Editorial

Drug Addiction- A Silent Killer for Society

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Drug addiction is widely considered as a pathological state. The disorder of addiction involves the progression of acute drug use to the development of drug seeking behavior, the vulnerability to relapse, and the decreased, slowed ability to respond to naturally rewarding stimuli[1].

Addiction is a primary, chronic, neurobiological disease, with genetic, psychosocial and environmental factors influencing the development and manifestations. Physical dependence is a state of adaptation that is manifested by a drug class specific withdrawal syndrome that can be produced by abrupt cessation, rapid dose reduction, decreasing the blood level of drug and or administration of an antagonist[2].

Drug addiction is an epidemiological problem worldwide including Bangladesh. National survey shows that 0.16% population are related with substance use disorder among which adolescent group are more vulnerable. Epidemiological studies indicate that experimentation with addictive drugs and onset of addictive disorders is primarily concentrated in adolescence and young adulthood. An exploration of developmental changes in neurocircuitry involved in impulse control has significant implications for understanding adolescent behavior, addiction vulnerability, and the prevention of addiction in adolescent and adulthood.

Substance use disorders are a leading cause of medical morbidity, mortality and health expenditure in the United States and other developed countries. Regional availability of substance and social trends influence the prevalence of specific substance abuse disorders.Three major observations suggest that the Developmental periods of adolescent and adulthood are primary correlates of substance use and substance use disorders, operating across cultural trends and substances. First, adolescent and young adults generally exhibit higher rates of experimental use and substance use disorders than older adults, as indicated by studies of the general population spanning the last two decades and with the use of alternate diagnostic criteria.

Second, addictive disorders identified in adults most commonly have onset in adolescent or young adulthood . For example , most adult U.S smokers start smoking before 18 and the onset of daily smoking is uncommon after age 25. Over 40% of adult alcoholic experience alcoholism related symptoms begin between age 15 and 19, and 80% of all cases of all alcoholism begin before age 30. The age of initiation of illicit drug use in adults with substance use disorders is 16 years , with 50% of cases beginning between age 15 and 18 and rare initiation after age 20.

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Third, earlier onset of substance use predicts greater addiction severity and morbidity including use of and substance use disorders associated with multiple substances. Two key variables in the genesis of addictive disorders are the 1) degree / amount of drug intake and 2) the inherent vulnerability to addiction given a fixed amount of drug intake. Understanding whether one or both these factors are greater in adolescence is important in explaining the developmental onset of substance use disorders . Although cultural , peer, and family influences contribute to drug availability and substance experimentation, several lines of evidence suggest that sociocultural aspects particular to adolescent life alone don't fully account for greater drug intake. Although marketing and the availability of legal drugs (alcohol and nicotine) are pervasive across the groups in American society and are legally sanctioned only for adults, the onset of substance use disorders associated with these drugs is concentrated in adolescent and young adulthood and does not increase in a cumulative manner with increase age.

Genetic and neurobiological factors within individuals are thought to Lower the threshold of drug exposure required for " tripping the switch" from experimental to addictive drug use. Growing clinical evidence suggests that adolescence represents a period of heightened biological vulnerability to the addictive properties of illegal and legally sanctioned substance. For instance, adolescents demonstrate a more precipitous progression of illicit drug use than adults. Despite smoking fewer cigarettes than adults, adolescent show higher rates of dependence at similar level of use, although rates of alcohol use are similar throughout adolescence and adulthood, rates of abuse / dependence vary inversely with age. It is shown that basic and clinical evidence for adolescent neurodevelopment as a critical period of addiction vulnerability. Behaviors seemingly characterized by impulsivity and suboptimal decision making are described as normatives traits of adolescence corresponding to the development of motivational circuitry involve in the pathophysiology of addiction.

The prevalence of substance use disorders is elevated in adults with Schizophrenia, major affective disorders, antisocial and borderline personality disorders and pathological gambling. Adolescent with antecedent or fully expressed versions of these disorders are also more likely to have substance use disorders. Associations of these mental illnesses and and adolescence with substance use disorders suggests that common brain mechanisms may underlie vulnerability to substance use disorders in these contexts.

Psychiatric disorders commonly identified with disturbances in reward motivation and substance use disorder co morbidity is associated with impulsivity. Understanding the relationship between impulsivity and substance use disorders may prove important to understanding the pathogenesis of substance use disorders and their greater prevalence in specific clinical contexts, including adolescence. Individuals with poor impulse control show a thematic tendency to engage in behaviors characterized by long - term disadvantageous outcomes.

When examining the biological basis of drug addiction, one must first understand the pathways in which drugs act and how drugs can alter those pathways. The reward circuit , also referred to as the Mesolimbic system, is characterized by the interaction of the several areas of the brain.

The ventral tegmental area (VTA) consists of dopamine rigid neurons which respond to glutamate. The VTA supports learning and sensitization development and release dopamine (DA) into the forebrain[3]. These neurons also projects and release DA into the nucleus accumbens, through the Mesolimbic pathway. Vertically all drugs causing drug addiction increase the dopamine release in the Mesolimbic pathway[4].

The nucleus accumbens(NAcc) consists mainly of medium-spiny projection neurons(MSNs) which are GABA neurons. The NAcc is associated with acquiring and eliciting conditioned behaviors and involved in the increased sensitivity to drugs as addiction progresses[3].

The prefrontal cortex, more specifically the anterior cingulate and orbitofrontal Cortices , is importantvfornthe integration of information which contribute to whether a behavior will be elicited. It appears to be the area in which motivation originates and the salience of stimuli are determined[5].

The baspo lateral amygdala projects into the NAcc and is thought to be important for motivation as well[5]. More evidence is pointing towards the role of the hippocampus in drug addiction because of its importance in learning and memory[6]. So, cortical-striatal- thalamic -cortical loops are described as parallel because specific sub regions of the prefrontal cortex projects to specific compartments within the striatum, which in turn maintain some degree of segregation in projection to the thalamus and back to the cortex. Firing pattern in both the nucleus accumbens and prefrontal cortex are influenced by glutamatergic inputs from the hippocampus and amygdala, suggesting the abnormalities in this distal structures may produce both mental illness and motivational disorders.

Several explanations have been presented to explain addiction. These divide, more or less, into the models which stress biological or genetic causes for addiction, and those which stress social or purely psychological causes. Of course there are also many models which attemp to see addiction as both a physiological and psycho- social phenomenon.

References

1. Nutt D, King LA, Saulsbury W, Blakemore C. "Development of a rational scale to assess the harm of drugs of potential misuse".Lancet , 2007;369:1047-53.
2. The American Academy of Pain Medicine, the American Pain Society, and the American Society of Addiction Medicine."Definitions Related to the Use of opioids for the treatment of Pain"; 2001 (Http://www.painmed.org/pdf/definitions.pdf)
3. Jones S, BonciA ."Synaptic plasticity and drug addiction".CurrOpionPharmacol , 2005;5:20-5 doi:10.1016/j.coph.2004.08.011.PMID 15661621
4. Rang H. P. Pharmacology.Edinburgh : Churchill Livingstone, 2005: 596.
5. Floresco SB, Ghods- SharifiS ." Amygdala-prefrontal cortical circuitry regulates effort based decision making". Cereb. Cortex 2007;17:251-60.doi:10.1093/cercor/ bhj 143.PMID 16495432.
6. Eich AJ, HarburgGC . "Opiates psychostimulants , and adult hippocampal neurogenesis: Insights for addiction and stem cell biology". Hippocampus , 2006;16:271-86. doi:10.1002/hipo.20161.PMID 16411230