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(REVIEW ARTICLE)

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# Review of psychopharmacology

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#### Abstract

In India, one of the primary causes of non-fatal disease burden is psychopharmacological diseases. The prevalence of psychopharmacological disorders in India was one in seven. Despite being used to treat psychological disorders like anxiety, psychosis, depression, and mania, psychopharmacological medications can have unwanted side effects, including tardive dyskinesia, weight gain, weight loss, muscle cramps, dysphoria, gastrointestinal upset, eye problems, and issues with blood tests. We exposed numerous screening techniques for assessing psychopharmacological diseases such anxiety, depression, psychosis, and mania in the context of preclinical investigations in this study. These techniques are highly helpful for assessing psychopharmacological medications such as anti-anxiety, anti-psychotic, anti-depressant, and anti-manic medications.

**Keywords:** Preclinical research; Anti-psychotics; Antidepressants; Anti-manic medications; Psychopharmacological diseases

# 1. Introduction

The study of how medicines impact mood, perception, thinking, and behaviour is known as psychopharmacology.

Psychoactive medicines are those that work on the nervous system to produce these effects. Psychopharmacology is the umbrella word for psychology and pharmacology, two broad disciplines. As a result, psychopharmacology makes an effort to link medication activities and side effects to psychological processes. A psychopharmacologist needs to understand the nervous system's functions as well as how psychoactive medications affect them. A psychopharmacology is a scientist who researches psychoactive medicines or a medical professional who specializes in prescribing them, such as a psychiatrist.

There are many and highly comorbid mental problems, including mood, anxiety, and sleep disorders

That have been cured with herbal medicines since the dawn of time. The use of herbal remedies and complementary and alternative medicine (CAM) is common among those who suffer from anxiety and mood disorders nowadays. According to data from a nationally representative sample of 2055 adults interviewed in 1997–1998, 57% of those who experienced anxiety attacks and 54% of those who had severe depression said they had used herbal remedies and complementary and alternative medicine (CAM) treatments in the preceding 12 months to manage their disorders. There are several terms used to characterize this field than psychopharmacology. The phrase behavioural pharmacology is another. Many people confuse behavioural pharmacology with psychopharmacology, whereas some others place behavioural pharmacy in the psychology discipline of behaviour analysis. In this way, just like other stimuli in behaviour analytic models, medicines function as behavioural relevant stimuli. Another name for psychopharmacology is neuropsychopharmacology. The nervous system is represented by the prefix neuro. Despite

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the titles being similar, the field of neuropsychopharmacology has a focus on how medications affect the neurological system.

# 2. Psychopharmacology of fluoride

Despite not being a psychopharmacological agent. Because fluoride can reach the brain from medicinal or environmental sources, it may have an impact on cerebral function. remarked that the central nervous system's fluoride metabolism had not been completely and systematically investigated. However, they suggested that fluorine might directly harm the nervous system. This happened as a result of individuals with nervous system symptoms who had high body fluoride levels and whose symptoms decreased after being removed from the greater level of fluoride exposure, which led to a drop in body fluoride levels. Recent research on human chronic fluorine toxicity

#### 2.1. Absorption and entry of fluoride into the brain

Due to low levels of divalent and trivalent cations, the gastrointestinal tract absorbed about 807 mg of fluoride from the food that was consumed. A lower ph. indicated that hydrofluoric acid rather than ionic fluoride was the permeating component and that absorption from the stomach was improved. Interstitial fluid and plasma fluoride concentrations are thought to be almost comparable since fluoride is not bound by plasma proteins. Fluoride distribution in different soft tissues has been studied. Instead of being automatically controlled, as was formerly thought, plasma fluoride levels rise in direct proportion to the chronic level of fluoride intake. Plasma concentrations can be utilized as a gauge for earlier ion exposure. Fluoride is thought to be in diffusion equilibrium across cell membranes as opposed to being ionic. It was discovered that in healthy persons, the blood and cerebrospinal fluid fluoride levels were in a dynamic equilibrium, with the latter being similar to or slightly lower than the former. Once fluoride was intravenously injected into rats, a low brain tissue to plasma fluoride ratio of 0.08 was discovered and taken into consideration. The bloodbrain barrier was largely impervious to fluoride, at least temporarily.



Figure 1 Effect of Fluoride on central nervous system Acute toxicity

The "probably toxic dose" for fluoride is around 5 mg/kg, which is the lowest dose that could result in toxic signs and symptoms, including death, and should prompt right away therapeutic intervention and hospitalization. Due of fluoride's affinity for calcium, tetany, a symptom of hypocalcaemia, may result. Nervous system depression and blood coagulation impairment associated with seizures. Chronic toxicity:

Fluorosis of the skeleton and teeth is a recognized symptom of chronic fluoride poisoning. However, a condition of fluoride-induced cerebral damage has not yet been sufficiently documented to merit inclusion in reference works on organic psychiatry. In research of 68 cryolite employees, 22% of whom exhibited symptoms that were identified as having a nerve character's involvement, 84% of whom had skeletal fluorosis. Sleepiness. Indisposition. Optic neuritis was characterized as developing in conjunction with the therapeutic use of sodium fluoride as headache and giddiness. It was discovered through double-blind research that some people who consumed 1mg of fluoride daily experienced

migraine-like headaches, visual abnormalities, and depression. 97% of the 60 aluminum smelter workers tested had skeletal fluorosis, and it was shown that 23% of them had psychological disorders, including depression, mental lassitude, and memory problems.

#### 2.2. Actigraphy in human psychopharmacology

When adapted self-winding wristwatches were used to gauge children's hyperactivity in the late 1950s, the mechanical measurement of human activity was born. Since the introduction of the first wrist-worn solid state actigraphy, technological advancements have led to enhanced recording sophistication and versatility, size reduction, memory expansion, and longer monitoring device operation. Actigraphy has been employed in published work in the field of human psychopharmacology and examines research that have measured drug-induced changes in both nocturnal and diurnal behaviour using actigraphy in well-designed clinical trials.

Actigraphy and measurement of sleep: Actigraphy has been used extensively over the years to evaluate sleep/wake cycles. There are several automatic sleep/wake detection algorithms that have been created and improved, and their measurements of sleep time now match well with conventional sleep. Actigraphy has various benefits over previous methods, including lower costs, less need for technical specialists, quicker and simpler analysis, greater subject acceptance, and long-term continuous monitoring. They are constrained, nevertheless, in that they can only distinguish between sleep and wakefulness and cannot provide details on the architecture of sleep. The use of actigraphy for the assessment of sleep disorders has been thoroughly reviewed by the American Sleep Disorders Association. Actigraphy and the measurement of total sleep time, the percentage of sleep, and sleep onset latency, for the majority of normal and clinical samples, have a significant association of between 85 and 95%, according to a number of papers. The amount of sleep is the amount of time recorded as sleep while in bed. Se%, which is the test divided by the duration in bed, is a very good overall indicator of a night's sleep. The majority of the aforementioned writers have utilized their own staging algorithms rather than those in the pertinent proprietary software package, which presents a challenge in trying to evaluate the usefulness of actigraphy in assessing changes in sleep. The staging algorithms that are included with the instruments must be proven to be correct if actigraphy is to be accepted as a valuable tool in psychopharmacology



Figure 2 Actigraphy and measurement of sleep

The measurement of daytime sedation: Oversleeping during the day (EDS) is a very common disease that is linked to severe morbidity. Lack of sleep, sleep disordered breathing, sleep-wake rhythm problems, and central disorders of hypersomnolence are just a few of the reasons of EDS. EDS may also be an indication of a more serious medical or psychological condition. Psychometric tests and/or subjective questionnaires or rating scales have traditionally been used to assess how much a central nervous system (CNS) active drug affects activities of daily living during the day. However, the use of psychometric testing raises interesting methodological issues. Sedatives are a broad category of medications with a variety of modes of action that can cause central nervous system (CNS) depression. Barbiturates were used in the first half of the 20th century to treat anxiety and sleeplessness, but benzodiazepines took their position as the preferred medication in the second half of the previous century. Other sedatives, in addition to those two classes of medicines, are employed for that goal. They claimed that compared to sleeping, all daytime activities had activity levels that were noticeably different. Consequently, it has been assumed. In their investigation of the hypnotic effects and afterglow of zolpidem (10 and 20 mg).

#### 2.3. Psychopharmacological disorder

A person with more than three somatic symptoms (such as tension and irritability) and excessive concern and anxiety is said to have generalized anxiety disorder (GAD). This condition must last for at least six months before a diagnosis can be made. The largest body of research on herbal treatments for psychiatric diseases is focused on anxiety, with the majority of individual phytomedicines having demonstrated anxiolytic efficacy. The most common mental illnesses in the US are anxiety disorders. The ability to recognize and treat anxiety disorders, such as panic disorder, social phobia, obsessive-compulsive disorder, generalized anxiety disorder (gad), and posttraumatic stress disorder, has significantly improved over the past three decades. It is now understood that untreated patients with these frequent, typically chronic illnesses are severely disabled. The key developments in the pharmaceutical treatment of anxiety disorders are highlighted in this overview. Evidence of the effectiveness of different pharmaceuticals. And a discussion of the drawbacks of drug therapy. Psychopharmacological research is discussed in relation to certain crucial clinical issues that are still open.

The most prevalent category of psychiatric illnesses is anxiety disorders, which are frequently linked to functional impairment and are among the primary causes of disability and time away from work, along with depression. Due to their high frequency and the higher costs of medical and psychiatric care, anxiety disorders may also have a bigger financial impact than any other mental diseases, including mood disorders.

# 3. Major depressive disorder

Major depressive disorder (MDD), sometimes known as "unipolar depression," is characterized by depressed mood or decreased interest or pleasure with at least four other symptoms (such as changes in weight or appetite). Change, exhaustion, psychomotor agitation, sleep disorders such insomnia or hypersomnia, lack of concentration, suicidal thoughts, and feelings of worthlessness. According to the World Health Organization (WHO), the number of persons with MDD (including dysthymia) is currently estimated to be 322 million, an increase of more than 18% over the ten years since 2005, making it the biggest cause of illness and disability globally (WHO, 2017). While there was no significant difference between the saffron prescription and the antidepressant groups, a significant result with a large effect size was found in Favor of saffron treatment versus placebo control in treating depressive symptoms, indicating that both treatments were equally effective in reducing depression symptoms.

# 4. Ethnic and cultural factors in psychopharmacology

#### 4.1. General issues

Discussion and argument over ethnicity definitions and group classifications is intense. Definitions of ethnicity might be based on social structures, personal identities, or—as is frequently the case in medical practice and research—on a level that emphasizes phenotypic commonalities rather than genotypic distinctions. Ethno-pharmacogenetic variants become clinically significant when they are linked to phenotypic similarities and pharmacogenetic variability in drug metabolism.

#### 4.2. Personality factors

Single ethnic group, various social and culturally distinct behaviours, beliefs and social settings are important. Personality is melded by social and cultural factors. Ethnicity and culture might indirectly influence personality traits, which in turn might influence an individual's response to medication.

#### 4.3. Biological factors

Several enzymes are involved in the metabolism of drugs and other foreign substances, and these enzymes' activity differ significantly between individuals and ethnic groups for both hereditary and environmental factors. Although there are significant disparities between individuals and between ethnic groups, it is less known what causes these variations.

#### 4.4. Environmental factors

Pharmacogenetics and related pharmacokinetics of psychiatric medicines can be impacted by environmental factors. A differential response that is related with ethnicity can result from changes of the metabolism if certain cultural and racial groups are exposed to certain environmental conditions over an extended period of time. The prognosis and result of psychiatric treatment can be influenced by social and family support on a general level, as well as by individual

aspects including how one responds to stressors. People who are exposed to more stressors and who also have low levels of social support and tolerance are more likely to experience mental illness as well as poor social and clinical outcomes.

# 5. Conclusion

Pathological emotional memory can be prevented or removed with the therapeutic use of psychopharmacology. The capacity for normal development, storage, and retrieval of working and semantic memory can also be improved non-therapeutically. There would seem to be some proof that long-term exposure to fluoride may cause some people to experience cognitive impairment, namely memory and focus problems. These signs and symptoms are similar to those of chronic fatigue syndrome. The evidence is currently suggestive rather than conclusive. There are numerous potential mechanisms that could mediate such effects. Further research should be done on this connection between fluoride and psychiatric symptomatology. Both subjective and objective (psychometric) assessments of sleep and sedation frequently reflect the effects on activity. The reporting of actigraphy results, however, may be skewed in favour of good results, so two cautionary points must be made. Actigraphy is infrequently the major variable, and it's likely that many more studies have employed it but failed to submit the results since there was no quantifiable effect. This must, however, remain a speculation owing to the nature of the study. It is important to support the actigraphy community in creating such approaches and defining variables. The use of actigraphy in future carefully planned studies examining the effects on the central nervous system of drugs on both sleep and daytime sedation, however, is seen to be justified by the author due to the substantial evidence of utility shown in this study.

# **Compliance with ethical standards**

#### Disclosure of conflict of interest

No conflict of interest to be disclosed.

#### References

- [1] Introduction to Psychopharmacology, Drugs and the Neuroscience of Behaviour. Sage publications, 1-29, 2018.
- [2] Kessler, R.C., Chiu, W.T., Demler, O., Merikangas, K.R., Walters, E.E., 2005. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch. Gen. Psychiatry 62, 617– 627.
- [3] Kessler, R.C., Soukup, J., Davis, R.B., Foster, D.F., Wilkey, S.A., Van Rompay, M.M., Eisenberg, D.M., 2001. The use of complementary and alternative therapies to treat anxiety and depression in the United States. Am. J. Psychiatry 158, 289–294.
- [4] Elkins, G., Rajab, M.H., Marcus, J., 2005. Complementary and alternative medicine use by psychiatric inpatients. Psychol. Rep. 96, 163–166
- [5] Yu-huan H and Si-shung W (1988) Fluoride in cerebrospinal fluid of patients with fluorosis . Journal of Neurdlogy, Neurosurgery and P s\_tchiatr,-, 51, l 59 l 1 593.
- [6] Anand JK and Roberts JT (1990) Chronic fluorine poisoning in man: a review of literature in English (1946-1989) and indications for research. Biomedicine and P harmac ot he rap y, 44, 417-420.
- [7] Whitford GM, Pashley DH and Reynolds KE (1979) Fluoride tissue distribution: short term kinetics. American Journal of physiology, 236. Fl 4 I -Fl 48.
- [8] Yu-huan H and Si-shung W (1988) Fluoride in cerebrospinal fluid of patients with fluorosis . Journal of Neurdlogy, Neurosurgery and P s\_tchiatr,-, 51, l 59 l 1 593.
- [9] Waldbott GL (1979) Preskeletal fluorosis near an Ohio enamel factory: a preliminary report. Veterinary and Human Toxicology,2l,4-8.
- [10] Geeraerts F, Gijs G, Finne E and Crokaert R (1986) Kenetics [sic] of fluoride penetration in liver and brain. Fluoride,19. 108-r r2
- [11] Whitford GM (1990) The physiological and toxicological characteristics of fluoride. Journal of Dental Research,69. 539-549.

- [12] Baltazar RF, Mower MM, Reider R, Funk M anc Salomon J (1980) Acute fluoride poisoning leading to fatal hyperkalemia. C he s t. 78. 660 663.
- [13] Singh A, Jolly SS. Bansal BC and Marhur CC (1963) Endemic fluorosis: epidemiological, clinical and biochemical study of chronic fluorine intoxication in Panjab (India). ,Vedicine,42, 229-246.
- [14] Lishman WA (1987) Organic Psychiarm: the Psychological Consequences of Cerebral Disorder, 2nd edn. Blackwell Scientific Publications, Oxford.
- [15] Geall MG and Beilin LI (1964) Sodium fluoride and optic neuritis. Britrs& Medical Journal, 2, 355-356
- [16] Grimbergen GW (1974) A double-btind test for determination intolerance to fluoridated water (preliminary report). Fluoride,7,146-152
- [17] Czerwinski E and Lankosz W (1977) Fluoride-induced changes in 60 retired aluminium workers. Fluoride.l0. 125-136.
- [18] Waldbott GL (1979) Preskeletal fluorosis near an Ohio enamel factory: a preliminary report. Veterinary and Human Toxicology,2l,4-8.
- [19] Colburn TR, Smith BM, Guarini JJ, Simmons NN. 1976. An ambulatory activity monitor with solid state memory. Instrument Soc Am Trans 15: 149–154.
- [20] Kripke DF, Mullaney DJ, Messin S, Wyborney VG. 1978. Wrist actigraphic measures of sleep and rhythms. Electroenceph Clin Neurophysiol 44: 674–676.
- [21] Stanley N, Hindmarch I. 1997. Actigraphy can measure antidepressant induced daytime sedation in healthy volunteers. Hum Psychopharmacology 12: 437–443.
- [22] Kripke DF, Mullaney DJ, Messin S, Wyborney VG. 1978. Wrist actigraphic measures of sleep and rhythms. Electroenceph Clin Neurophysiol 44: 674–676.
- [23] Mullaney DJ, Kripke DF, Messin S. 1980. Wrist-actigraphic estimation of sleep time. Sleep 3: 83–92.
- [24] Sadeh A, Hauri PJ, Kripke DF, Lavie P. 1995. The role of actigraphy in the evaluation of sleep disorders. Sleep 18: 288–302.
- [25] Stanley N, Dorling MC, Dawson J, Hindmarch I. 2000. The accuracy of Mini-Motionlogger and Actiwatch in the identification of sleep as compared to sleep EEG. Sleep 23(Suppl. 2): A386.
- [26] Newman J, Stampi C, Dunham DW, Broughton R. 1988. Does wrist-actigraphy approximate traditional polysomnographic detection of sleep and wakefulness in narcolepsy-cataplexy. Sleep Res 17: 343.
- [27] Stanley N, Dorling MC, Dawson J, Hindmarch I. 2000. The accuracy of Mini-Motionlogger and Actiwatch in the identification of sleep as compared to sleep EEG. Sleep 23(Suppl. 2): A386.
- [28] Kecklund G, A° kerstadt T, Sigurdson K. 1991. Actigraphy and subjective sleep quality. Sleep Res 20A: 499.
- [29] Stanley N, Hindmarch I. 1997. Actigraphy can measure antidepressant induced daytime sedation in healthy volunteers. Hum Psychopharmacology 12: 437–443.
- [30] Borbe'ly AA, Youmbi-Balderer G, Jaggi-Schwarz K. 1988. Zolpidem (10 and 20 mg): hypnotic action and residual effects after a single bedtime dose. In Imidazopyridines in Sleep Disorders, Sauvanet JP, Langer SZ, Morselli PL (eds). Raven Press: New York; 205–210.
- [31] Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE: Prevalence, severity, and comorbidity of-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005; 62:617–627
- [32] Iancu SC, Batelaan NM, Zweekhorst MB, Bunders JF, Veltman DJ, Penninx BW, van Balkom AJ: Trajectories of functioning after remission from anxiety disorders: 2-year course and outcome predictors. Psychol Med 2014; 44:593–605
- [33] Ahola K, Virtanen M, Honkonen T, Isometsä E, Aromaa A, Lönnqvist J: Common mental disorders and subsequent work disability: a population based Health 2000 Study. J Affect Disord 2011; 134:365–372
- [34] Griebel G, Holmes A: 50 years of hurdles and hope in anxiolytic drug discovery. Nat Rev Drug Discov 2013; 12:667–6879. Celada P, Bortolozzi A, Artigas F: Serotonin 5-HT1A receptors as targets for agents to treat psychiatric disorders: rationale and current status of research. CNS Drugs 2013; 27:703–716

- [35] Lewis, P., Rack, P., Vaddadi, K., et al (1980) Ethnic differences in drug response. Postgraduate Medical Journal, 56, 46--49.
- [36] Lin, K.-M. (1996) Psychopharmacology in cross-cultural psychiatry. Mount Sinai Journal of Medicine, 63, 283-284.
- [37] Lin, K.-M. (1996) Psychopharmacology in cross-cultural psychiatry. Mount Sinai Journal of Medicine, 63, 283-284.
- [38] Smith, M. & Mendoza, R. (1996) Ethnicity and pharmacogenetics. Mount Sinai Journal of Medicine, 63, 285-290. Westermeyer, J. (1989) Psychiatric Care of Migrants. Washington, DC: APA Press.
- [39] Lin, K.-M. (1996) Psychopharmacology in cross-cultural psychiatry. Mount Sinai Journal of Medicine, 63, 283-284. Lau, J., Smith, R., et al (1988) Comparison of alprazolam
- [40] Dawkins, K. (1996) The interaction of ethnicity, socio-cultural factors and gender in clinical psychopharmacology. Psychopharmacology Bulletin, 32, 283-289