# Insulin Resistance in Depression: A Meta-Analysis of Variation

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**What is the Methodological Question Being Addressed?** Should we stratify participants according to the presence of insulin resistance in clinical trials targeting insulin pathways?

**Introduction** If Insulin resistance is altered in depression, and if it is a universal occurrence in depression, remains unclear. This particular point has implications for clinical trials design targeting insulin pathways since insulin resistance can be an essential factor for stratification. We aim here to verify whether insulin resistance is altered in depression and whether alterations are homogenous or if heterogeneity exists. We also aimed to investigate if alterations in insulin resistance are causal to the development of depression.

**Methods** We identified 8,769 articles through electronic searches and conducted a meta-analysis of blood insulin levels and HOMA index in depression. We pooled the ES results according to the inverse variance method accounting for random effects. Meta-analyses of relative variability of individuals with depression were performed using the coefficient of variation ratio (CVR). Finally, we performed Two-sample Mendelian randomization (2SMR) to assess the causal associations between insulin resistance and depression.

**Results** In total, 66 articles were included. Insulin levels were increased in acute depression in participants without (ES 0.26, 95% CI 0.02 to 0.50, n=1,181) and with (ES 0.30, 95% CI 0.21 to 0.39, n=220,422) antidepressants but not in remission. HOMA index was increased only in those with medicated acute depression. In addition, neither insulin nor HOMA increased after a treatment course with antidepressants both in responders and in non-responders. The insulin and HOMA CVR were increased in acute depression. We found no support for causality using fasting insulin as the instrumental variant in a 2SMR.

**Conclusion** Insulin resistance is increased in acute depression but not in remission. There is evidence for subgroups of insulin resistance in depression, consistent with the hypothesis that metabolic alterations are specific to a subset of patients. However, we did not find support for a causal relationship between insulin resistance and depression. Studies targeting insulin pathways in depression should stratify participants according to the presence of insulin resistance.

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## Keywords


| Keywords |
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| biomarker |
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**Disclosures** None

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