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## AIMS AND SCOPE

The primary aim of *World Journal of Diabetes* (*WJD*, *World J Diabetes*) is to provide scholars and readers from various fields of diabetes with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

*WJD* mainly publishes articles reporting research results and findings obtained in the field of diabetes and covering a wide range of topics including risk factors for diabetes, diabetes complications, experimental diabetes mellitus, type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes, diabetic angiopathies, diabetic cardiomyopathies, diabetic coma, diabetic ketoacidosis, diabetic nephropathies, diabetic neuropathies, Donohue syndrome, fetal macrosomia, and prediabetic state.

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LETTER TO THE EDITOR

# Reconsidering the role of depression and common psychiatric disorders as partners in the type 2 diabetes epidemic

Angelo Emilio Claro, Clelia Palanza, Marianna Mazza, Alessandro Rizzi, Andrea Corsello, Linda Tartaglione, Giuseppe Marano, Giovanna Elsa Ute Muti Schuenemann, Marta Rigoni, Alfredo Pontecorvi, Luigi Janiri, Paola Muti, Dario Pitocco

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## Abstract

Common psychiatric disorders (CPDs) and depression contribute significantly to the global epidemic of type 2 diabetes (T2D). We postulated a possible pathophysiological mechanism that through Bridge-Symptoms present in depression and CPDs, promotes the establishment of emotional eating, activation of the reward system, onset of overweight and obesity and, ultimately the increased risk of developing T2D. The plausibility of the proposed pathophysiological mechanism is supported by the mechanism of action of drugs such as naltrexone-bupropion currently approved for the treatment of both obesity/overweight with T2D and as separate active pharmaceutical ingredients in drug addiction, but also

from initial evidence that is emerging regarding glucagon-like peptide 1 receptor agonists that appear to be effective in the treatment of drug addiction. We hope that our hypothesis may be useful in interpreting the higher prevalence of CPDs and depression in patients with T2D compared with the general population and may help refine the integrated psychiatric-diabetic therapy approach to improve the treatment and or remission of T2D.

**Key Words:** Depression; Glucagon-like peptide-1 receptor agonists; Diabetes mellitus type 2; Stress psychological; Sleep wake disorders; Food addiction



**Core Tip:** Common psychiatric disorders (CPDs) and depression are important contributors to the epidemic of type 2 diabetes (T2D). Bridge-Symptoms of depression and CPDs, promote the onset of emotional eating, activation of the reward system and the development of overweight/obesity and T2D. The efficacy of naltrexone-bupropion in the treatment of overweight/obesity and drug addiction and the emerging evidence of the possible effectiveness of glucagon-like peptide 1 receptor agonists in drug addiction support our hypothesis. We propose insights to interpret the higher prevalence of CPDs and depression in patients with T2D compared to the general population and to improve the integrated psychiatric-diabetic therapy.

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## TO THE EDITOR

We read with great interest the work of Wang *et al*[1] and thank our colleagues for their significant and valuable work. It was our pleasure and honor to read their paper. However, we would like to propose a possible insight into the pathogenesis, maintenance and treatment of type 2 diabetes (T2D) that our colleagues have not tackled and that we believe is critically important to address. Namely how psychological aspects affect T2D.

In fact, 90% of all diabetes cases worldwide are due to T2D and it is known that the main risk factors for the development and maintenance of T2D are overweight and obesity[2], regardless of genetic predisposition[3]. Within this framework, the epidemic of overweight and obesity that we are witnessing is playing a crucial role in determining the epidemic of T2D[4,5].

Further confirming the extent to which overweight and obesity are linked to the development of T2D, many clinical trials of bariatric surgery and medical therapy have shown that in the first 6 years after diagnosis, patients with T2D, through a ≥ 10%-15% reduction in body weight, can achieve remission of T2D (remission defined as a hemoglobin level < 6.5% or 48 mmol/mol, after cessation of insulin or pharmacotherapy)[6]. Consequently, T2D should not always be considered a chronic, incurable disease[6]. Nevertheless, despite the possibility of T2D remission through sufficient weight loss, the incidence of T2D remission in routine care settings is still low (< 1%)[7]. This low remission rate may occur because the concept of remission through sufficient weight loss in the early years of the disease is still poorly known among clinicians and patients. Moreover, even when patients are aware of this possibility, not all patients possess the appropriate characteristics to achieve remission. However, weight loss can lead to improved glycemic control, reduced development and worsening of complications and reduced need for antidiabetic or insulin therapy even in patients who cannot achieve remission[8-10]. Therefore, for all patients with T2D, a normal weight without central overweight or obesity is paramount and recommended.

In this regard, an important aspect underlying the difficulty of achieving adequate weight loss to gain remission and/or improvement of T2D, which Wang *et al*[1] did not mention in their work, is the high prevalence of depression and common psychiatric disorders (CPDs) in patients with T2D[11-14].

This aspect is crucial when it comes to the treatment of T2D, as it is emerging from the literature that depression and CPDs can drive the development of overweight, obesity and consequently T2D[11,12].

In fact, there is a strong connection between T2D, depression and CPDs. Patients with T2D have higher rates of depression when compared to the general population: 25% of patients with T2D have depression[12,13], while in the general population the prevalence of depression is 5%[15]. The prevalence of CPDs in patients with T2D is around 40%[12,16] whereas the prevalence of CPDs in the general population is 17.6%[17].

Depression promotes weight gain and carries a 60% increased risk of developing T2D[12,18] through three of its symptoms that we have renamed Bridge Symptoms (BS): altered eating behavior[19], inadequate sleep duration[20,21] and fatigue or lack of energy (asthenia) leading to reduced physical activity[19,22]. BS are also present in CPDs and increase the risk of developing T2D[11,12]. However, below, we will refer to the link between depression and BS as the knowledge on this connection in the field of depression is more extensive in the literature.

## ALTERED EATING BEHAVIOR

According to one of the most acknowledged theories on depression, patients who suffer from depressive disorders are those who are unable to cope with high levels of stress[23,24]. In particular, stress causes the initiation of mental and subsequently biological processes that can lead to the development of depressive disorders[23,25,26].

Long-lasting external stressful events or stressful internal thoughts or beliefs can cause damage to self-esteem and pathological lowering of mood[23,24].

Although stressful events are present in everyone's life, only some people develop depression. The reason behind this is that stress per se is not capable of inducing depression, but it is how the individual reacts to these stressful events that predicts the eventual development of the illness[23,24]. If a person is able to recognize stress and deal with it by reducing or eliminating it, or have a positive response to it, he or she will not develop depression[24].

Psychological stress activates the same brain areas that are activated during experiences of physical pain, namely the anterior part of cerebral insula (AIC) and the antero-dorsal portion of the cingulate cortex (ACC)[27-29]. These areas in turn have connections with the hypothalamic-pituitary-adrenal axis and with the autonomic control nuclei of the brainstem triggering the activation of systemic inflammation, the immune system and the so-called fight-or-flight response[23]. In the past, during the time of our hunter-gatherer predecessors, because stressors primarily resulted from physical clashes and were acute in nature, the fight-or-flight response (which prepares the body for combat, activates the immune system and inflammation) allowed our ancestors to be prepared in case physical harm occurred, and was thus regarded as positive[23,30]. Therefore, this type of response predisposed to fight and activated the immune system and inflammation that were ready in case of physical harm[23,30]. In today's lifestyle, where stress is mainly a consequence of psychological problems, this reaction has negative consequences. In fact, unrecognized or continuous and uneliminated psychological stress can lead to a feeling of psychological discomfort due to activation of the pain-related brain areas and determine the activation of chronic subclinical inflammation[31-33]. In this way a suboptimal compensation response can be triggered: Emotional eating (EE).

EE is defined as the propensity to overeat in response to stress and negative feelings[34]. The intake of certain foods in fact can act as self-medication to reduce depressed mood and brain-wide activation of pain-related areas.

Foods rich in simple sugars (comfort foods) can improve mood because they increase the production of serotonin, which has a positive effect on mood[21], proportionally to their glycemic index[35].

On the other hand, foods rich in fats defined as "palatable foods" stimulate the release of hypothalamic endorphin (an endogenous agonist of mu-opioid receptors)[36,37] which switches off activation of the AIC and ACC, causing a reduction of ailment resulting from stress and depression.

Thus, these types of foods are preferentially consumed during EE due to their stress-relieving and antidepressant effect.

EE can also occur in healthy people who do not suffer from depression, for example in those who experience sadness following an acute negative stressful event[38]. However, sadness is a physiological and temporary state in response to a negative event, as is the associated EE. In contrast, depression is a chronic condition that does not respond to life events. As a result, EE in depression can become chronic and lead to constant weight gain, increasing the risk of developing T2D [34].

Repeated EE can also lead to activation of the reward system, affecting the strengthening of reward neural pathways and the development of the tendency to enact the behavior[39]. In some cases, this can lead to the onset of food addiction [34,39,40].

Both past and emerging pharmacological evidence supports these arguments.

The first example of a drug that acts on the mechanisms described is naltrexone-bupropione (NB), composed of naltrexone, an antagonist of the μ-opioid receptor, and bupropion, an antidepressant inhibitor of neuronal dopamine and norepinephrine reuptake.

NB was marketed in 2014 in the European Union (EU) as a drug for weight management in adults with a BMI of 30 $kg/m^2$ or between 27 $kg/m^2$ and < 30 $kg/m^2$ with one or more weight-related comorbidities, such as T2D, dyslipidemia or arterial hypertension[41,42].

Naltrexone alone is used to treat alcohol use disorder and opioid dependence[43], while bupoprion is an antidepressant medication approved for the treatment of major depression and for smoking cessation[44]. Moreover, in a recent clinical trial, NB was found to be effective in the treatment of methamphetamine use disorder[45]. Therefore, NB, acting on the same brain pathways on which comfort and palatable food act during EE, can induce weight loss. This evidence supports our claims on the link between depression, EE, and the development of T2D.

Initial evidence is also beginning to emerge in the literature on how glucagon-like peptide 1 (GLP-1) receptor agonists, act to dampen "food noise", the incessant and distressing desire to keep eating, further confirming the strong correlation between stress, depression, BS, EE, food addiction, reward, and the development of T2D[46]. In fact, GLP-1 has already been shown to reduce food cravings and drug addiction in animal models[47] and in small-sample studies in humans[48-50]. For this reason, at least nine phase 2 clinical trials are underway or planned to test whether GLP-1 can help patients quit alcohol, cigarettes, opioids, or cocaine use[46]. These clinical trials are underway after people with obesity and T2D reported decreased cravings for wine and cigarettes while on the treatment[50].

## INADEQUATE SLEEP DURATION

Stress also induces adrenergic hyperactivation leading to reduced sleep duration. It is no coincidence that about 40% of patients with T2D and approximately 50% of patients with depression suffer from insomnia[51], while the prevalence of

insomnia in the general population is 10%[52]. These data on insomnia and the higher rate of depression in patients with T2D compared to the general population are a further indication of how T2D and depression go hand in hand[53].

Insomnia influences appetite hormones by resulting in an increase in the ghrelin/leptin ratio, increasing the production of endocannabinoids and lowering GLP-1 levels[54,55]. This leads to increased hunger and appetite, resulting in positive energy balance and weight gain[54,56]. Indeed, short sleep duration has been shown to be a risk factor for the development of obesity and weight gain and may impact weight loss intervention[57]. The inflammatory state that occurs in patients with T2D and depression also inhibits the action of leptin at the hypothalamic level, reducing the sense of satiety[58].

Furthermore, insomnia causes changes in melatonin, cortisol, and catecholamine levels that affect glucose tolerance and reduces insulin sensitivity[55].

Poor sleepers also have less energy to carry out physical activity[55], show a greater intake of calories from comfort and palatable foods and reduced intake of sleep inducer foods[21]. Retrospective studies show that people who report better sleep quality are (33%) more likely to achieve weight loss success[59,60].

## REDUCED PHYSICAL ACTIVITY

Patients suffering from depression are also sedentary and perform very little physical activity[22]. Physical activity, in addition to improving glycemic control, cardiovascular complications, lipid levels and blood pressure, has been associated with a greater chance of losing weight in association with diet[61-63].

People with T2D are also responsible for much of the management of their disease, but if they have psychological disorders, these lead to poor self-management and early development of adverse outcomes including early mortality[64, 65].

BS may therefore act by driving the development of T2D in lean and otherwise healthy individuals, making weight loss impossible in patients with T2D, or inducing weight regain in patients who have managed to achieve remission of T2D through weight loss.

Furthermore, patients with T2D who do not have a true psychiatric disorder, often present with subthreshold psychiatric symptoms[66] which are also more frequent in patients with T2D than in the general population[66]. Comorbidity of subthreshold psychiatric disorder is also starting to be linked with poorer self-management, quality of life and metabolic control in T2D[66].

## CONCLUSION

We would like to thank our colleagues once more for their article, but we believe it is important to mention the psychological aspects related to T2D. We deem it necessary to perform psychiatric screening in patients suffering from T2D in order to try to intervene more incisively on the development of T2D and its remission, considering how CPDs and depression favor its onset and maintenance[7,11,12]. It is also necessary to evaluate stress and sleep disturbances in all patients with T2D, even in those without depression and CPDs. We assume that an integrated diabetic-psychiatric therapy is crucial in order to attempt to obtain an increase in the remission rate or an improvement in T2D in patients with comorbid CPDs and depression, with subthreshold mental disorders or simply with high levels of stress and/or insomnia[12]. This could help reduce the T2D epidemic, driving a reduction in years of life lost given that diabetes is the 9$^{th}$ leading cause of death globally, and an important contributor to the first and second leading cause of death globally (ischemic heart disease and stroke). Moreover, this can lead to substantial economic savings given that T2D absorbs 12% of global health expenditure[67].

## FOOTNOTES

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