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DRUGS ACTING ON CENTRAL NERVOUS SYSTEM: A REVIEW

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ABSTRACT

Central nervous system is branch which deals with study of various drugs which affect on human brain regarding with its mood, behaviourand other things. It includes various drugs and their classes like antidepressants, antipsychotic, etc. It mainly depends on neurotransmitters in the brain if their function is disturbed then all things goes wrong. Psychopharmacology is the scientific studies of the effects of drugs have on mood, sensation, thinking, and behavior. It is distinguished from neuropsychopharmacology, which emphasizes the correlation between drug-induced changes in the functioning of cells in the nervous system and changes in consciousness and behavior. The field of psychopharmacology studies a wide range of substances with various types of psychoactive properties, focusing primarily on the chemical interactions with the brain. There may be 2 possibilities either neurotransmitter increased levels or decreased level in the brain and when both this things go out of their limit problem arises. In case of antipsychotic increased dopamine level is found while in antidepressant there is decrease in serotonin level are found in both cases problem may arises due to inappropriate balance of neurotransmitter.

INTRODUCTION:

Psychoactive drugs interact with particular target sites or receptors found in the nervous system to induce widespread changes in physiological or psychological functions. The specific interaction between drugs and their receptors is referred to as "drug action" The widespread change in physiological or psychological function is referred to as "drug effect". These drugs may originate from natural sources such as plants and animals, or from artificial sources such as chemical synthesis in the laboratory

Not often mentioned or included in the field of psychopharmacology today, are psychoactive substances not identified as useful in modern mental health settings orreferences. These substances are naturally occurring, but nonetheless psychoactive, and are compounds identified through the work of ethnobotanists and ethnomycologists (and others who study the native use of naturally occurring psychoactive drugs).

However, although these substances have been used throughout history by various cultures, and have a profound effect on mentality and brain function, they have not always attained the degree of scrutinous evaluation that lab-made compounds have. Nevertheless, some, such as psilocybin and mescaline, have provided a basis of study for the compounds that are used and examined in the field today. Hunter-gatherer societies tended tofavour hallucinogens, and today their use can still be observed in many surviving tribal cultures.The exact drug used depends on what the particular ecosystem in given tribe lives in can support, and are typically found growing wild. Such drugs include various psychoactive mushrooms containing psilocybin or muscimol and cacti containing mescaline and other chemicals, along with myriad other psychoactive-chemical-containing plants. These societies generally attach spiritual significance to such drug use, and often incorporate it into their religious practices. With the dawn of the Neolithic and the proliferation of agriculture, new psychoactives came into use as a natural by-product of farming. Among them were opium, cannabis, and alcohol derived from the fermentation of cereals and fruits. Most societies began developing harbours, lists of herbs which were good for treating various physical and mental ailments. For example, St. John's Wort was traditionally prescribed in parts of Europe for depression (in addition to use as a general-purpose tea), and Chinese

medicine developed elaborate lists of herbs and preparations. These and various other substances that have an effect on the brain are still used as remedies in many cultures.

Neurotransmitters

Psychoactive drugs exert their sensory and behavioural effects almost entirely by acting on neurotransmitters and by modifying one or more aspects of synaptic transmission. Neurotransmitters can be viewed as chemicals through which neurons primarily communicate; psychoactive drugs affect the mind by altering this communication.

Drugs may act by serving as a precursor for the neurotransmitter;

1. Inhibiting neurotransmitter synthesis;
2. Preventing storage of neurotransmitter in the presynaptic vesicle;
3. Stimulating or inhibiting neurotransmitter release
4. Stimulating or blocking post-synaptic receptors;
5. Stimulating auto receptors, inhibiting neurotransmitter release;
6. Blocking auto receptors, increasing neurotransmitter release
7. Inhibiting neurotransmission breakdown;
8. Blocking neurotransmitter reuptake by the presynaptic neuron

Hormones

The other central method through which drugs act is by affecting communications between cells through hormones. Neurotransmitters can usually only travel a microscopic distance before reaching their target at the other side of the synaptic cleft, while hormones can travel long distances before reaching target cells anywhere in the body. Thus, the endocrine system is a critical focus of psychopharmacology because;

1. Drugs can alter the secretion of many hormones
2. Hormones may alter the behavioral responses to drugs
3. Hormones themselves sometimes have psychoactive properties; and
4. The secretion of some hormones, especially those dependent on the pituitary gland, is controlled by neurotransmitter systems in the brain.

Psychopharmacological substances

Alcohol

Alcohol is mainly act as depressant it acts on GABA receptors which mainly act as depressant. Its effect may vary according to dose at low dose patient feel relaxed at moderate dose patient act drowsy but at higher dose patient feel very confident with reckless behaviour.

Alcohol mainly act on 2 receptor

1. GABA (Gamma Amino Butyric Acid)
2. NMDA

Alcohol give depressant effect on NMDA receptor and modulators effect on GABA receptor.

Alcohol shows paradoxical dis-inhibition. i.e. inhibition of neurotransmitter.

It initially acts on GABA receptor so we get excitory action then acts on 2nd receptor and gives depressant action.

Ethanol:

Alcohol is hydroxyl derivative of aliphatic hydrocarbons. When unqualified, Alcohol refers to ethyl alcohol or ethanol. Pharmacology of alcohol is important for its presence in beverages (which have been used since record history), alcoholism and for alcohol intoxication, rather than a medicinal substance.

Fermentation reaction for manufacturing of alcohol by sugars -

$C_6H_{12}O_6 \rightarrow 2CO_2 + 2C_2H_5OH$

Various actions

CNS– It gives dose dependent depression It shows paradoxical dis-inhibition.

GIT – It increases appetite Increases gastric acid secretion Causes gastritis and aggregate peptic ulcer

CVS- Cutaneous vasodilation and give flushing action.

Metabolism

Ethanol -----→ acetaldehyde -------→ acetic acid

Initially it catalysed by alcohol dehydrogenase then with aldehyde dehydrogenase.

Alcohol is depressant, the impacts of which may differ as per measurement sum, recurrence, and chronicity. As an individual from the soothing trancelike class, at the least dosages, the

individual feels casual and less restless. In calm settings, the client may feel lazy, however in settings with expanded tactile incitement, people may feel uninhibited and more sure.

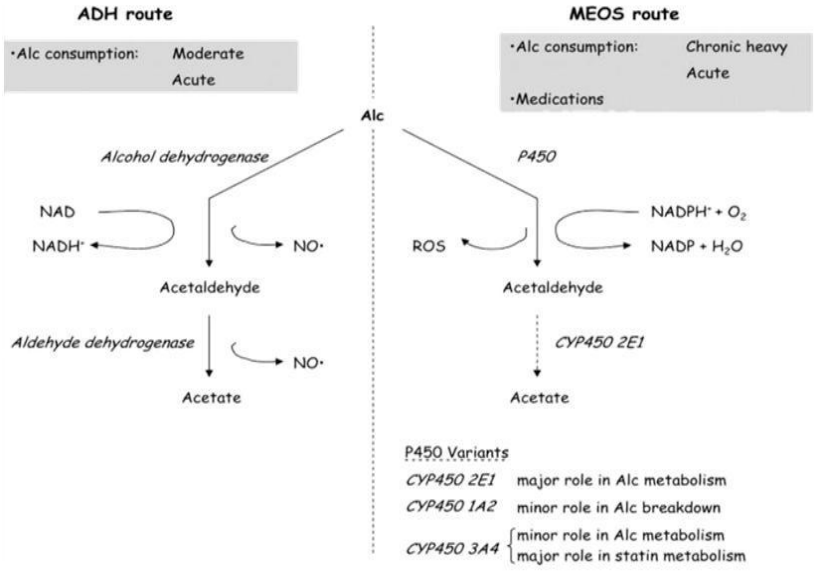

Fig. 1: Metabolism of alcohol

High dosages of liquor quickly expended may deliver amnesia for the occasions that happen amid inebriation. Different impacts incorporate decreased coordination, which prompts slurred discourse, impeded fine-engine abilities, and postponed response time. The impacts of liquor on the body's neurochemistry are harder to analyze than some different medications. This is on the grounds that the concoction idea of the substance makes it simple to infiltrate into the cerebrum, and it additionally impacts the phospholipid bilayer of neurons. This enables liquor to widespread affect numerous ordinary cell works and alters the activities of a few neurotransmitter frameworks. Liquor hinders glutamate (a noteworthy excitatory neurotransmitter in the sensory system) neurotransmission by decreasing the adequacy at the NMDA receptor, which is identified with memory misfortune related with inebriation. It additionally adjusts the capacity of GABA, a noteworthy inhibitory amino corrosive neurotransmitter. The fortifying characteristics of liquor prompting rehashed utilize – and in this manner additionally the instruments of withdrawal from constant liquor utilize – are mostly

because of the substance's activity on the dopamine framework. This is additionally because of liquor's impact on the opioid frameworks, or endorphins, that have sedative like impact

Therapeutic uses

Alcohol

Has been advocated by many for use as an appetite stimulant, as an antifoaming agent to treat pulmonary edema, as a regular soporific medication, and as ready source of intravenous calories. None of these uses has had much proven efficacy. A "hot toddy" has little effect on upper respiratory infections, although popular belief has claimed that at the first sign of coldness should has on the bed -Post.

Antidepressant

Antidepressants reduce symptoms of mood disorders primarily through the regulation of norepinephrine and serotonin (particularly the 5-HT receptors). After chronic use, neurons adapt to the change in biochemistry, resulting in a change in pre- and postsynaptic receptor density and second messenger function.

Monoamine oxidase inhibitors (MAOIs) are the oldest class of antidepressants. They inhibit monoamine oxidase, the enzyme that metabolizes the monoamine neurotransmitters in the presynaptic terminals that are not contained in protective synaptic vesicles. The inhibition of the enzyme increases the amount of neurotransmitter available for release. It increases norepinephrine, dopamine, and 5-HT and thus increases the action of the transmitters at their receptors. MAOIs have been somewhat disfavoured because of their reputation for more serious side effects.

Tricyclic antidepressants (TCAs) work through binding to the presynaptic transporter proteins and blocking the reuptake of norepinephrine or 5-HT into the presynaptic terminal, prolonging the duration of transmitter action at the synapse.

Depression usually leads to

1. Difficulty getting to sleep
2. Poor quality sleep
3. Fewer Hours of sleep
4. More awakenings during the night

5. In severe cases, waking very early in the morning and
6. Being unable to get back to sleep
7. Daytime tiredness

Selective serotonin reuptake inhibitors (SSRIs) selectively block the reuptake of serotonin (5-HT) through their inhibiting effects on the sodium/potassium ATP-dependent serotonin transporter in presynaptic neurons. This increases the availability of 5-HT in the synaptic cleft.[6] The main parameters to consider in choosing an antidepressant are side effects and safety. Most SSRIs are available generically and are relatively inexpensive. Older antidepressants, such as the TCAs and MAOIs usually require more visits and monitoring, and this may offset the low expense of the drugs. The SSRIs are relatively safe in overdose and better tolerated than the TCAs and MAOIs for most patients.[9]

Side effect of antidepressant

Antidepressants can sometimes cause a wide range of unpleasant side effects, including:

1. Nausea
2. Increased appetite and weight gain
3. Loss of sexual desire and other sexual problems, such as erectile dysfunction and decreased orgasm
4. Fatigue and drowsiness
5. Insomnia
6. Dry mouth
7. Blurred vision
8. Constipation
9. Dizziness
10. Agitation
11. Irritability
12. Anxiety

Antipsychotics

Antipsychotics, also known as neuroleptics or major tranquilizers, are a class of medication primarily used to manage psychosis, principally in schizophrenia and bipolar disorder

Dopamine theory of schizophrenia

Some researchers have suggested that dopamine systems in the mesolimbic pathway may contribute to the 'positive symptoms' of schizophrenia (whereas problems with dopamine function in the mesocortical pathway may be responsible for the 'negative symptoms', such as abolition and alogia). Abnormal expression, thus distribution of the D2 receptor between these areas and the rest of the brain may also be implicated in schizophrenia, specifically in the acute phase. A relative excess of these receptors within the limbic system means Broca's area, which can produce illogical language, has an abnormal connection to Wernicke's area, which comprehends language but does not create it. Note that variation in distribution is observed within individuals, so abnormalities of this characteristic likely play a significant role in all psychological illnesses.

Individual alterations are produced by differences within glutamatergic pathways within the limbic system, which are also implicated in other psychotic syndromes. Among the alterations of both synaptic and global structure, the most significant abnormalities are observed in the uncinate fasciculus and the cingulated cortex.. The combination of these creates a profound dissymmetry of prefrontal inhibitory signalling, shifted positively towards the dominant side. Eventually, the cingulated gyrus becomes atrophied towards the anterior,due to long-Term Depression (LTD) and Long-Term Potentiation (LTP) from the abnormally strong signals transversely across the brain.] This, combined with a relative deficit in GABAergic input to Wernicke's area, shifts the balance of bilateral communication across the corpus callosum posteriorly. Through this mechanism, hemispherical communication becomes highly shifted towards the left/dominant posterior. As such, spontaneous language from Broca's can propagate through the limbic system to the tertiary auditory cortex. This

retrograde signalling to the temporal lobes that results in the parietal lobes not recognizing it as internal results in the auditory hallucinations typical of chronic schizophrenia.

In addition, significant cortical grey matter volume reductions are observed in this disorder.Specifically, the right hemisphere atrophies more, while both sides show a marked decrease in frontal and posterior volume. This indicates that abnormal synaptic plasticity occurs, where certain feedback loops become so potentiated, others receive little glutaminergic

transmission. This is a direct result of the abnormal dopaminergic input to the striatum, thus (indirectly) disinhibition of thalamic activity. The excitatory nature of dopaminergic transmission means the glutamate hypothesis of schizophrenia is inextricably intertwined with this altered functioning. 5-HT also regulates monoamine neurotransmitters, including dopaminergic transmission. Specifically, the 5-HT2A receptor regulates cortical input to the basal ganglia and many typical and atypical antipsychotics are antagonists at this receptor. Several antipsychotics are also antagonists at the 5-HT2C receptor, leading to dopamine release in the structures where 5-HT2C is expressed; striatum, prefrontal cortex, nucleus accumbens, amygdala, hippocampus (all structures indicated in this disease), and currently thought to be a reason why antipsychotics with 5HT2C antagonistic properties improves negative symptoms. More research is needed to explain the exact nature of the altered chemical transmission in this disorder.

Recent evidence on a variety of animal models of psychosis, such as sensitization of animal behaviour by amphetamine, or phencyclidine (PCP, Angel Dust), or excess steroids[citation needed], or by removing various genes (COMT, DBH, GPRK6, RGS9, RIIbeta), or making brain lesions in new-born animals, or delivering animals abnormally by Caesarian section, all induce a marked behavioural supersensitivity to dopamine and a marked rise in the number of dopamine D2 receptors in the high-affinity state for dopamine. This latter work implies that there are multiple genes and neuronal pathways that can lead to psychosis and that all these multiple psychosis pathways converge via the high-affinity state of the D2 receptor, the common target for all antipsychotics, typical or atypical. Combined with less inhibitory signalling from the thalamus and other basal ganglic structures, from hypotrophy the abnormal activation of the cingulate cortex, specifically around Broca's and Wernicke's areas, abnormal D2 agonism can facilitate the self-reinforcing, illogical patterns of language found in such patients. In schizophrenia, this feedback loop has progressed, which produced the widespread neural atrophy characteristic of this disease. Patients on neuroleptic or antipsychotic medication have significantly less atrophy within these crucial areas. As such, early medical intervention is crucial in preventing the advancement of these profound deficits in bilateral communication at the root of all psychotic disorders. Advanced, chronic schizophrenia cannot respond even to

clozapine, regarded as the most potent antipsychotic, as such, a cure for highly advanced schizophrenia is likely impossible, so the value of early intervention cannot be stressed enough.

All proven antipsychotics are postsynaptic dopamine receptor blockers (dopamine antagonists). For an antipsychotic to be effective, it generally requires a dopamine antagonism of 60%-80% of dopamine D2 receptors.

First generation (typical) antipsychotics:

Traditional neuroleptics modify several neurotransmitter systems, but their clinical effectiveness is most likely due to their ability to antagonize dopamine transmission by competitively blocking the receptors or by inhibiting dopamine release. The most serious and troublesome side effects of these classical antipsychotics are movement disorders that resemble the symptoms of Parkinson's disease, Because the neuroleptics antagonize dopamine receptors broadly, also reducing the normal dopamine-mediated inhibition of cholinergic cells in the striatum.

Second-generation (atypical) antipsychotics:

The concept of "a typicality" is from the finding that the second generation antipsychotics (SGAs) had a greater serotonin/dopamine ratio than did earlier drugs, and might be associated with improved efficacy (particularly for the negative symptoms of psychosis) and reduced extrapyramidal side effects. Some of the efficacy of atypical antipsychotics may be due to 5-HT2 antagonism or the blockade of other dopamine receptors. Agents that purely block 5-HT2 or dopamine receptors other than D2 have often failed as effective antipsychotics.

Side effects

Certain antipsychotic drugs cause significant weight gain and high cholesterol levels, and they may increase the risk of diabetes. People considering an antipsychotic for bipolar disorder should be screened for their risk of heart disease, stroke, and diabetes, according to a study published in Diabetes Care.

Common side effects of antipsychotic medications include:

1. Blurred vision
2. Dry mouth
3. Drowsiness
4. Muscle spasms or tremors

5. Weight gain
6. Tardative dyskinesia
7. Bradykinesia
8. Malignant hyperthermia
9. Hyperprolactemia
10. Neuroleptics Malignant syndrome

Benzodiazepines

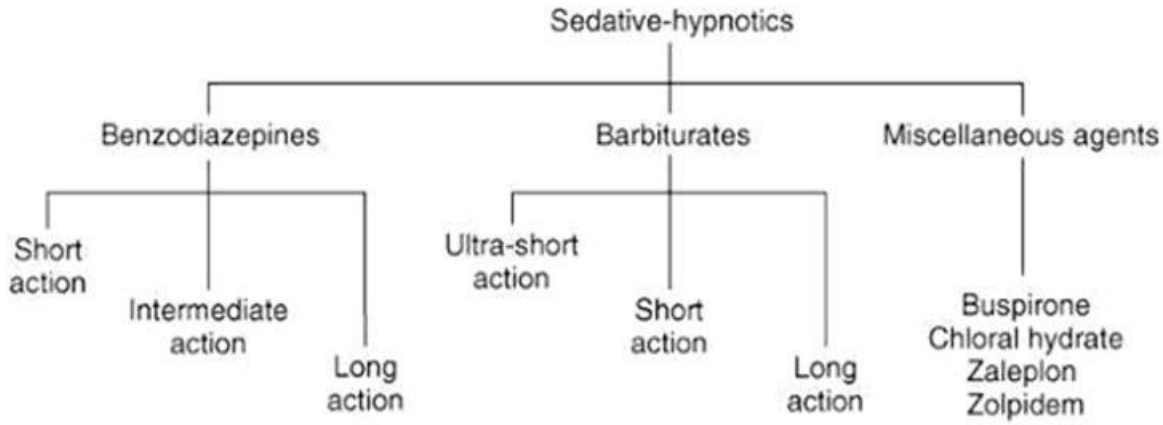

Fig. 2: Classification of Sedative-hypnotics

Sleep

The standard figure given for the average length of the sleep cycle in an adult man is 90 minutes. The sleep stages one of the important key factors to a person's health. N1 (stage 1) is when the person is drowsy or awake to falling asleep. Brain waves and muscle activity start to decrease at this stage. N1 (stage 2) is when the person experiences a light sleep which eye movement have stopped by this time. The brain waves and muscle have decreased more and become slow and steady. The heart rate and body temperature goes down. N3 (stage 3 or even 4) is the most difficult stage to be awakened. Every part of the body is now relaxed such as the person's breathing, blood pressure, and the temperature of the body. The National Sleep Foundation discusses the different stages of NREM and the importance of them. They quote for REM sleep that," A unique state, in which dreams usually occur. The brain is awake and body paralyzed." This unique stage is usually when the person is in the deepest stage of sleep and dreams. This

figure was popularized by Nathaniel Kleitman around 1963.] Other sources give 90–110 minutes1] or 80–120 minutes.

The cycle can be defined as lasting from the end of one REM period to the end of the next,] or from the beginning of REM, or from the beginning of non-REM stage 2. (The decision of how to mark the periods makes a difference for research purposes because of the unavoidable inclusion or exclusion of the night's first NREM or its final REM phase if directly preceding awakening.)

A 7–8-hour sleep probably includes five cycles, the middle two of which tend to be longer.REM takes up more of the cycle as the night goes on.

Benzodiazepines are often used to reduce anxiety symptoms, muscle tension, seizure disorders, insomnia, symptoms of alcohol withdrawal, and panic attack symptoms. Their action is primarily on specific benzodiazepine sites on the GABAA receptor. This receptor complex is thought to mediate the anxiolytic, sedative, and anticonvulsant actions of the benzodiazepines. Use of benzodiazepines carries the risk of tolerance (necessitating increased dosage), dependence, and abuse. Taking these drugs for a long period of time can lead to withdrawal symptoms upon abrupt discontinuation. Hallucinogens cause perceptual and cognitive distortions without delirium. The state of intoxication is often called a "trip". Onset is the first stage after an individual ingests (LSD, psilocybin, or mescaline) or smokes (dimethyltryptamine) the substance. This stage may consist of visual effects, with an intensification of colours and the appearance of geometric patterns that can be seen with one's eyes closed. This is followed by a plateau phase, where the subjective sense of time begins to slow and the visual effects increase in intensity. The user may experience synesthesia, a crossing-over of sensations (for example, one may "see" sounds and "hear" colours). In addition to the sensory-perceptual effects, hallucinogenic substances may induce feelings of depersonalization, emotional shifts to a euphoric or anxious/fearful state, and a disruption of logical thought. Hallucinogens are classified chemically as either indoleamines (specifically tryptamines), sharing a common structure with serotonin, or as phenethylamines, which share a common structure with norepinephrine. Both classes of these drugs are agonists at the 5-HT2 receptors; this is thought to be the central component of their hallucinogenic properties. Activation of 5-HT2A may be

particularly important for hallucinogenic activity. However, repeated exposure to hallucinogens leads to rapid tolerance, likely through down-regulation of these receptors in specific target cells.

Hypnotics

Hypnotics are drugs that are used to help people fall asleep. There are many types of hypnotic drugs, and doctors recommend and prescribe them based on the type of sleep problem you have. Lifestyle and sleep habit changes also are effective for treating certain types of sleep problems. If you have chronic insomnia or problems sleeping contact your doctor or other health care professional. Doctors specializing in sleep problems (sleep medicine) can help you determine the cause of your insomnia and treat it.

Hypnotic drugs also called sleep aids, sleeping pills, or soporifics.

Uses of hypnotics

Doctors prescribe hypnotics to treat insomnia. Insomnia is a sleep problem that may involve difficulty falling or staying asleep. Inadequate sleep affects mood, energy levels, health, and work performance.

Common causes of insomnia are stress, traumatic events, depression, anxiety, and medications. If you know the cause of your insomnia and treat it, it may reduce the need for sleep medications to aid or induce sleep.

Hypnotic side effects

Side effects of hypnotics depend upon the type of hypnotic used.

Some common side effects of some hypnotics include:

1. Headache
2. Nausea
3. Short-term forgetfulness
4. Rebound insomnia
5. Dry mouth
6. Hallucinations
7. Dizziness
8. Drowsiness

Withdrawal symptoms (for example, anxiety, or insomnia)

a. Unpleasant taste
b. Confusion
c. Dependence

Other side effects of hypnotics may include:

1. Dizziness
2. Cough
3. Abnormal dreams
4. Diarrheal
5. Upper respiratory infections
6. Stomach upset
7. Loss of coordination
8. Sleepiness
9. Hair loss
10. Dry skin
11. Loss of appetite
12. Nausea
13. Fatigue

Possible serious side effects may include:

1. Sleep paralysis
2. Behavioural changes
3. Abnormal thinking
4. Suicidal thinking
5. Worsening of depression
6. Sleep driving and other complex behaviours
7. Anaemia
8. Hives
9. Exfoliate dermatitis

Benzodiazepines are addictive hypnotics and are federally controlled substances. People can develop a physical dependence after several days of taking them, and the risk is higher during long-term use.

Doctors should take care when prescribing benzodiazepine drugs to people with a history of drug abuse or alcoholism because they are more likely to become addicted to benzodiazepines.

Can I drink alcohol if I'm taking a hypnotic drug

Do not drink alcohol before are after taking a sleep medication because drinking alcohol while using a sleep medication or sedative may lead to severe drowsiness.

OTC (over-the-counter) hypnotics

Diphenhydramine (for example, Benadryl) and doxylamine (for example, Unisom) are over-the-counter (OTC) drugs that can help people fall asleep. These drugs are antihistamines that cause drowsiness and sedation. Only use these medications for a few days. Talk with your doctor or other health care professional if you have insomnia for more than a couple of weeks.

You should read the instructions and warnings before taking OTC sleep medicine because they can have serious adverse effects if not used properly. They also may interfere with the action of other drugs.

Side effects reported by people taking diphenhydramine or doxylamine include:

1. Dry mouth
2. Blurred vision
3. Constipation
4. Grogginess
5. Dizziness

Natural, herbal hypnotics and hypnotic supplements

Synthetic melatonin capsules, pills, or tablets may help people fall asleep. Melatonin is most often used for treating jet lag. Melatonin takes few days, up to a few weeks to work when it is used for treating sleep problems.

Valerian is another supplement available to treat insomnia.

Types of hypnotics (hypnotic drugs)

The FDA has approved five types of hypnotic medications for the treatment of sleep problems, which include:

1. Benzodiazepines
2. No benzodiazepine receptor agonists
3. Melatonin receptor agonists
4. Orexin receptor agonist
5. Antidepressants

These medications are all effective for treating sleep problems, but they work in different ways. Some sleep medicines only last a few hours (short-acting medications) while others last longer in the body (long-acting medications). Doctors and other health care professionals choose sleep medications based on the type of sleep problem you have. For example, people who have trouble falling asleep will benefit from a short-acting sleep medicine. People who have trouble staying asleep will benefit more from long-acting sleep drugs.

List and examples of brand and generic names of hypnotic drugs

There are over 40 different sedative/hypnotic drugs available. Talk to your doctor, pharmacist, or other health care professional for any addition information about the generic forms available.

Melatonin receptor agonists

1. Ramelteon (Rozerem)
2. Prexin receptor antagonist
3. Suvorexant (Belsomra)
4. Benzodiazepines
5. Estazolam (Prosom)
6. Flurazepam (Dalmane)
7. Quazepam (Doral)
8. Temazepam (Restoril)
9. Triazolam (Halcion)
10. Non-benzodiazepine receptor agonists
11. Eszopiclone (Lunesta)
12. Zaleplon (Sonata)

13. Zolpidem (Ambien, Ambien CR, Intermezzo, Zolpimist, Edluar)
14. Antidepressants
15. Doxepin (Selinor)
16. Mirtazapine (Remeron)
17. Trazodone (Oleptra)

The dose of these drugs vary. Talk to your doctor about the exact dose you need to fall asleep.

Supplements interact with hypnotics

Combining sleep medications or using other medications that also cause drowsiness will lead to severe drowsiness. Stimulants such as amphetamines or coffee may reduce the effect of sleep medicines.

Pregnant or breastfeeding

Researchers have not studied the effects of most hypnotics in pregnant women. Therefore, they should not be used if you are pregnant unless it is absolutely necessary. Pregnant women who have sleep problems should try improving their sleep hygiene, relaxation, and ways to reduce stress. Benzodiazepine use is harmful to the fetus when taken by pregnant women during their first trimester so you should avoid them if you are pregnant. Diphenhydramine may have a low risk of causing harm during pregnancy.

CONCLUSION:

In this review human brain and its functional roles along with its moods, behaviour and the various drugs which act on this system were reviewed. Human Central Nervous System is the wide branch; it includes very complicated pathways and mechanisms.

Here we studied the neurotransmission process in our brain and the effect of all categories of drugs. Depression, Insomnia, Schizophrenia, Parkinson, Psychosis, etc. major disorders occur due to imbalance of various neurotransmitters. So in this project I tried to focus and cover all the topics related to CNS with deep study. It helps me and all to understand the mechanisms and drugs, along with some adverse effect also.

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