New Onset Capgras and Cotard Delusions in Schizoaffective Disorder

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Abstract

Background. Cotard is a syndrome characterized by ideas of damnation or rejection, anxious melancholia, and nihilistic delusions concerning one's own body or existence. Capgras is a syndrome in which the patient believes that the identities of close relatives or friends are replaced by others. Capgras and Cotard delusion are rarely reported in one individual simultaneously, which we will describe in this poster.

Case Report. DD is a 38-year-old female with history of schizoaffective disorder, bipolar type, PTSD, and reported TBI who presented to the ED exhibiting paranoia and delusions in the setting of medication non-adherence and increased psychosocial stressors. She endorsed her organs were rotting, and that she was stolen by imposters who claim to be her family from birth. Chart review revealed previous hospitalizations involving mania and paranoid delusions; however, these Cotard and Capgras delusions were new. She also had reported TBI injury from domestic abuse, as well as emotional and sexual trauma. Her Cotard delusions resolved with risperidone 6 mg daily and valproic acid 500 mg BID. However, her Capgras delusions were maintained after 22 days of inpatient hospitalization. On discharge, she continued to refuse reconnecting with her family and was subsequently set up with an intensive outpatient program.

Discussion. Cotard and Capgras delusions are considered to reflect different interpretations of similar anomalous experiences. The persecutory delusions and suspiciousness often noted in Capgras contribute to the patient's mistaking a change in themselves for a change in others, whereas people who are depressed in Cotard exaggerate the negative effects of the same change whilst attributing it to themselves. Although these two delusions are phenomenally distinct, they may therefore represent attempts to make sense of fundamentally similar experiences. The anatomical origin of these disorders have been reported to be from a disconnection between the temporal cortex and the limbic system as described in a patient with ischemic stroke. DD's PTSD and TBI with possible damage to her left tempoparietal lobe may have predisposed her to comorbid delusions. Although DD's Cotard delusions abated with antipsychotic treatment, her Capgras delusions were maintained. Recently losing custody of children may have contributed to the depression that precipitated her Cotard subtype 1 delusion. Her response to risperidone is consistent with a previous case report that highlighted the effectiveness of antipsychotics in the absence of ECT with Cotard delusion. In regards to her ongoing Capgras delusion, the chronic abuse by DD's family may have led to a more persistent psychodynamically meaningful negation of DD's sense of relationship to her family.

Conclusion. Capgras and Cotard syndromes, though rare, can present together. Additional studies are needed to understand the pathophysiology and treatment outcomes.

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TAAR1 Agonist Ulotaront Improves Glycemic Control and Reduces Body Weight in Rodent Models of Diabetes, Obesity, and Iatrogenic Weight Gain

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Abstract

Introduction. Preclinical evidence has identified the trace amine-associated receptor 1 (TAAR1) as a novel regulator of metabolic control. Ulotaront is a TAAR1 and 5-HT_{1A} agonist currently in Phase 3 clinical trials for the treatment of schizophrenia. Here we summarize preclinical results assessing the effects of ulotaront on weight and metabolic parameters.

Methods. Effects of ulotaront administration were evaluated on oral glucose tolerance (oGTT), gastric emptying, and in rodent models of weight gain (high-fat diet [HFD]-, corticosterone-, and olanzapine-induced).

Results. Following 15-day oral administration of ulotaront, rats on HFD showed dose-dependent reduction in body weight, food intake, and liver triglyceride content compared to controls. In addition, a more rapid reversal of olanzapine-induced weight gain and food intake was observed in rats switched to ulotaront (vs. vehicle). Consistent with weight-lowering effects in rats, chronic ulotaront treatment normalized corticosterone-induced weight gain in mice. Assessment of oGTT showed a dose-dependent reduction of glucose excursion in response to acute ulotaront administration in naive and diabetic *db/db* mice. Ulotaront administration also delayed gastric emptying in mice—a likely mechanism driving reductions in glucose excursions during the oGTT. Whole-brain c-fos imaging of ulotaront-treated mice revealed increased neuronal activity in several brain regions associated with regulation of food intake and metabolic signals.

Conclusions. The data indicate that ulotaront not only lacks metabolic liabilities typically associated with antipsychotics but can reduce body weight and improve glucose tolerance in rodent models. The underlying mechanisms may include TAAR1-mediated peripheral effects on glucose homeostasis and/or direct modulation of homeostatic and hedonic neurocircuits regulating energy balance. The beneficial metabolic effects of ulotaront may suggest a substantially improved risk-benefit profile compared to established antipsychotics.

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