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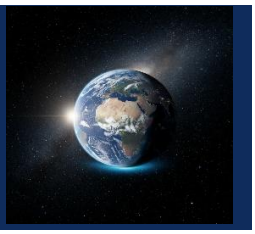

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LITERATURE REVIEW

GLP-1 receptor agonists in the regulation of the brain–metabolism axis: implications for mental health

a narrative review

HIGHLIGHTS

- GLP-1 receptor agonists, originally developed for type 2 diabetes and obesity, also modulate central pathways relevant to mental health.
- Anti-inflammatory effects, neuroprotection and modulation of the HPA axis underpin potential antidepressant and anti-anxiety actions of GLP-1RAs.
- Preclinical and early clinical data suggest a reduction in alcohol and nicotine use during GLP-1RA therapy, with effects independent of weight loss.

- Cognition, satiety regulation and reward processing may be improved through GLP-1RA action in mesolimbic and hypothalamic structures.
- Robust randomised controlled trials are still needed to confirm long-term psychiatric safety and identify patient subgroups most likely to benefit.

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ABSTRACT

BACKGROUND: Glucagon-like peptide-1 receptor agonists (GLP-1RAs) were originally developed for the treatment of type 2 diabetes and obesity. Growing preclinical and clinical evidence indicates that GLP-1RAs also influence the central nervous system, with potential implications for depression, addictive behaviours, anxiety and cognition.

AIM: To summarise current evidence on the effects of GLP-1 receptor agonists on selected mental health outcomes — depression, anxiety, substance use disorders and cognition — and to discuss mechanisms linking metabolic and psychiatric pathways.

MATERIALS AND METHODS: A narrative review was performed using PubMed/MEDLINE for the period 2020–2025. Search terms combined "GLP-1", "GLP-1 receptor agonist", "GLP-1RA", "mental health", "depression", "anxiety", "addiction", "substance use", "cognition" and "systematic review" or "meta-analysis". Original studies, conference abstracts and non-full-text publications were excluded.

RESULTS: GLP-1RAs modulate the hypothalamic–pituitary–adrenal (HPA) axis, dopaminergic reward circuits and neuroplasticity. Reviews and meta-analyses suggest possible antidepressant and anxiolytic effects, especially in patients with comorbid obesity or diabetes. Cohort studies indicate reduced incidence of alcohol use disorder and decreased alcohol-related events during GLP-1RA therapy. Effects on nicotine and other substances are biologically plausible but supported by fewer data. Psychiatric safety appears favourable, with no consistent signal for increased suicidality.

CONCLUSIONS: GLP-1 receptor agonists emerge as candidate modulators of the brain–metabolism axis with potential benefits for mental health, addictive behaviours and cognition. Robust randomised controlled trials are required to confirm clinical efficacy, define patient subgroups and clarify long-term psychiatric safety.

KEYWORDS GLP-1 receptor agonists; semaglutide; liraglutide; depression; anxiety; substance use disorder; cognition; mental health.

PLAIN LANGUAGE SUMMARY

GLP-1 receptor agonists are medications originally developed for type 2 diabetes and obesity. They mimic a natural gut hormone that helps regulate blood sugar and appetite. Recent research suggests that these drugs also act on the brain — calming stress responses, influencing the reward system, and supporting neuronal health. Early studies indicate that they may help reduce symptoms of depression and anxiety, especially in people who are also overweight or have diabetes. Other studies suggest that they may decrease the urge to drink alcohol or, in some cases, to smoke. So far, the safety profile in terms of mental health is reassuring, and the risk of suicidal thoughts does not appear to be increased. However, large, well-designed clinical trials are still needed before GLP-1RAs can be routinely recommended in mental health care.

TABLE OF CONTENTS

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1. INTRODUCTION

Mental health disorders are among the leading causes of disability worldwide. At the same time, the global burden of obesity and type 2 diabetes continues to rise. These conditions are increasingly recognised as metabolically, immunologically and behaviourally intertwined with depression, anxiety and addictive disorders, forming what some authors describe as the brain–metabolism axis.

Glucagon-like peptide-1 receptor agonists (GLP-1RAs) such as liraglutide, semaglutide and tirzepatide were developed to improve glycaemic control and reduce body weight. Beyond their metabolic effects, growing preclinical and clinical evidence suggests that these drugs act on central nervous system structures involved in reward, stress and cognition.

This narrative review summarises current evidence on the effects of GLP-1RAs on selected mental health outcomes — depression, anxiety, cognition and substance use — and discusses biological mechanisms that may explain these effects. Particular attention is paid to clinical data on alcohol use disorder, where both cohort and randomised studies are available.

2. METHODOLOGY

This study was conducted as a narrative review based on a selection of systematic reviews, meta-analyses, scoping reviews and comprehensive reviews addressing the impact of glucagon-like peptide-1 receptor agonists (GLP-1RAs) on mental health.

The analysis aimed to identify and summarise the most recent evidence regarding the effects of GLP-1RAs on mental health outcomes. A literature search was performed using the PubMed/MEDLINE database, one of the primary sources of biomedical literature.

Relevant articles were identified through a structured search of publications available in PubMed, with particular emphasis on recent reports and high-quality review articles.

A combination of keywords and Boolean operators was used, including “GLP-1” OR “glucagon-like peptide-1”, “GLP-1 receptor agonist” OR “GLP-1RA”, “mental health” OR “psychiatric” OR “depression” OR

"anxiety", "addiction" OR "substance use", "cognition" OR "cognitive function", "safety" OR "psychiatric adverse events", "systematic review" OR "meta-analysis" OR "scoping review" OR "comprehensive review".

Example query: ("GLP-1" OR "GLP-1 receptor agonist") AND ("mental health" OR depression OR anxiety OR addiction OR cognition) AND ("systematic review" OR "meta-analysis" OR "scoping review" OR "comprehensive review").

The search covered publications released within the last 5 years. In this study, we deliberately restricted ourselves to PubMed to ensure consistency in the selection process.

Inclusion and exclusion criteria

Inclusion criteria

Publications meeting the following conditions were included in the analysis: (1) review articles (systematic review, meta-analysis, scoping review, comprehensive review); (2) publications indexed in PubMed; (3) studies concerning GLP-1 or GLP-1RA; (4) analysis of the impact on mental health (including: depression and anxiety, other mental disorders, addictions, cognitive functions, psychiatric safety); (5) articles in English.

Exclusion criteria

The following publications were excluded: (1) original studies (RCTs, cohort studies, case reports); (2) articles without a mental health component; (3) exclusively preclinical studies (if there is no reference to clinical implications); (4) non-full-text publications (e.g. conference abstracts).

3. THE BIOLOGY OF GLP-1 AND NEUROPSYCHIATRIC MECHANISMS

Glucagon-like peptide-1 (GLP-1) receptor agonists mimic the action of the naturally secreted incretin hormone GLP-1. While naturally secreted incretin hormones act for a short period, the effects of GLP-1 agonists last much longer. To date, these drugs have been used to treat type 2 diabetes and obesity, but recent reports from clinical observations and preliminary analyses suggest that GLP-1 may also influence mental health.

GLP-1 agonists work by stimulating glucose-dependent insulin secretion, inhibiting glucagon secretion, delaying gastric emptying, as well as by influencing the satiety center.

The effect on the nervous system, and consequently on mental health, may be related to the distribution of GLP-1 receptors. Numerous GLP-1 receptors are widely distributed throughout the central nervous system, including the hypothalamus, hippocampus, amygdala, prefrontal cortex and brainstem [1,2]. Through these structures, GLP-1RAs may modulate the HPA axis, dopaminergic reward circuits, neuroinflammation and neuroplasticity [3,4].

Anti-inflammatory and neuroprotective effects have been described in preclinical models, including reduced microglial activation, lower levels of pro-inflammatory cytokines and improved insulin signalling in the brain [4,5]. These mechanisms are increasingly considered relevant to the pathophysiology of depression and neurodegenerative disorders, and they provide a biological rationale for the potential psychiatric applications of GLP-1RAs.

4. GLP-1 RECEPTOR AGONISTS IN DEPRESSION AND ANXIETY

Depression and anxiety frequently coexist with obesity and type 2 diabetes. Shared mechanisms include chronic low-grade inflammation, HPA-axis dysregulation, impaired insulin signalling in the brain and alterations of the gut–brain axis. GLP-1 receptor agonists, which act simultaneously on metabolic and central pathways, are therefore intuitive candidates for psychiatric repurposing.

4.1. Antidepressant effect of GLP-1RAs

Systematic reviews and meta-analyses suggest that GLP-1RAs may reduce depressive symptoms, particularly in patients with comorbid obesity or type 2 diabetes [5,6,14]. Improvements have been reported on validated scales such as PHQ-9 and HADS, although effect sizes vary across studies. Observational data indicate a lower incidence of new-onset depression in patients treated with semaglutide or liraglutide compared with other antidiabetic regimens [6,14].

4.2. Mechanisms — HPA axis and neuroinflammation

Chronic stress and dysregulation of the HPA axis are well-established contributors to depression. Preclinical data demonstrate that GLP-1RAs attenuate stress-induced cortisol responses, normalise glucocorticoid receptor signalling and reduce hippocampal neuroinflammation [3,4,11]. These mechanisms may underlie the antidepressant signal observed in clinical studies and provide a link between metabolic and affective regulation.

4.3. Effects on anxiety

Evidence on anxiety is more limited but biologically plausible. Several reviews and meta-analyses of psychiatric symptoms suggest improvements in anxiety scores during GLP-1RA therapy, especially in patients with metabolic comorbidities [11,12]. Reductions in anxiety may reflect improvements in body image, physical function and inflammatory profile.

4.4. Effects on satiety, weight and quality of life

Weight loss induced by GLP-1RAs is consistently associated with improved quality of life, self-esteem and physical performance [16]. These factors contribute to mental well-being independently of direct central effects. Improvements in sleep and reduced binge-eating behaviour have also been reported.

4.5. Limitations and patient subgroups

Most of the available evidence is derived from secondary analyses of trials primarily designed for metabolic outcomes, and from observational studies subject to confounding. Patient subgroups most likely to benefit — for example, those with comorbid depression and obesity — remain to be precisely defined in dedicated psychiatric trials [13,14].

5. GLP-1 RECEPTOR AGONISTS IN SUBSTANCE USE DISORDERS

The dopaminergic reward system plays a central role in the development and maintenance of substance use disorders. GLP-1 receptors are present in mesolimbic structures, including the ventral tegmental area and nucleus accumbens, where they modulate dopamine release and motivational behaviour. These mechanisms are the starting point for analyzing the potential role of GLP-1 receptor agonists in modulating addictive behaviors [8,9].

5.1. Alcohol

5.1.1. GLP-1 — a modulator of alcohol reinforcement

Current preclinical data indicate that the behavioral and neurochemical consequences of alcohol exposure are attenuated by GLP-1 receptor activation. A reduction in voluntary alcohol intake, a decrease in alcohol preference, and a reduction in operant behaviors associated with obtaining alcohol have been demonstrated following administration of GLP-1RAs [8,9]. This demonstrates that not only the act of consumption but also the motivation to seek the substance is modulated. Alcohol increases dopamine release in the nucleus accumbens. This enhances its rewarding value, whereas GLP-1RA activation leads to a reduction in this response. This can be interpreted as a weakening of positive reinforcement [9]. A correlation has also been demonstrated between GLP-1 receptor blockade and increased alcohol consumption. This may suggest the existence of an endogenous mechanism inhibiting excessive consumption.

5.1.2. Effects on the abstinence phase and relapse

The period of abstinence also plays a significant role in alcohol dependence. It is characterized by negative emotional states and an increased susceptibility to relapse. In studies, liraglutide and semaglutide reduced the severity of withdrawal symptoms and decreased the intensity of alcohol consumption following a period of deprivation [8,9]. GLP-1RAs therefore not only enhance positive reinforcement but also reduce the discomfort associated with abstinence.

5.1.3. Clinical Data: RCTs vs. Cohort Studies

In randomized trials involving patients with AUD, overall analyses did not show a clear reduction in alcohol consumption [10]. However, these results should be interpreted with caution, as both the number of participants and the duration of follow-up were limited. On the other hand, in cohort analyses involving populations of hundreds of thousands of people, the use of GLP-1RAs was associated with a significant reduction in the risk of alcohol-related events. This applied to both new diagnoses of AUD and relapses [9,10]. The magnitude of the observed effect may suggest that the impact of GLP-1 on alcohol-related behaviors is of great clinical significance, even though this does not always translate into statistically significant differences in small RCT samples. Importantly, this effect is not solely secondary to weight loss, as the reduction in AUD risk occurred independently of body weight [9]. This demonstrates a direct impact on the central mechanisms regulating reward value.

5.2. Nicotine

The data on nicotine are less extensive than those on alcohol. However, the shared dopaminergic mechanism suggests a similar effect of GLP-1RAs — nicotine also increases dopamine release in the nucleus accumbens. The presence of GLP-1 receptors in mesolimbic structures provides the basis for modulating this response. Clinical studies have shown that, in some analyses, GLP-1RAs reduced nicotine use [7]. From a neurobiological perspective, it can be assumed that GLP-1RAs reduce the subjective rewarding value of nicotine, limit the intensity of craving, and also influence relapse behavior.

5.3. Other substances

Preliminary data from preclinical models suggest possible effects of GLP-1RAs on opioid, cocaine and amphetamine self-administration, again via modulation of mesolimbic dopaminergic transmission [7,15]. Clinical evidence in humans, however, remains very limited, and dedicated trials are needed.

6. DISCUSSION

Evidence accumulated over the past five years supports the concept that GLP-1 receptor agonists act on a broad brain–metabolism axis, with implications that extend well beyond glycaemic control and weight loss. Modulation of HPA-axis activity, reward processing and neuroinflammation provides a biologically coherent framework linking metabolic disease, mood disorders and addiction [3,4,11,14].

Clinical findings are most consistent for depressive symptoms in patients with comorbid obesity or diabetes, and for alcohol-related outcomes in large cohort studies [5,6,9,14]. Smaller randomised trials, often underpowered for behavioural endpoints, do not always reproduce these effects, highlighting the need for dedicated psychiatric trials with appropriately selected populations and outcomes [10,13].

Safety considerations are equally important. Current systematic reviews do not show a consistent increase in suicidality or other serious psychiatric adverse events during GLP-1RA therapy, although vigilance remains warranted in vulnerable patients [11,12]. Long-term effects on cognition, particularly in patients with metabolic syndrome or early neurodegeneration, are an area of active investigation [4,16].

From a translational perspective, GLP-1RAs may become a useful adjunct in integrated care models that combine metabolic and psychiatric management, especially for patients in whom obesity, diabetes and mood disorders coexist. Their role in primary prevention of mental health complications among high-risk metabolic populations deserves further evaluation [17,18,19].

7. CONCLUSIONS

GLP-1 receptor agonists exemplify a shift from organ-centred pharmacology towards integrated treatments acting on the brain–metabolism axis. Available evidence indicates antidepressant and anti-anxiety signals, favourable effects on substance use — most notably alcohol — and possible benefits for cognition and quality of life [5–14].

These effects rest on a converging mechanistic basis: modulation of the HPA axis, dopaminergic reward circuits, neuroinflammation and neuroplasticity [3,4,11]. Importantly, several effects appear to be independent of weight loss, suggesting a direct central action of GLP-1RAs [9].

Robust randomised controlled trials with adequate power, longer follow-up and psychiatric primary endpoints are required to confirm clinical efficacy, refine indications and identify patient subgroups most likely to benefit [13,14]. Until such evidence becomes available, GLP-1RAs should be used primarily for their approved metabolic indications, with mental health benefits considered as a potentially valuable secondary outcome.

Integrating GLP-1RAs into multidisciplinary care models — combining endocrinology, psychiatry, primary care and behavioural interventions — may offer an effective strategy for patients in whom metabolic and mental health disorders coexist. Their potential to address both somatic and psychosocial aspects of disease aligns well with contemporary models of integrated, patient-centred medicine.

DISCLOSURE

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Conceptualization: Michalik Zuzanna, Kryś Zuzanna.

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Writing – Introduction and Methodology: Michalik Zuzanna, Kapelka Klaudia.

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CRediT Author Contributions (taxonomy)

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