Epidemiology and Social Psychiatry

O002

Anxiety and Mood Disorders on the Rise: Exploring Clinical Profiles and Risk Factors in the Netherlands

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Introduction: While an increase in mental disorders has been suggested, the role of societal changes, such as sociodemographic, vulnerability, or health-lifestyle factors, on this increase is scarce. This information is however crucial for health care policy and planning.

Objectives: We examined trends in the 12-month prevalence of anxiety and mood disorders, their clinical profiles, and how sociodemographic, vulnerability, and health-lifestyle risk factors may have contributed to these trends.

Methods: We used data from 11,615 respondents (mean age= 43.5, 53.5% female) of the Netherlands Mental Health Survey and Incidence Study, examining the general population in 2007-2009 (NEMESIS-2, n= 6,646) and 2019-2021 (NEMESIS-3, n= 4,969). The Composite International Diagnostic Interview 3.0 was used to determine DSM-IV diagnoses. Logistic regression and interaction analyses were then conducted to assess the association between risk factors and the prevalence of anxiety and mood disorders, while also evaluating changes over time between the two cohorts.

Results: The 12-month prevalence of all types of anxiety and mood disorders significantly increased from 2007–2009 to 2019–2022, with increases ranging from 56% to 125%. Clinical profiles of those with disorders were not milder in 2019-2022; there was greater mental health care use, a higher number of disorders, and an earlier age of onset. There was no consistent evidence that sociodemographic, vulnerability, or health-lifestyle risk factors became more prevalent over time or had a greater relative impact over time.

Conclusions: Our study showed a consistent increase in 12-month prevalence across all anxiety and mood disorders over the past decade. This rise in prevalence could not be explained by an increase in the absolute or relative impact of specific risk factors, nor were there significant differences in clinical profiles over time.

Disclosure of Interest: None Declared

Anxiety Disorders and Somatoform Disorders

O003

The relationship between kynurenine metabolites and executive functions in patients with generalized anxiety disorder

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Introduction: Generalized anxiety disorder (GAD) is a common psychiatric condition characterized by excessive worry, concentration issues, and insomnia. Despite numerous studies, its neurometabolic mechanisms remain unclear.

Objectives: This study aims to compare the levels of kynurenine pathway metabolites between GAD patients and healthy control groups, and to investigate the relationship between kynurenine metabolism products, executive functions, and disease severity in GAD patients.

Methods: The study included 41 GAD patients and 41 healthy controls. Participants were enrolled after ruling out major depressive disorder using the Beck Depression Inventory. They then completed the Sociodemographic and Clinical Data Form, the Wisconsin Card Sorting Test (WCST), the Trail Making Test (TMT) A and B, the Digit Span Test (DST), the Verbal Fluency Test (VFT), the Stroop Test, and the State-Trait Anxiety Inventory. Venous blood samples were collected for serum metabolite measurements. Levels of kynurenic acid (KYNA), quinolinic acid (QUIN), tryptophan (TRP), 3-hydroxykynurenine (3-HK), kynurenine (KYN), and 3-hydroxyanthranilic acid (3-HAA) were measured using liquid chromatography-mass spectrometry (LC-MS).

Results: We found that GAD patients performed significantly worse in terms of the number of categories completed on the WCST ($p=0.014$), TMT-A ($p<0.001$), TMT-B ($p=0.015$) and Stroop Test sub-scores ($p<0.001$) compared to the healthy control group. GAD patients had significantly higher QUIN levels and a lower KYNA/QUIN ratio ($p<0.001$) than the healthy control group, while the control group had a higher 3-HK/KYN ratio ($p=0.008$). A negative correlation was found between DST scores and 3-HAA ($r = -0.311$, $p = 0.048$), as well as between the KYNA/KYN ratio and the stroop test subscore ($r = 0.368$, $p = 0.019$). In the GAD group, we found a positive correlation between kynurenine levels and state anxiety scores ($r=0.34$; $p=0.032$). In regression analysis, the KYNA/QUIN ratio significantly reduced GAD risk ($p=0.001$; OR: 0.531), independent of test performance.

Conclusions: Our study suggests that neurotoxic metabolites in the kynurenine-tryptophan metabolism may explain the executive function impairments observed in GAD. A key finding is that higher KYNA/QUIN ratios significantly reduce GAD risk, which

is etiologically important and provides valuable guidance for future research.

Disclosure of Interest: None Declared

Obsessive-Compulsive Disorder

O005

Effectiveness of Repeated Ketamine Infusions in Treatment Non-Responding Obsessive Compulsive Disorder: a Randomised Controlled Trial

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Introduction: Though glutamate modulators have been increasingly used with some success in cases of OCD resistant to SRIs, there is limited data on the use of ketamine in OCD. There is one study on the use of multiple ketamine infusions in SRI-resistant OCD, but no studies have yet compared the effectiveness of multiple infusions of ketamine with multiple infusions of an active comparator agent. Benzodiazepines are commonly prescribed for OCD despite the lack of recommendations. The current study was a hospital-based prospective, single-blind, randomised controlled trial conducted over a period of one and a half years to compare the effectiveness of multiple infusions of ketamine with those of midazolam.

Objectives: To look into the immediate effects of ketamine infusion in terms of reduction in illness severity in OCD treatment non-responders in comparison to midazolam infusion.

To determine the time-point associated with the largest change in symptom severity in patients receiving repeated ketamine infusions in comparison to midazolam infusions.

To determine the overall proportion of response of OCD treatment non-responders to ketamine infusions in comparison to midazolam infusions.

Methods: In a hospital setting, we compared the effectiveness of 6 sessions of ketamine infusions (0.5 mg/kg body weight) with that of 6 sessions of midazolam infusions (0.045 mg/kg body weight), given on alternate days on a Monday-Wednesday-Friday schedule, in 30 patients with treatment non-responding OCD. Assessments were made using rating scales: DY-BOCS, MADRS, HAM-A, CADSS and SAFTEE.

Results: At 1 hour and 4 hours after the 1st ketamine infusion, 26% of patients achieved treatment response, while none in the midazolam group did so at these time points (Figures 1 and 2). Maximum symptom reduction occurred after the 1st infusion. By the 6th infusion, 40% of ketamine patients achieved treatment response, compared to 20% in the midazolam group. At 4 weeks after the last infusion, only 1 patient (6%) in the ketamine group maintained treatment response, with none in the midazolam group. Overall, the result indicates that the ketamine group showed significant improvement compared to the midazolam group (F=1.541, p=0.048) with a medium effect size (η2=0.056) (Fugure 3). There were no statistically significant differences between the two groups in terms of overall reductions in MADRS.

Image 1:

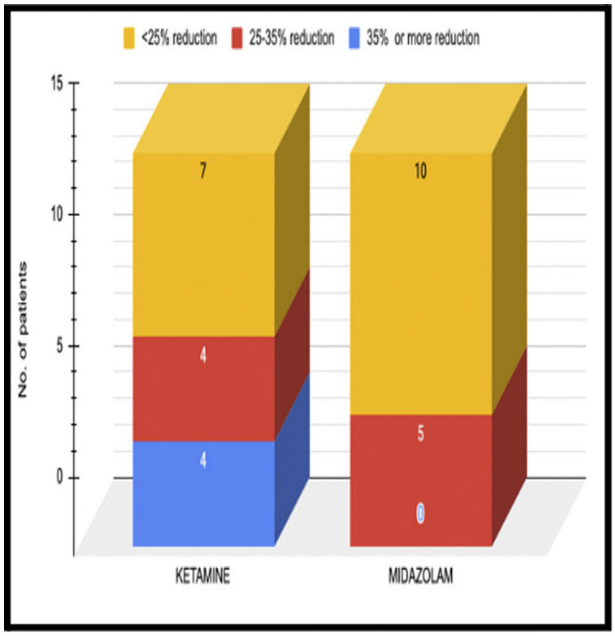

Figure 1: Stacked column chart showing the distribution of degrees of % reductions in DY-BOCS Global Score in ketamine and midazolam groups at 1 hour after 1st infusion.

Image 2:

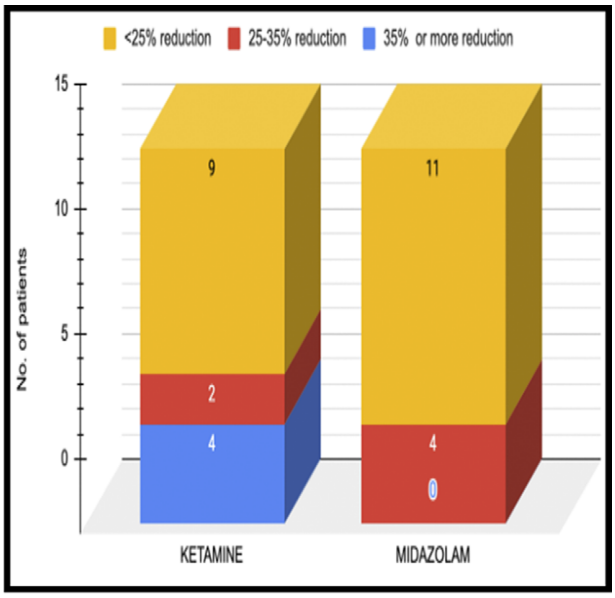

Figure 2: Stacked column chart showing the distribution of degrees of % reductions in DY-BOCS Global Score in ketamine and midazolam groups at 4 hours after 1st infusion.