of exercise in diabetes. *Endotext*. MDText.com.
19. Bawa H, Poole L, Cooke D, Panagi L, Steptoe A, Hackett RA (2020) Diabetes-related distress and daily cortisol out in people with type 2 diabetes. *Diabetes Research and Clinical Practice*. 169: 108472.
20. Khawagi WY, Al-Kuraishy HM, Hussein NR, Al-Gareeb AI, Atef E, Elhussieny O, et al. (2024) Depression and type 2 diabetes: A causal relationship and mechanistic pathway. *Diabetes, obesity, and Metabolism*. 26: 3031-3044.

21. Croarkin PE, Levinson AJ, Daskalakis ZJ (2011) Evidence for GABAergic inhibitory deficits in major depressive disorder. *Neuroscience Biobehavioral Reviews*. 35: 818-825.
22. Cowen PJ, Browning M (2015) What has serotonin to do with depression? *World Psychiatry*. 14: 158-160.
23. Stahl SM (2013) *Stahl's essential psychopharmacology: Neuroscientific basis and practical applications* (4th ed.). Cambridge University Press.
24. Lyra e Silva N, Lam MP, Soares CN, Munoz DP, Milev R, De Felice FG, Lustman PJ, Clouse RE, et al. (2005) Depression in diabetic patients: The relationship between mood and glycemic control. *Journal of Diabetes and its Complications*. 19: 113-122.
25. Güemes A, Gregoriou P (2018) Review of the role of the nervous system in glucose homeostasis and future perspectives towards the management of diabetes. *Bioelectronic Medicine*. 4: 2-18.
26. Owens-Gary MD, Zhang X, Jawanda S, McKeever Bullard K, Allweiss P, Smith BD, et al. (2018) The importance of addressing depression and diabetes distress in adults with type 2 diabetes. *Journal of General Internal Medicine*. 34: 320-324.
27. Yabut, JM, Crane JD, Green AE, Keating DJ, Khan WI, Steinberg GR, et al. (2019) Emerging roles for serotonin in regulating metabolism: new implications for an ancient molecule. *Endocrine Reviews*. 40: 1092-1107.
28. Schnurr TM, Jakupovic H, Carrasquilla GD, Angquist I, Graup N, Sorensen TIA, et al. (2020) Obesity, unfavorable lifestyle and genetic risk of type 2 diabetes: a case-cohort study. *Diabetologia* 63: 1324-1332.
29. Diabetes Prevention Program Research Group (2009) The 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet*. 374: 1677-1686.
30. Kelly SJ, Ismail M (2015) Stress and type 2 diabetes; a review of how stress contributes to the development of type 2 diabetes. *Annual Review of Public Health*. 18: 441-462.
31. Molife C (2018) Is depression a modifiable risk factor for diabetes burden? *Journal of Primary Care & Community Health*.
32. Yates BE, De Letter MC, Parrish EM (2020) Prescribed exercise for the treatment of depression in a college population: An interprofessional approach. *Perspectives in Psychiatric Care*. 56: 894-899.
33. Diabetes Prevention Research Group (2005) Strategies to identify adults at risk for type 2 diabetes: The Diabetes Prevention Program. *Diabetes Care*, 28: 138-144.
34. Sampson M, Clark A, Bachmann M, Garner N, Irvine L, Howe A, et al. (2020) Norfolk Diabetes Prevention Study (NDPS) Group. Lifestyle intervention with or without lay volunteers to prevent type 2 diabetes in people with impaired fasting glucose and/or nondiabetic hyperglycemia. *JAMA Internal Medicine*. 181: 140-141.
35. Miidera H, Enomoto M, Kitamura S, Tachimori H, Mishima K (2020) Association between the use of antidepressants and the risk of type 2 diabetes: A large, population-based Cohort Study in Japan. *Diabetes Care*. 43: 885-893.
36. Piper C, Marossey A, Griffiths Z, Adegboye A (2017) Evaluation of a type 2 diabetes prevention-program using a commercial weight management-provider for non-diabetic hyperglycemic patients referred by primary care in the UK. *BMJ Open Diabetes Research & Care*. 5: e000418
37. Karasu SR (2013) Psychotherapy-lite: Obesity and the role of the mental health practitioner. *The American Journal of Psychotherapy*. 67: 3-22.
38. Schneider K, Sullivan JC, Pagago SL (2011) Translation of the diabetes prevention program into a community mental health organization for individuals with severe mental illness: a case study. *Translational Behavioral Medicine*. 1: 453-460.
39. Brown C, Geiszler LC, Lewis KJ, Arbesman M (2018) Effectiveness of interventions for weight loss for people with serious mental illness: A systematic review and meta-analysis. *American Journal of Occupational Therapy*. 72: 7205190030p1-7205190030p9.
40. Lorenz GC, Levey MC, Case R (2013) Integrated care through education: improving care for those with serious mental illness and/or intellectual disability and diabetes in rural India. *Journal of Rural Mental Health*. 37: 16-24.
41. Rubin RR, Ma Y, Morrero DG, Peyrot M, Barret-Connor EL, Kahn SE, et al. (2008) Diabetes Prevention Program Research Group. Elevated depression symptoms, antidepressant medicine use, and risk of developing diabetes during the diabetes prevention program. *Diabetes Care*. 31: 420-426.
42. Khanbatay T, Callahan CM, Stewart JC (2018) Effect of collaborative depression treatment on risk for diabetes; A 9-year follow-up of the IMPACT randomized controlled trial. *PLOS ONE*.
43. Brühlw, Schwarzer C, Berns C, Scho S, Schneefeld J, Koester D, et al. (2021) Physical activity reduces clinical symptoms and restores neuroplasticity in major depression. *Frontiers in Psychiatry*.
44. Ma J, Rosas LG, Lv N, Xiao L, Snowden M, Vendetti EM, et al. (2019) The effect of integrated behavioral weight loss and problem-solving therapy on body mass index and depressive symptoms among patients with obesity and depression. The RAINBOW Randomized Clinical Trial. *Journal of the American Medical Association*. 321: 869-879.
45. Speyer H, Jakobsen AS, Westergaard C, Nørgaard HC, Pisinger C, Krogh J, et al. (2019) Lifestyle interventions for weight management in people with serious mental illness: a systematic review with metaanalysis, trial sequential analysis and meta-regression analysis, exploring the mediators and moderators of treatment effects. *Psychotherapy & Psychosomatics*. 88: 350-362.
46. Vornanen M, Kontinen H, Peltonen M, Haukkala A (2021) Diabetes and cardiovascular disease risk perception and risk indicators: A 5-year follow-up. *International Journal of Behavioral Medicine*. 28: 337-348.
47. Imai H, Furukawa TA, Haycshai SA, Goto A, Izumi K, Hayashino Y, et al. (2017) J-DOIT2 Study Group Risk perception, self-efficacy, trust for physician, depression, and behavior modification for diabetic patients. *Journal of Health Psychology*. 25: 350-360.
48. Ojha SK, Nandave M, Sharma C (2007) Effects of buspirone on blood sugar levels in humans. *Indian Journal of Pharmaceutical Sciences*. 69: 414-417.
49. Bektur E, Sahin E, Baycu C (2019) Mirtazapine may show anti-hyperglycemic effect by decreasing GLUT2 through leptin and galanin

- expressions in the liver of type 1 diabetic rats. *Iranian Journal of Basic Medical Science*. 22: 676-682.
50. Park YS, Sung KW (2019) Selective serotonin reuptake inhibitor escitalopram inhibits 5-HT₃ receptor currents in NCB-20 cells. *The Korean Journal of Physiology & Pharmacology*. 23: 509–517.
51. Kesim M, Tiryaki A, Muci E, Kadioglu NI, Yarsis E (2011) The effects of sertraline on lipids, glucose, insulin and HBA1C levels; A prospective clinical trial on depressive patients. *Journal of Respiratory Medical Science*. 16: 1525-1531.
52. Das A, Jain PD, Ravan JR, Madaan A (2020) Vilazodone-induced hyperglycemia: A case series. *Indian Journal of Psychological Medicine*. 44: 206-207.
53. Alruwaili NS, Al-Kuraishy HM, Al-Gareeb AI, Albuhadily AK, Ragab AE, et al. (2023) Antidepressants and type 2 diabetes: highways to knowns and unknowns. *Diabetology and Metabolic Syndrome*. 15: 179.
54. Wium -Anderson IK, Osler M, Jorgensen MB, Rungby J, Wium-Anderson MK (2022) Diabetes, antidiabetic medications and risk of depression-A population—based cohort and nested case-control. *Psychoneuroendocrinology*. 140: 105715.
55. Sakkal S (2018) Improving sleep by trazadone improves glucose control. *Diabetes*.
56. Galaviz KI, Venkat Narayan KM, Lobelo F, Weber MB (2015) Lifestyle and the Prevention of type 2 Diabetes: A status report. *American Journal of Lifestyle Medicine*. 12: 4-20.
57. Sarris J, Thomson R, Hargraves F, Eaton M, de Manincor M, Veronese N, et al. (2020) Multiple lifestyle factors and depressed mood: a cross-sectional and longitudinal analysis of the UK Biobank (N=84,860). *BMC Medicine*. 18: 354.
58. Grahek I, Shenhav A, Musslick S, Krebs RM, Koster EHW (2019) Motivation and cognitive control in depression. *Neuroscience Biobehavioral Reviews*. 27: 371-381.