vehicle-mediated) expression. Also in cortical ROIs, only a treatment-x-time interaction was observed. Third group of observations – Antipsychotic comparisons. In striatal ROIs, again only a treatment-x-time effect was found. Gene expression was significantly higher by all antipsychotics compared to vehicle and by haloperidol compared to asenapine and olanzapine. These effects were completely lost after prolonged treatments, since no treatment effect was found at this time-point. Notably, prolonged treatment significantly reduced antipsychotic-mediated expression (with the exception of olanzapine) and increased basal expression.

These data indicate that significant differences in gene expression among ROIs were not moderated by duration of treatment or type/doses of antipsychotics. However, duration of treatment significantly moderated antipsychotic-effect on gene expression. Differential acute effects of antipsychotics on gene expression in both the striatum and the cortex were lost after prolonged administration. However, haloperidol induced higher gene expression in both the cortex and striatum, but haloperidol-mediated modulation of gene expression was lost with time. Asenapine maintained a modulatory effect with time, since it modulated gene expression acutely in striatum and chronically in the cortex.

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P.890 The effect of ketamine on psychopathology and implications for understanding schizophrenia: a meta-analysis

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Background: Ketamine's ability to induce the full range of psychotic symptoms has established it as model of psychosis in both humans and animals. This model has been important for gaining a greater understanding of the nature of glutamate and NMDA receptor dysfunction in schizophrenia. It has also been used in the development of drugs targeting the glutamate system. Despite this, it is not known how consistently ketamine generates symptoms, the average magnitude of its effect, or the experimental design factors that might influence this. Our aim was to conduct a meta-analysis to determine the effect of ketamine on psychopathology in healthy participants, and the experimental factors affecting this.
Methods: In accordance with the PRISMA guidelines, MEDLINE, EMBASE and PsychINFO databases were systematically searched for studies investigating the change in the Brief Psychiatric Rating Scale (BPRS), in response to an acute ketamine challenge in healthy participants compared to a placebo condition. At least two independent investigators selected the studies, and extracted study-level data for a random-effects meta-analysis. The primary outcome measure was the standardized mean difference (Hedges' g) in total, positive and negative BPRS scores in healthy participants in response to an acute ketamine challenge, compared to a placebo condition. To determine the impact of the study design on the results, sub-group analyses were conducted examining the effect of: within-person versus independent group study design, blinding status, infusion method and different positive symptom inclusion criteria.
Results: Our search identified 7991 citations, of which 26 studies met the inclusion criteria. The overall sample included 408 healthy participants exposed to the ketamine condition, and 412 healthy participants exposed to the placebo condition. We found that the acute administration of ketamine induced an increase in psychopathology in healthy participants. This was seen as a statistically significant increase in the total BPRS score (Hedges $g = 1.64$, 95% CI, 1.20 to 2.08, $p < 0.0001$) positive BPRS score (Hedges $g = 1.63$, 95% CI, 1.28 to 1.98, $p < 0.0001$) and negative BPRS score (Hedges $g = 1.46$, 95% CI, 1.11 to 1.82, $p < 0.0001$) compared with a placebo condition. The use of a non-blinded or single blinded method significantly increased the magnitude of the effect for total ($p = 0.002$) and negative BPRS scores ($p = 0.001$). A within-person design increased the magnitude of effect for the total BPRS score ($p = 0.015$). A meta-regression of effect size against age found that increasing age reduced the magnitude of effect for negative symptoms ($p = 0.02$).
Conclusions: These findings suggest that acute ketamine administration consistently induces psychopathology, with a similar effect on positive and negative symptoms in healthy participants. The findings also highlight the importance of methodological design on the magnitude of the effect.

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P.891 Experimental study of the influence of atorvastatin and rosuvastatin on inflammation

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Purpose: Frequent and prolonged use of statins as lipid-lowering agents raises the issue of their pleiotropic effects and the need to extend their clinical indications. Accumulating experimental evidence suggests that statins have neuroprotective effects against a variety of CNS diseases. In addition to endothelial protection, modulation of nitric oxide synthase (NOS), and antioxidative effects, statins inhibit a number of inflammatory processes known to be important to brain damage [1]. Kalonia et al investigate the neuroprotective effect of statins in quinolinic acid induced neurotoxicity in rats. Atorvastatin, simvastatin and fluvastatin significantly decrease the TNF - α level and striatal lesion volume in quinolinic acid treated animals indicating their anti-inflammatory effect [2]. Traumatic brain injury is another condition which triggers inflammatory response. Wang et al demonstate that atorvastatin and simvastatin reduce functional neurological deficits after experimental