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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