is commonplace in 5%-45%, then clinicians might opt to try aripiprazole or the substituted benzamide amisulpride, and closely clinically monitoring for OCS accentuation, in which case, add-on SSRI might be a reasonable course of action, given additive neuroprotective actions by virtue of SSRI-enhanced dentate gyrus neurogenesis. A caution, however, should be exercised here regarding significant pharmacokinetic interactions between SSRIs known to be potent cytochrome P450 inhibitors (e.g., fluoxetine on 2D6) and AAPs (e.g., risperidone as a substrate for 2D6).

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Ahmed Naguy
Al-Manara CAP Centre, Kuwait Centre for Mental Health, Shuwaikh, Kuwait

Address for correspondence: Dr. Ahmed Naguy
Al-Manara CAP Centre, Kuwait Centre for Mental Health, Jamal Abdul Nasser Street, Shuwaikh, Kuwait.
E-mail: ahmednagy@hotmail.co.uk

REFERENCES
1. Dold M, Aigner M, Lanzenerberger R, Kasper S. Antipsychotic augmentation of serotonin reuptake inhibitors in treatment-resistant obsessive-compulsive disorder: A meta-analysis of double-blind, randomized, placebo-controlled trials. Int J Neuropsychopharmacol 2013;16:557-74.
2. Ducasse D, Boyer L, Michel R, Loundou A, Macgregor A, Micoulaud-Franchi JA, et al. D2 and D3 dopamine receptor affinity predicts effectiveness of antipsychotic drugs in obsessive-compulsive disorders: A metaregression analysis. Psychopharmacology (Berl) 2014;231:3765-70.
3. Lim M, Park DY, Kwon JS, Joo YH, Hong KS. Prevalence and clinical characteristics of obsessive-compulsive symptoms associated with atypical antipsychotics. J Clin Psychopharmacol 2007;27:712-3.
4. Zink M. Comorbid obsessive-compulsive symptoms in schizophrenia: Insight into pathomechanisms facilitates treatment. Adv Med 2014;2014:317980.
5. Lin SK, Su SF, Pan CH. Higher plasma drug concentration in clozapine-treated schizophrenic patients with side effects of obsessive/compulsive symptoms. Ther Drug Monit 2006;28:303-7.
6. Kwon JS, Joo YH, Nam HJ, Lim M, Cho EY, Jung MH, et al. Association of the glutamate transporter gene SLC1A1 with atypical antipsychotics-induced obsessive-compulsive symptoms. Arch Gen Psychiatry 2009;66:1233-41.
7. Poyurovsky M, Weizman A, Weizman R. Obsessive-compulsive disorder in schizophrenia: Clinical characteristics and treatment. CNS Drugs 2004;18:989-1010.

Overestimation of Depression Prevalence among Adolescent Students

Sir,

I read with interest the study on the prevalence of depression among adolescent students by Jha et al.[1] in May–June issue of 2017. The authors have used a Hindi translation of Beck Depression Inventory II (BDI-II) to screen 1485 adolescents (of which 1412 responded) aged 14–18 years and reported a point prevalence of any depression to be 49.2%. Guilty feeling, pessimism, sadness, and past failure were the most common self-reported symptoms, which the authors have wrongly interpreted as factors responsible for depression. The factors associated with self-reported depression in their study included “school factors” such as an inability to cope, teasing and physical punishment at school, and “family factors” such as parental conflict and financial constraints.

The reported rates of depression vary according to the instrument used for screening. In a meta-analysis of the
studies on Iranian adolescents,[2] the mean prevalence of depression was 43.55% using the BDI, 15.87% using Symptom Checklist 90, and 13.05% using Children’s Depression Inventory. Similar to the study by Jha et al.,[1] several others Indian studies[3-6] using BDI/BDI-II have reported high prevalence rates of depression among adolescents. One study from Mangalore[7] on college students estimated a prevalence of depression up to 80% using BDI-II screening.

Studies using a two-stage method, where screening is followed by confirmation using structured interviews, give more reliable estimates of depression. In a study from Sweden,[8] 88% of adolescents scored >16 on BDI; however, only 13% of low scorers had depression diagnosis following structured interview. The study by Sarkar et al.[9] using Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS-Present and Lifetime) found a prevalence of the depressive disorder among students from standard I–VII to be 3.13%. Similarly,[10] screening with BDI followed by K-SADS-Epidemiological version 5 in Nigerian adolescents aged 13–18 years resulted in 6.9% prevalence of depression.

In the meta-analysis by Jane Costello et al.,[11] the prevalence estimate for depression under 13 years of age was 2.8% (standard error [SE] 0.5%) and for 13–18 years it was 5.6% (SE 0.3%). They concluded that there is no increase in the prevalence of child, and adolescent depression and the concerns regarding the epidemic of depression are not true. Nevertheless, a recent study in the USA reported an increase in the 12-month prevalence of major depressive episodes from 8.7% in 2005 to 11.3% in 2014 in adolescents.[12]

Although the authors assert that the Hindi translation of BDI-II was extensively piloted, they have not stated the psychometric properties of the translated scale. Another instrument, Patient Health Questionnaire 9, has been well validated as a screening tool[13] and has a Hindi version for use in India, could have been used in the study. Furthermore, the cutoff score of 13 may overestimate depression; hence, a score of 23 for screening has been suggested based on Youden’s index.[14]

**Financial support and sponsorship**
Nil.

**Conflicts of interest**
There are no conflicts of interest.

Samir Kumar Praharaj

Department of Psychiatry, Kasturba Medical College, Manipal, Karnataka, India

**Address for correspondence:** Dr. Samir Kumar Praharaj Department of Psychiatry, Kasturba Medical College, Manipal - 576 104, Karnataka, India. E-mail: samirpsyche@yahoo.co.in

**REFERENCES**

1. Jha KK, Singh SK, Nirala SK, Kumar C, Kumar P, Aggrawal N. Prevalence of Depression among School-going Adolescents in an Urban Area of Bihar, India. Indian J Psychol Med 2017;39:287-92.
2. Sajjadi H, Mohaqeqi Kamal SH, Rafiey H, Vameghi M, Forouzan AS, Rezaei M. A systematic review of the prevalence and risk factors of depression among Iranian adolescents. Glob J Health Sci 2013;5:16-27.
3. Nagendra K, Sanjay D, Gouli C, Kalappanavar NK, VinodKumar CS. Prevalence and association of depression and suicidal tendency among adolescent students. Int J Biomed Adv Res 2012;3:714-9.
4. Malik M, Khanna P, Rohilla R, Mehta B, Goyal A. Prevalence of depression among school going adolescents in an Urban area of Haryana, India. Int J Community Med Public Health 2015;2:624-6.
5. Rani M, Karunanidhi S, Basilea W. Risk and protective factors to depressive symptoms in school-going adolescents. J Indian Assoc Child Adolesc Mental Health 2010;6:101-19.
6. Nair MK, Paul MK, John R. Prevalence of depression among adolescents. Indian J Pediatr 2004;71:523-4.
7. Naushad S, Faroogui W, Sharma S, Rani M, Singh R, Verma S. Study of proportion and determinants of depression among college students in Mangalore city. Niger Med J 2014;55:156-60.
8. Olsson G, von Knorring AL. Beck’s Depression Inventory as a screening instrument for adolescent depression in Sweden: Gender differences. Acta Psychiatr Scand 1997;95:277-82.
9. Sarkar S, Sinha VK, Praharaj SK. Depressive disorders in school children of Suburban India: An epidemiological study. Soc Psychiatry Psychiatr Epidemiol 2012;47:783-8.
10. Adewuya AO, Ola BA, Aloba OO. Prevalence of major depressive disorders and a validation of the Beck Depression Inventory among Nigerian adolescents. Eur Child Adolesc Psychiatry 2007;16:287-92.
11. Jane Costello E, Erkanli A, Angold A. Is there an epidemic of child or adolescent depression? J Child Psychol Psychiatry 2006;47:1263-71.
12. Mojtabai R, Olsson M, Han B. National trends in the prevalence and treatment of depression in adolescents and young adults. Pediatrics 2016;138. pii: E20161878.
Aggressive Behavior and Short–Long Polymorphisms of Monoamine Oxidase A: An Example of Effect of Genetic Molecular Mass Change in Psychological Medicine

Sir,

Aggressive behavior is usually an unwanted serious behavior and there are many reports in psychological medicine on the underlying genetic factors. Of several genetic polymorphisms, monoamine oxidase A (MAOA) polymorphism is widely mentioned for the clinical relationship to aggressive behavior.\[1\] In a recent reported, it was proved that having short polymorphism is related to the increased risk behavior and harmful practice comparing to having long polymorphism.\[2\] Indeed, based on the basic concern on quantum molecular genetics, the genetic molecular mass change due to different polymorphism can be expected. It is no doubt that the patients with short polymorphism have a less molecular weight of MAOA comparing to those with long polymorphism and this means less catalysis of monoamine can be expected in ones with short polymorphism. Indeed, the high accumulated level of the monoamine is proved to relate to the agitation state in psychiatric patients,\[3\] and the observation might be well relating to the already mentioned effect of short versus long MAOA polymorphism. The effects of molecular mass change can be well demonstrated for case aggressive behavior and MAOA short and long polymorphism, similar to that seen in other polymorphism related medical disorders.\[4,5\]

**Financial support and sponsorship**
Nil.

**Conflicts of interest**
There are no conflicts of interest.

**Beuy Joob, Viroj Wiwanitkit**
Sanitation 1 Medical Academic Center, Bangkok, Thailand, 1Tropical Medicine, Hainan Medical University, Hainan Sheng, China

**Address for correspondence:** Dr. Beuy Joob
Sanitation 1 Medical Academic Center, Bangkok, Thailand.
E-mail: beuyjoob@hotmail.com

**REFERENCES**

1. Zhang Y, Ming QS, Yi JY, Wang X, Chai QL, Yao SQ. Gene-gene-environment interactions of serotonin transporter, monoamine oxidase a and childhood maltreatment predict aggressive behavior in Chinese adolescents. Front Behav Neurosci 2017;11:17.
2. Wagels L, Votinov M, Radke S, Clemens B, Jung S, Habel U, et al. Blunted insula activation reflects increased risk and reward seeking as an interaction of testosterone administration and the MAOA polymorphism. Hum Brain Mapp 2017;[InPress].
3. Nikolac Perkovic M, Svoj Strac D, Nedic Erjavec G, Uzun S, Podobnik J, Kozumplik O, et al. Monoamine oxidase and agitation in psychiatric patients. Prog Neuropsychopharmacol Biol Psychiatry 2016;69:131-46.
4. Joob B, Wiwanitkit V. HSD11B1 rs846908 polymorphisms and tacrolimus concentrations: Quantum chemical analysis.