learning is particularly suitable to address this limit, given its ability to make single-subject predictions on unseen data, while managing highly correlated data [4].

Aims. The present study aimed at assessing whether clinical data, individual polygenic risk scores (PRSs) for different psychiatric disorders, and adverse childhood experiences, could predict lifetime suicide attempts among mood disorder patients using machine learning.

Methods. Blood samples of 154 currently depressed mood disorder patients were genotyped with Infinium PsychArray-24 BeadChip. After quality check of genetic data, genotype imputation was conducted with Minimac4, and PRSs for major depressive disorder, bipolar disorder, schizophrenia, anorexia nervosa, attention and hyperactivity disorder, and autism spectrum disorder were calculated with PRS-cs and PLINK1.9. Adverse childhood experiences (Childhood Trauma Questionnaire, CTQ), clinical data including diagnosis, number of lifetime episodes, and severity of the current depressive episode (Hamilton Depression Rating Scale, HDRS-21), were assessed at participants' enrolment. Clinical data, CTQ, and PRSs were entered as predictors into an elastic net regression, both separately and combined. A nested 5-fold cross-validation was employed to estimate models' performances in discriminating suicide attempters (SA, n=25) from non-attempters (nSA, n=129). Finally, a 5000 bootstrap procedure allowed to estimate the importance of predictors.

Results. Accuracies of the elastic net models are summarised in Table 1. Specifically, the model combining clinical data, CTQ, and PRSs achieved the highest accuracy in discriminating SA from nSA, with a total accuracy of 74%, sensitivity of 32%, specificity of 82%, and 0.6 of area under the receiver operator curve (AUC).

Table 1. Summary of models' performances differentiating SA from nSA.

Predictors	Total accuracy (%)	Sensitivity (%)	Specificity (%)	Balanced accuracy (%)	AUC
Clinical	55	48	57	52	0.6
CTQ	52	44	54	49	0.45
PRSs	47	52	46	49	0.47
Combined	**74**	**32**	**82**	**57**	**0.6**

The five top-ranked predictors of the combined model were HDRS-21 agitation and retardation, number of lifetime episodes, CTQ sexual abuse, and schizophrenia PRS.

Conclusions. The model combining clinical, environmental, and genetic data outperformed the ones built on single modalities. Our results hence highlight the importance of integrating different features to create an effective suicidality predictive model in mood disorders. Future studies may include other types of data, such as neuroimaging or inflammatory markers, helping in tailoring early prevention strategies within a precision psychiatry framework.

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OBESITY AND DEPRESSIVE SYMPTOMS: INVESTIGATING THE MEDIATING ROLE OF FASTING GHRELIN SERUM LEVELS

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Introduction: Obesity has been shown to be associated to higher depressive symptoms and lowered serum levels of the orexigenic hormone ghrelin. Previous studies suggest that ghrelin might exert antidepressant effects in mice. Furthermore, patients with MDD showed lowered ghrelin levels compared to healthy participants. However studies report depressiogenic effects of ghrelin as well. It is yet unknown whether the association between obesity and depressive symptoms can be explained through lowered fasting serum ghrelin levels. In the current study we thus aim to systematically address this issue by analyzing the relationship between obesity, fasting ghrelin serum levels and depressive symptoms in a population based sample.

Methods: We used data from the LIFE-study (from the Leipziger Forschungszentrum für Zivilisationskrankheiten), a population based panel study with over 10.000 participants from Leipzig in eastern Germany. The sample was stratified for age and gender and focused on the population in the age-range from 40 to 79 years. Depressive symptoms were assessed by the Center for Epidemiological Studies Depression Scale, a questionnaire that measures self-reported depressive symptoms and ghrelin serum levels were quantified using radioimmunoassay after an overnight fast. We excluded all participants from the analysis that had a severe acute disease (except depression, obesity, diabetes, hyperlipidemia and hypertension), an underweight (BMI <18.5) or were treated with psychotropic drugs.We used hierarchical linear mixed effects models with multiple sets of control variables and mediation analyses implemented in R to test our hypotheses. The statistical significance of the models were assessed with full-null-model comparisons and p-values were Bonferroni-adjusted for multiplicity control. The whole project was pre-registered on the open science framework [1].

Results: Preliminary data analysis revealed an association between higher BMI and more frequent depressive symptoms ($\beta = 0.114$, $p < 0.001$, $n = 8982$). The model explained 0.6% of the variance (adjusted R-squared = 0.006). Furthermore, we could find a stable relationship between higher BMI and lower fasting ghrelin serum levels ($\beta = -23.04$, $p < 0.001$, $n = 1613$). The model with BMI as a predictor explained 4.59% of the variance in ghrelin levels (adjusted R-squared = 0.04591). The last model yielded no significant results for an association between lowered ghrelin serum levels and more frequent depressive symptoms ($p = 0.57$, $n = 1613$). Because we could not corroborate the association between depressive symptoms and ghrelin serum levels, no mediation analysis was performed.

Conclusion: Even though our data confirmed the link between BMI and depressive symptoms our results could not confirm that fasting ghrelin serum levels mediate this relationship. The next generation of research should investigate other factors that could explain the increased susceptibility of individuals living with obesity for depressive symptoms. Especially traumatic experiences in early childhood, lack of psychosocial support and weight-related stigma may be considered.

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LINKING MENOPAUSE, HISTORY OF DEPRESSION, AND PROXIES OF BIOLOGICAL AGING IN THE UK BIOBANK COHORT

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