Methods: Data were available from a sample of 40 severe MDD patients defined by DSM-IV diagnostic criteria. 19 (47,5%) men and 21 (52,5%) women, ages 17–54 years was assessed through Structured Clinical Interview For DSM-IV Axis I Disorders and have a 17-item Hamilton Depression Rating Scale of 18 or higher. The 5-HTTLPR variant was genotyped according to published protocols. Three allele variants of the gene polymorphism were identified based on the PCR fragment sizes: short (S; 486bp, 14 repeats), long (L; 529bp, 16 repeats), or extra-long (XL; 612 bp, 20 repeats)
Results: The HDRS score was 21,42 ± 1,920. This study exhibits high frequency of S/S genotype (50%), lower frequency of L/S genotype (30%), L/L genotype (17,5) and L/XL (2,5%) in severe MDD patients.
Conclusions: These results support the possibility of serotonin transporter polymorphism role in the etiology of MDD.
Key Words: Severe Major Depressive Disorder, Serotonin Transporter, SLC6A4, Indonesia

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Concomitant Use of Benzodiazepine Hypnotics and Alcohol in Patients with Schizophrenia, Depression and Insomnia: A Preliminary Finding

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Abstract
Objective: Concomitant use of benzodiazepines and alcohol seems prevalent in general clinical settings; however, previous studies have not focused solely on psychiatric patients. The objectives of this study were two-fold: (1) to investigate the prevalence of concomitant use of benzodiazepine hypnotics and alcohol in outpatients with mixed psychiatric diagnoses and (2) to examine the extent of awareness on the side of their psychiatrists about the concomitant use.
Methods: A questionnaire survey was carried out for outpatients with schizophrenia, depression and primary insomnia (ICD-10) who were receiving benzodiazepine hypnotics at Kawasaki Municipal Hospital, Kanagawa, Japan. After providing informed consent, participants were asked to fill in a sleeping dairy for seven days in which use of alcohol and hypnotics was also recorded, if any. In addition, their treating psychiatrists were asked as to whether or not they thought their patients were using them concomitantly.
Results: Forty-four patients (mean±SD age = 54.9 ± 13.4 years; 19 females) were included: schizophrenia (n=16), depression (n=15) and primary insomnia (n=13). The prevalence rates of concomitant use of benzodiazepine hypnotics and alcohol were 56.3% (9/16) in schizophrenia, 33.3% (5/15) in depression and 46.2% (6/13) in primary insomnia. In contrast, the rates of suspicion regarding the concomitant use by their treating psychiatrists were 55.6% (5/9), 20.0% (1/5) and 33.3% (2/6), respectively. No differences in the severity of sleep-related symptoms were observed between concomitant users and others. In participants with depression, concomitant users tended to receive more antidepressants than nonusers (Defined Daily Dose, 1.1 vs 0.7, p=0.052) although symptom severity was not significantly different.
Conclusions: Nearly half of psychiatric patients concomitantly used benzodiazepine hypnotics and alcohol, which raises a serious safety concern. Although these preliminary findings need to be confirmed by further investigations, they emphasize the need of closer attention to those hazardous combinations.

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Association of cerebral amyloidosis, systolic blood pressure, and regional neuronal injury with late-life onset depression

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Abstract
Objective: Previous studies suggested that Alzheimer's disease (AD) process may possibly contribute to late life-onset depression (LLOD). We aimed to investigate whether LLOD is associated with cerebral amyloidosis and regional cortical atrophy, the two key brain changes in AD process, considering vascular risks together.
Methods: Twenty nine non-demented individuals who first experienced major depressive episode (MDE) after age of 60 years were recruited as LLOD subjects, and 27 non-demented elderly individuals who had no life-time experience of MDE were included as normal controls (NC). All participants received a comprehensive clinical assessment including vascular risks evaluation, magnetic resonance imaging, ^{11}C-labeled Pittsburgh Compound B (PiB) positron emission tomography and plasma beta-amyloid (Aβ) peptides level assessment.
Results: Among LLOD subjects, 48% of them had comorbid mild cognitive impairment (MCI) diagnosis, while none of NC subjects did. In VBM analysis, LLOD, irrespective of comorbid MCI diagnosis, was associated with prominent prefrontal cortical atrophy (FWE corrected $p<0.05$, $k=100$). LLOD with comorbid MCI ($LLOD_{MCI}$) subgroup showed increased cerebral PiB retention ($p=0.036$) and plasma $A\beta_{1-40}$ ($p=0.006$) and $A\beta_{1-42}$ peptides ($p=0.03$), as measures of cerebral amyloidosis, compared to NC, while overall LLOD group and LLOD without MCI ($LLOD_{woMCI}$) did not. LLOD individuals had higher systolic blood pressure (SBP) than NC subjects ($p=0.017$), particularly in subjects with $LLOD_{woMCI}$ ($p=0.026$). Multiple logistic regression analysis including diagnostic group (LLOD vs. NC) as a dependent variable showed that prefrontal cortical atrophy was significantly associated with LLOD diagnostic state ($p=0.002$), while cerebral PiB retention and SBP did not after controlling age, gender, and education.
Conclusion: Our findings suggest that AD process probably contributes to LLOD occurrence via prefrontal neuronal injury from MCI stage, while vascular process, high SBP in particular,