sulfur taste. Scrambled eggs had no smell but mild sulfur taste, which changed over time to a rotten egg smell and taste. With nose clips, scrambled eggs had 0/10 taste, without the nose clips the smell of sulfur was 3/10.

RESULTS: Olfaction: Normosmia to threshold and Retronasal Smell Index: 2 (abnormal): Gustatation : Normogeusia to all. Mild hypogeusia to sodium chloride. MRI: Multiple foci of periventricular and deep white matter demyelization.

DISCUSSION: Rotten egg smell maybe mediated through retro-nasal pathways, since nasal obstruction eliminated the rotten egg taste. Eggs can possibly be developed as a home device to assess chemosensory function.

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Burning Mouth Syndrome as a Focus of Delusion

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BACKGROUND: Parasitosis is a fixed belief of being infested with pathogens against all medical evidence [Freudenmann RW, Lepping P, 2009] Method: Case Report: A 53 year old right handed female presented with progressively severe BMS for 1 years. She noticed that aromas would project from her nose into her mouth and would experience this taste for days. Looking at the source of the odor would precipitate her to sense the smell of the product, immediately followed by the taste and then burning of the tongue, mouth and vagina. Fumes would eminent from her mouth, nose and anus. Five days prior she stopped eating and drinking. She had not brushed her teeth, showered, nor bathed for 3 weeks. Odors smell like ammonia and blood, which upon inhalation, effuse into her mouth which tastes like chemicals. Thereupon, she immediately experiences burning of her tongue and palate.

RESULTS: Abnormalities: Disheveled: Cacosmious. Personal hygiene poor. Facial expression odd and inappropriate. Loud but low quantity of speech. Unable to interpret similarities or proverbs. Calculation: poor.

CONCLUSION: In those who present with BMS, query as to the delusional nature of their symptoms is warranted and may suggest a treatment strategy.

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Long-Term Outcomes with Valbenazine 40 mg/day in Adults With Tardive Dyskinesia

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ABSTRACT: Study Objective: Tardive dyskinesia (TD), a persistent and potentially disabling movement disorder, is associated with prolonged exposure to antipsychotics and other dopamine receptor blocking agents. Valbenazine (VBZ) is a novel and highly selective vesicular monoamine transporter 2 (VMAT2) inhibitor approved for the treatment of TD in adults. Using data from two long-term phase 3 studies (KINECT 3 [K3], NCT02274558; KINECT 4 [K4], NCT02405091) and a rollover study (1506, NCT02736955), the long-term outcomes of once-daily VBZ on TD were examined in participants who received 40mg or had a dose reduction from 80 to 40mg.

METHODS: The effects of VBZ 40mg (as well as VBZ 80mg) were evaluated in the following studies: the pivotal K3 study (6 weeks double-blind, placebo controlled), the extension phase of K3 (42 additional weeks of VBZ, 4 week discontinuation), and the open-label K4 study (48 weeks of VBZ, 4 week discontinuation). Completers from K3 extension and K4 were invited to participate in 1506 (up to 72 additional weeks of VBZ or until commercial availability of VBZ). Few participants reached Week 60 (n=4) or Week 72 (n=0) in the 1506 study before termination. Analyses focused on VBZ 40mg in two populations: pooled K3/K4 (participants who received VBZ 40mg throughout K3 or K4 or who had a dose reduction [80/40mg] during K3 or K4); and 1506 (participants who received VBZ 40mg from beginning of K3 or K4 to last visit in 1506 or who had a dose reduction [80/40mg] at any time). Outcomes for the K3/K4 population included mean change from baseline (CFB) in Abnormal Involuntary Movement Scale (AIMS) total score (sum of items 1-7) and AIMS response (≥50% total score improvement from baseline) at Week 48 of K3 or K4. Outcomes for the 1506 population included a Clinical Global Impression of Severity-Tardive Dyskinesia (CGIS-TD) score ≤2 ("normal, not at all ill" or "borderline ill").

RESULTS: In the K3/K4 population, AIMS CFB to Week 48 indicated mean TD improvements in participants who received 40mg continuously (40mg, -5.7 [n=54]) and in

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those who had a dose reduction to 40mg (80/40mg, -6.2 [n=13]). In addition, a majority of these participants had an AIMS response after 48 weeks of treatment (40mg, 53.7%; 80/40mg, 53.8%). In the 1506 population, the percentage of participants who had a CGIS-TD score ≤2 (rating of "normal, not at all ill" or "borderline ill") at Week 12 was 63.6% (7/11) in the 40mg group and 30.8% (4/13) in the 80/40mg group. Data from Weeks 24 to 60 of 1506 were limited by the small sample sizes (<10 participants each in 40mg or 80/40mg group at each of these visits).

CONCLUSIONS: Based on these analyses and results from published studies, VBZ 40mg may be an effective long-term option for some TD patients. Dose reductions from 80 to 40mg, if necessary, did not appear to compromise long-term benefit.

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Reframing the Approach to the Diagnosis and Treatment of Borderline Personality Disorder in Adolescents

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BACKGROUND: Using the same DSM-5 criteria as in adults, BPD in adolescents is defined as a 1-year pattern of immature personality development with disturbances in at least 5 of the 9 domains listed in the DSM-5. BPD can now be reliably diagnosed as young as 13 using one of several standardized clinician, or self-rated diagnostic instruments. Unfortunately published US and Canadian positions regarding pharmacological treatment have been, With regard to evidence-based studies, pharmacological treatment is not recommended and, if ultimately required, should be limited to second-generation antipsychotics. Fortunately, the last decade s extensive advancements in brain-mapping have provided more clarity about the various brain dysfunctions underlying the symptoms/traits presenting in BPD, providing new opportunities to address these primarily Fronto-Limbic dysfunctions neuropharmacologically and potentially, significantly ameliorate. Thus, in turn, likely enhancing the effectiveness of the newer available therapies.

OBJECTIVES: The current study explores the feasibility of more effectively managing BPD symptoms/traits with a unique medication protocol consisting of two medications; an anticonvulsant (oxcarbazepine) and a dopaminergic (amantadine HCl), without use of an antipsychotic medication.

METHODS: Subjects were 147 females, ages 13-16, with the diagnosis of BPD treated with the described medication protocol in a residential facility. Positive outcome was described as achievement and maintenance of greater than 50% improvement from baseline admission state of functioning for 1 year. They were discharged when stable and having achieved greater than 50% improvement from baseline. Outpatient prescribers were requested to be compliant with the treatment protocol. However, some were non-compliant, substituting antipsychotic medication instead. Care givers were surveyed at 6 months and 1 year to determine whether their child was maintaining greater than 50% improvement.

RESULTS: The percent maintaining greater than 50% improvement was calculated for those whose caregivers reported continuation of the medications as prescribed, versus those whose prescribers changed the medications to the Community Standard. Of those compliant with the medication protocol, 61 of 86 (71%) maintained >50% improvement. Of those moved to the Community Standard approach, 19 of 61 (31%) maintained >50% improvement. Using Chi Square analysis, there was a significant relationship between maintenance of improvement and medication protocol compliance. Chi Square, Fisher's exact test = $p<0.001$.

CONCLUSION: The results indicate that, for adolescents 1 year post-discharge from residential treatment for BPD, continuation of the above described medication protocol provides significantly higher rates of maintenance of achieved symptom improvement. Further controlled studies are needed.

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Successful Treatment of Major Depressive Disorder with Moclobemide After Recurrent Hyponatremia Induced by Multiple Antidepressant Classes

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ABSTRACT: Background: Antidepressant-induced hyponatremia/syndrome of inappropriate antidiuretic hormone (SIADH) can cause significant morbidity and mortality. Antidiuretic hormone release due to stimulation of central serotonin 5HT1C, 5HT2 and α-1 adrenergic receptors is thought to cause this adverse effect (Spigset, 1995). Evidence on which antidepressants are more likely to cause hyponatremia is inconsistent (Coupland, 2011;

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