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Introduction It is widely recognized that parents and peers play a critical role in the adolescent's introduction to alcohol.
Objectives The aim of the study was to examine the relationship of parental and peers drinking to adolescent drinking behavior.
Methods A cross-sectional study was carried out in four colleges and schools in Sfax in Tunisia, in May and June 2016. The sample consisted of 317 pupils, and was determined through a simple randomized sampling. These adolescents were asked to answer a self-administered questionnaire, after their consent. Alcohol use disorders identification test (AUDIT) was used to evaluate alcohol dependence.
Results The mean age was 16 years, with a sex-ratio of 1.07. The participants reported having drunk alcohol at least once in 18.9% of cases and 41.66% of them still consume. According to AUDIT, 1.6% of alcohol users presented an alcohol misuse and 21.6% presented dependence. They reported that parents' attitude toward their alcohol use was favorable in 27.11% of cases. Among dependent adolescents, the prevalence of fathers' alcohol consumption was 20% while that of friends was 70%. Adolescent drinking was significantly correlated to fathers, mothers and peers drinking ($P < 0.001$, $P = 0.004$, $P < 0.001$ respectively), mothers and peers smoking ($P = 0.05$, $P < 0.001$ respectively), fathers and peer's cannabis use ($P < 0.001$, $P < 0.001$ respectively).
Conclusion Findings suggest that negative family and peers influence increased risk of alcohol consumption in adolescents. Understanding the influences on parents' beliefs about their children's drinking and the functions of social networks in preventing alcohol consumption may be necessary to address adolescent risky drinking.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0582

Drug metabolizing enzyme and transporter genes associated with plasma risperidone level in Thai autism spectrum disorder

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Background The associations between genetic variants of drug metabolizing enzyme and transporter (DMET) genes and steady-state plasma concentrations of risperidone, 9-hydroxyrisperidone, total active-moiety, and metabolic ratio remain unclear.
Objective The objective of the present study was to present the results of the association between genetic variants of DMET gene and steady-state plasma concentration risperidone and its metabolite using Affymetrix DMET Plus genotyping microarray.
Methods Subjects eligible for this study included male and female adolescents with ASD diagnosed according to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria and being treated with risperidone for at least 4 weeks prior to the blood sample collection. Blood samples were drawn prior to the next dose of risperidone intake to determine the steady-state plasma trough concentrations of risperidone and 9-hydroxyrisperidone. Genotyping profile was obtained using the microarray. Steady-state plasma risperidone and 9-hydroxyrisperidone were measured using liquid chromatography/tandem mass spectrometry (LC-MS/MS) assay.
Results The polymorphisms of UGT2B4, CYP2D6 were highly associated with metabolic ratio. Of all the DMET analysis, ABCB11 (3084A > G, 420A > G, 368G > A, and 236G > A) and ADH7 (690G > A and −5360G > A) were found to be associated with plasma concentrations of risperidone ($P < 0.01$). In addition, 6 genetic variations among the SLC transporter family were associated with the plasma concentration of 9-hydroxyrisperidone.
Discussions This study provides a pharmacogenomic approach to investigate further among the DMET genetic variants which influence plasma concentration of risperidone. The treatment of ASD should be based on genetic factors making the challenge of psychopharmacological treatment more efficacious with lesser adverse events.
Disclosure of interest The author has not supplied his/her declaration of competing interest.

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Exome sequencing detection of genetic markers for Thai autism spectrum disorder

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Background Autism spectrum disorders (ASD) are neurodevelopmental disorders characterized by abnormalities in 3 domains; social interaction, communication/language, and restricted and repetitive behavior. The study of ASD prevalence in Thailand showed that it is approximately 9.9 children per 10,000 population for children 1–5 years old. ASD has a strong genetic basis, although the genetics of autism are complex and it is unclear. The objective of this study was to identify the genetic markers of Thai ASD.
Methods Exome sequencing was performed with twelve unrelated ASD affected individuals from twelve families. Each sample was sequenced on SOLiD 5500xl genetic analyzer, and the resulting data was processed and analyzed on LifeScope Genomic Analysis software. Exome sequencing with two additional samples was performed Ion Proton System and the data was processed on Ion Reporter server. Tertiary data analysis with all fourteen exome sequencing data were performed by using Golden Helix software. In filtering process, data were annotated to various databases including UCSC KnownGenes for non-coding and synonymous variants filter, 1000 Genomes Project for high frequency variants filter, and dbNSFP for functional prediction.
Results The genetic markers were identified for Thai ASD associated variants (c.2014G > A in EIF2AK3, c.2951G > A in FGD6, and c.6119A > G in CHD8).
Conclusions these genetic markers were the most possible of causing variants Thai. We also demonstrated a potential of exome sequencing and bioinformatics pipeline to identify the possible causative variants of ASD, which could by applied in the case that unable to identified variants by other technique.
Disclosure of interest The author has not supplied his/her declaration of competing interest.

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EW0584

Hyperuricemia and metabolic adverse effect in children and adolescents with autism spectrum disorder treated with risperidone

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