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Introduction Lurasidone is an atypical antipsychotic that demonstrated efficacy in the treatment of adults with schizophrenia in the dose range of 37–148 mg/day.
Objective/Aims The objective of this analysis was to evaluate the efficacy of lurasidone in adolescent patients with schizophrenia.
Methods Adolescents (13–17 years old) diagnosed with schizophrenia were randomly assigned to six weeks of double-blind treatment with lurasidone 37 mg/day, 74 mg/day or placebo. Changes from baseline to week 6 in PANSS total and subscale (positive, negative, general psychopathology, excitability) scores were evaluated using mixed-model repeated-measures analysis.
Results A total of 326 patients (mean age, 15.4 years) were randomized and received lurasidone 37 mg/day ($n = 108$), 74 mg/day ($n = 106$), or placebo ($n = 112$). The PANSS total score at week 6 demonstrated a placebo-adjusted, least-squares (LS) mean improvement of −8.0 ($P < 0.001$; effect size [ES], 0.51) for the 37 mg/day group and −7.7 ($P < 0.001$; ES = 0.48) for the 74 mg/day group. Placebo-adjusted LS mean change for lurasidone 37 mg/day and 74 mg/day, respectively, was −3.2 ($P < 0.001$; ES = 0.62) and −3.2 ($P < 0.001$; ES = 0.60) on the PANSS positive subscale, −1.7 ($P = 0.011$; ES = 0.41) and −1.6 ($P = 0.022$; ES = 0.35) on the PANSS negative subscale, −2.8 ($P = 0.012$; ES = 0.38) and −2.8 ($P = 0.011$; ES = 0.37) on the PANSS general psychopathology subscale, and −1.1 ($P = 0.016$; ES = 0.36) and −1.8 ($P < 0.001$; ES = 0.53) on the PANSS excitability subscale.
Conclusions In adolescent patients with schizophrenia, lurasidone (37 mg/day and 74 mg/day) demonstrated statistically significant efficacy and clinically meaningful improvement across a wide spectrum of symptoms associated with schizophrenia. Sponsored by Sunovion Pharmaceuticals Inc. ClinicalTrials.gov identifier: NCT01911429.
Disclosure of interest Dr Correll reports being a consultant and/or advisor for Alkermes, Forum Pharmaceuticals Inc., Gerson Lehrman Group, IntraCellular Therapies, Janssen/J&J, Lundbeck, Medavante, Medscape, Otsuka, Pfizer Inc, ProPhase, Sunovion Pharmaceuticals Inc., Supernus, Takeda, and Teva providing expert testimony for Bristol-Myers Squibb Company, Janssen, and Otsuka serving on a Data Safety Monitoring Board for Lundbeck and Pfizer Inc and receiving grant support from Takeda. Drs Goldman, Cucchiaro, Deng and Loebel are employees of Sunovion Pharmaceuticals Inc.

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O063

Efficacy and safety of clozapine in patients with intellectual disability

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Introduction Aggression is common and a major behavioral problem in patients with intellectual disability (ID). Antipsychotics are frequently used for psychosis or challenging behavior. There is little literature regarding utilization of clozapine in patients with ID for aggressive behavior.
Aims and objectives The aims of the study were the evaluation of efficacy and safety of clozapine in treatment of aggression in patients with ID.
Methods A longitudinal naturalistic study including a cohort of 225 consecutive patients with intellectual disability admitted to an acute psychiatric unit between 1 January 2014 and 31 December 2015. Severity of symptoms was assessed at admission with Modified Overt Aggression Scale (MOAS) and Global Assessment of Functioning Scale (GAFS). The data included: demographics, main psychiatric diagnosis, IQ, alcohol/smoking, institutionalization, antipsychotics and another psychotropics, restraint, readiness to discharge (RDQ), side-effects and length of stay.
Results Of 225 potentially eligible individuals, 205 (92.7%) were treated with antypschotics and 110 male (53.56%) with mean age 32.37 (SD = 9.9). Thirty-seven patients (18%), 18 male (48.65%) were treated with clozapine, mean dose 309.45 mg/day (range 100–450 mg/day). Clozapine reduced need for restraint and duration of hospitalization compared with haloperidol ($P < 0.05$).
Conclusions Clozapine was efficient and safety for treating persistent aggression in patients with intellectual disability. There were no seizures, myocarditis or agranulocytosis during study. Larger and randomized trials are needed to fully explore the anti-aggressive benefit of clozapine.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O064

Childhood trauma and cortisol response to the Trier Social Stress Test in symptomatic patients with eating disorders

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Introduction Childhood trauma exposure is associated with the risk of eating disorders (EDs) in adulthood. The biological basis of this link may involve a persistent dysregulation of the endogenous stress response system, in particular the hypothalamic-pituitary-adrenal (HPA) axis, as a consequence of early life maltreatment.
Objective Adult patients with EDs and history of childhood trauma may have a dysregulation of the HPA axis that could be different from EDs patients without childhood trauma exposure.
Aims In order to assess the effects of childhood trauma experiences on HPA-axis activity in EDs, we compared the salivary cortisol response to the Trier Social Stress Test (TSST) of adult patients with EDs according to their history of childhood trauma.
Method Twenty-seven EDs patients and 13 healthy women participated in the study. Salivary cortisol responses during exposure to the TSST was measured. Participants also completed the childhood trauma questionnaire (CTQ) and eating-related psychopathological rating scales.
Results According to CTQ, 15 individuals with EDs reported childhood maltreatment whereas 12 EDs patients and all the healthy women did not experience childhood maltreatment. Compared with the control group, non-maltreated EDs patient group exhibited a slightly enhanced cortisol response to TSST, whereas the group of non-maltreated EDs patients showed a normal cortisol response. Moreover, EDs patients with childhood maltreatment exhibited statistically significant blunting of cortisol compared to non-maltreated ones.
Conclusions The present findings support the evidence that, in patients with EDs, there is a dysregulation of HPA-axis activity and that childhood trauma exposure may contribute to this dysregulation.
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