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Alcohol addiction and criminal behavior of culprits hospitalized pursuant to detention adjudged by the court as a protective measure in relation to an animal model

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Summary

Models of alcohol consumption and the effects of alcohol on the human body have been extensively studied using laboratory experiments on animals. These experiments demonstrated not only structural changes in the bodies of animals due to alcohol consumption but also behavioral disturbances. Pigs, due to their many anatomical, physiological, and behavioral similarities to humans, have become an ideal model. During the experiments, they exhibited voluntary alcohol consumption to the point of intoxication, and subsequently, despite negative symptoms of alcohol consumption (ataxia, consciousness disturbances, vomiting, etc.), they voluntarily continued to consume alcohol. Anatomical similarities in brain structure may provide probable patterns of injuries caused by alcohol consumption, just as in humans.

The aim of the study was to characterize the alcohol consumption habits among patients isolated in Forensic Psychiatry Department in relation to an animal model. The study group consisted of 24 men isolated in Forensic Psychiatry Department Medical University in Lublin studied with the use of a background survey and a questionnaire concerning the model of alcohol intake which is an own method developed for this research.

Knowing neurophysiological mechanisms responsible for the effects of alcohol consumption is crucial for conditioning in counteracting alcohol use disorder effectively. The studies of such processes in people are limited for ethical reasons, which are less restrictive regarding laboratory experimental animals.

The main age of the patients was 44 years old. No one in the study group was highly educated. 22.8% have consumed alcohol for more than 40 years. Over 45% of patients got drunk for the first time between 10 and 20 years of life. Almost half of the patients consumed alcohol for improvement of the mood. Around 9% had experiences with noncommercial alcohol. Only 36% participated in fights after alcohol intake. Half of the study group had retrograde amnesia and 23% drove after alcohol consumption. Only 22% were hospitalized in drying-out hospital. Almost 10% had serious diseases caused by alcohol intake. 46.6% were drunk during the commission of crimes. The most common psychotic disorder among these patients was Schizophrenia.

In studies of rats burdened with alcohol intake, as in intensive drinking at adolescence there was a stimulated increase in the number of mature and immature dendritic spines in pyramidal cells located in the V layer of the prefrontal cerebral cortex what was observed after adolescence, thus in adulthood of the rats. The main changes involved an increase in the number of unstable, lengthened, and thinned dendritic spines. Showing also a decline in the expression of catechol-O-methyltransferase.

The results of the above mentioned research on rats and observations of studies in people are a coherent whole presenting evidence on the damaging influence of typical alcohol intake in adolescence, showing itself in adulthood as destructive changes within the structure and functions of dopaminergic neurons, and also their functionally interdependent gabaergic neurons of the prefrontal cortex. The study group does not correlate their current condition with alcohol abuse. There is a great need for education to decrease the level of addictions and crimes.

Keywords: alcohol abuse, crime, schizophrenia

An increasing number of works published in medical literature indicate a causal connection between mental disorders, psychoactive substance addictions, and the occurrence of criminal and criminogenic behavior (8, 10, 15). This dependence is visible, especially in alcohol abuse and addiction (5). Research concerning alcohol problems indicates a higher occurrence rate among inpatients of forensic mental health wards than among the general population (13, 29). Attention is also paid to more and more visible co occurrence of three factors in forensic psychiatry which are mental disorders, addictions, and criminal behaviors (30). It is important to know the neurophysiological mechanisms responsible for the effects of alcohol consumption. The knowledge about these processes is based on research on laboratory experimental animals. In studies of rats burdened with alcohol there was a stimulated increase in the number of mature and immature dendritic spines in pyramidal cells located in the V layer of the prefrontal cerebral cortex. Dendritic spines are small projections on neurons that play a key role in synapses and signal transmission in the brain. Changes in their number and structure may indicate neuronal plasticity, which is important in the context of addiction and the brain's adaptation to the presence of psychoactive substances. Showing also a decline in the expression of catechol-O-methyl transferase (COMT). COMT is responsible for the metabolism of catecholamines such as dopamine, norepinephrine, and adrenaline. Changes in this enzyme can affect the functioning of neurotransmission and contribute to mood disorders and impulse control problems that are often seen in people who abuse alcohol.

The literature indicates that heavy drinkers (consuming $\geq$ 5 times a month) are significantly more likely to develop alcohol use disorder (AUD). Due to the significant prevalence of AUD, many experiments have been conducted using rodent models. They have played a major role in assessing the genetic and molecular implications of AUD; however, rodents have low alcohol preference, and therefore do not exhibit voluntary alcohol consumption (6). An attractive alternative for research on a human model of alcoholism is the pig, which anatomically, physiologically, and behaviorally exhibits some similarities to humans. In early studies of this species (11, 33, 34), with free access to water and alcohol in aqueous solution, bottle-choice tests were conducted under chronic ad libitum exposure. During this study, the animals displayed intoxication behaviors including ataxia, extreme passivity, altered states of consciousness, and occasional vomiting. Despite the negative symptoms, the animals continued to consume alcohol; upon withdrawal, static and volitional tremors, dilated pupils, and muscle fasciculation were observed. The study showed that despite alcohol intoxication, pigs continued to voluntarily consume alcohol, confirming the resemblance to the human model of alcohol dependence.

The aim of the study was to observe and compare the patterns of alcohol consumption between animals and culprits of prohibited acts (criminal) who according to detention adjudged by the court as a protective measure are inpatients of the Forensic Mental Health Ward at the Psychiatry Department and Clinic of Medical University in Lublin.

Material and methods

The research was conducted in a group of 24 men hospitalized pursuant to detention adjudged by the court as a protective measure at the Forensic Mental Health Ward at the Psychiatry Department and Clinic of Medical University in Lublin. Participation in the research was voluntary. The patients were informed about the aim of the research and the used methods.

Research tools were: a background survey and a questionnaire concerning the model of alcohol intake which is an own method developed for this research.

Results and discussion

The average age of the patients was 44 years and 2 months (range 26-62 years). None of the inpatients had a higher education degree. 68.2% of the researched patients had secondary education and 31.8% had only primary education.

Regarding the first contact with alcohol, only one person answered that they had never drank alcohol. 77.3% of the researched had drunk alcohol for the first time before their 20s, 18.2% of them before the age of 10. On the other hand, in 45.4% of the researched the first episodes of inebriety occurred before the age of 20 and in one person before the age of 10. Time of intense alcoholic drink intake was 10 years in 27.3%, 15 years in 22.8%, and more than 30 years in 13.6%.

The researched men most often drank beer (36.4%), vodka (27.3%), wine (182%), a combination of wine with vodka or beer (13.5%), and any kind of alcohol (4.5%). Over 90% of the researched admitted that they happened to combine alcohol with other substances, drugs, and medicines to intensify the intoxication effect. 9.1% admitted to regular non-consumable alcohol intake. Over 40% of the researched had to drink alcohol the next day after drinking to improve their mood. Half of the patients admitted to binge drinking, of which the longest was about four weeks.

72.7% of hospitalized had their own family, which in 31.8% of them saw the need for treatment. However, in 40.9% of the patients also other family members abused alcohol. In the case of 18.2%, their families broke down due to alcohol addiction and abuse. In 27.3% addiction was the cause of job loss.

22.7% of the patients were directed to treatment apart from the court detention and 31.8% tried that treatment. Almost 22.7% of the patients were also hospitalized at other wards than detoxification, whereas 10% developed disorders caused by alcohol abuse.

Over one-third of the patients (36.4%) started brawls, whereas half of them were beaten up or sustained in an accident when intoxicated. Almost one-fourth (22.7%) admitted to driving under the influence of alcohol. Half of the patients lost the ability to remember events while intoxicated.

In the researched group 41% of hospitalized patients were previously treated due to schizophrenia (F20), 23% due to persistent delusional disorders (F22), each 14% due to schizoaffective disorders (F25) and moderate mental retardation (F71), and 8% due to other mental disorders and dysfunctions caused by alcohol addiction or somatic disease.

45.5% of the researched were under the influence of alcohol when committing the prohibited act. In 33% of them, it was a crime concerning physical and mental abuse of a person from the closest family (Article 207 of the Criminal Code). In 19% of them the prohibited act concerned punishable threats (Article 190 of the Penal Code), and 23% were involved in a brawl or causing bodily injury (Article 157 and 158 of the Penal Code).

A key role in committing a crime among the researched group was alcohol, and the most common mental disease was paranoid schizophrenia. The vast majority of the researched drank alcohol already at a young age, and almost half of them experienced the first episode of inebriety before the age of 20. Almost one-third tried to treat alcohol addiction. In one of the studies, it was shown that abuse of psychoactive substances in patients with schizophrenia influences the risk of committing a crime involving violence (14).

Knowing neurophysiological mechanisms responsible for the effects of alcohol consumption at cellular, neural signaling, and genetic levels in adolescent development is crucial conditioning in counteracting alcohol use disorder effectively and in more adequate treatment of the patients. It is important to emphasize that studies of such processes in people have a restricted spectrum of disciplined approaches due to ethical reasons, which are less restrictive regarding laboratory experimental animals.

A particularly important cognitive and practical aim was to more completely recognize changes in the functioning of the dopaminergic system in the cerebral cortex as the most vulnerable to the toxic influence of alcohol in adolescence involving initiation and intensive alcohol intake as in addiction among the researched patients.

In studies of rats burdened with alcohol intake as in intensive drinking at adolescence, there was a stimulated increase in the number of mature and immature dendritic spines in pyramidal cells located in the V layer of the prefrontal cerebral cortex that was observed after adolescence, thus in the adulthood of the rats (9). The main changes involved an increase in the number of unstable, lengthened, and thinned dendritic spines, which indicated a disturbed pruning process, changed neuroplasticity, and inhibition of neural maturation while creating structurally mature pyramidal cells (9). Changes in the shape and density of dendritic spines are believed to be the structural basis of learning and memory (1). Addiction can therefore be considered a pathological form of memory.

An important statement of this research was also to show a decline in the expression of catechol-O-methyltransferase which determines the amount of dopamine synthesis and dopaminergic neurotransmission within the cerebral cortex (9). Similarly, there was a decrease in the expression of tyrosine hydroxylase, which is an enzyme limiting dopamine synthesis with a simultaneous increase of DNA methylation in the promoter region of the gene within exome II responsible for expression and amount of catechol methyltransferase. Indicated DNA hypermethylation within exome II released by ethanol influence and observed in the same rats only after entering adulthood was proved to improve epigenetic mechanism in the process of inhibition of expression and action of catechol-O-methyltransferase within the prefrontal cerebral cortex. In the research similarly directed changes were shown with inhibited modulation and activity of dopaminergic receptors in pyramidal cells of zone V, and also interneurons responsible for inhibiting influence and balance of activation and inhibition in the areas of the prefrontal cerebral cortex.

The effects of adolescent alcohol intake in the area of dopaminergic neurons turned out to be permanent damage to synthesis and expression and also functions of two crucial enzymes in the neurons of the prefrontal cerebral cortex, which determine the dopaminergic activity of performance and cognitive functions, including potential scope and ability of concentration, mindfulness, learning, critical perspective thinking along with satisfactory functioning of motivation, reward, and joy systems.

There is a theory that associates a dysregulation of glutamatergic transmission in the brain reward circuit with addiction development process. Back to the topic of dendritic spines, there are synapses containing a neurotransmitter called glutamate located on their surface.

According to the authors of one study, the development of behaviors related to alcohol addiction is also influenced by the process of autophosphorylation of the isoform of alpha-kinase dependent on calcium/calmodulin II by alcohol-induced remodeling of glutamatergic synapses in the hippocampus and amygdala (21).

Above mentioned regions of the brain are responsible for, among others: feeling emotions and processing information from short-term into long-term memory, in other words for the formation of memories (hippocampus) (20) and playing a key role in behavior and memory based on emotional associations (amygdala) (24).

Associating emotional events and memories with places can have a positive but also a negative side. For example, after a successful withdrawal period from drinking alcohol, alcoholics often do return to drinking despite their attempts to quit alcohol when they are in the same environment or surroundings that they associate with alcohol (22). This was the case among respondents at the Forensic Medicine Department of Lublin, e.g. in situations when other family members of addicts also showed alcohol addiction. The formation of associations between a substance and the place where it was taken can be relatively easily investigated using an animal experimental model. For example, by testing place preferences on rodents using a specially constructed cage, which enables checking whether the animal is more willing to stay in the compartment where it previously received the substance. When animals were given an addictive substance in a specific place, after stopping its administration they return to the same place in the cage, waiting for the next dose (7, 12).

Alcohol withdrawal syndrome consists of a number of somatic and psychiatric symptoms that are caused by the sudden withdrawal or reduction of the dose of ethyl alcohol, such as anxiety, irritability and convulsions. They are an important factor determining the so-called negative reinforcement motivating relapse in order to avoid them. The basolateral amygdaloid nucleus is a structure that has been confirmed to be involved in this emotional process and it has been proven that it is involved in reactions to alcohol withdrawal in both rats and humans. It has been shown, among other matters, that in humans it is responsible for the development of alcohol craving (3), and in rats it contributes to the generation of anxiety during alcohol withdrawal (36).

In vitro studies conducted on sections of the amygdala of animals both chronically receiving alcohol and after its withdrawal showed a significant increase in the density of NMDA receptors. Moreover, the same authors found that the case of chronic alcohol consumption leads to the amplitude and frequency of spontaneous excitation potential increase caused by the activation of AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid) receptors, which may manifest itself in the occurrence of convulsions during the period of abstinence (19, 25). AMPA receptors are the most common in the nervous system. They belong to ionotropic receptors, which, under the influence of ligand binding, undergo penetration for sodium (Na^+), potassium (K^+) and calcium (Ca^{2+}) ions. AMPA receptors with NMDA receptors are located in excitatory synapses, where they jointly open calcium channels in response to a stimulus, with NMDA receptors acting slower and longer, and AMPA faster and shorter (19).

The results of the above mentioned researches on rats and observations of studies in people are a coherent whole presenting evidence on the damaging influence of typical alcohol intake in adolescence, showing itself in adulthood as destructive changes within the structure and functions of dopaminergic neurons, and also their functionally interdependent gabaergic neurons of the prefrontal cortex.

Studies on monkeys show that after 6 months of systematic oral ethanol intake at a dose of $\geq$ 3 g/kg ethanol/day (12 alcoholic drinks), there is a significant reduction in brain volume in the region of the cerebral cortex. This condition persisted throughout the period of ethanol administration. Correlation analyses revealed a loss of cortical volume of approximately 0.11% of the intracranial vault for each daily drink (0.25 g/kg), as well as indicated a selective vulnerability of cortical and non-cortical areas of the brain. The pattern of volumetric changes after 15 months of ethanol intake indicates the first macroscopic sign of chronic exposure of the brain to ethanol (17).

Chronic alcohol consumption slows brain development in primates both during adolescence and in young adulthood. The remodeling of the brain that occurs at the transition between adolescence and adulthood is jeopardized as a result of alcohol consumption. Brain imaging in rhesus macaques (aged 3.5 to 7.5 years) before exposure to alcohol and after 6 and 12 months of systematic daily access to ethanol and water shows that alcohol consumption reduced the brain growth rate by 0.25 ml/year for each 1 g/kg of ethanol consumed daily. Excessive alcohol consumption affected the diminished growth rate of the thalamus. Thus, changes in brain volume during development from late adolescence to early adulthood in macaques are influenced by excessive alcohol consumption, which may be related to the continuation of excessive drinking later in adult life (28).

Among the patients subjected to court detention in the aftermath of committed crimes, the matter worth emphasizing is their age range involving adolescence when alcohol initiation and the first inebriety occurred. Causal agency in triggering alcoholism in an adult depends in 70% on alcohol initiation in adolescence. It is necessary to explain that in 2008 the World Health Organization recognized the whole decade of human development lasting from 10 to 20 years, between childhood and adulthood, as adolescence involving adolescence, youth, maturation, and pubescence. That is why when presenting causes of alcoholism and other addictive diseases of similar causal mechanisms, it is worth stressing the importance of adolescent alcohol initiation as the most dangerous and, at the same time, underestimated cause and risk of the disease transferred into adulthood and beginning in adolescence. It is appropriate to cite the WHO report on transmitting alcoholism from adolescence to adulthood (18).

Research confirms that excessive alcohol consumption in individuals aged 9 to 23 weakens the growth of white matter, with a decreasing volume and thickness of the cerebral cortex observed with age (23, 32). A reduction in the thickness of the cerebral cortex due

to exposure to ethanol vapors has also been demonstrated in experimental imaging studies on rodents (35). Although the differences in the length of alcohol metabolism between rodents and humans limit the ability to fully infer the effects of alcohol on the study subjects, a greater similarity is observed between humans and pigs. Thus, the similar patterns observed in pigs, including voluntary alcohol consumption and behaviors related to intoxication, allow for the establishment of a more representative model AUD and simultaneously increase the ability to determine factors associated with the development of AUD. The anatomical similarities between the brains of pigs and humans enable a more representative assessment of susceptibility and neurological tissue damage in response to AUD. In turn, similarities in liver structure result in a comparable process of alcohol elimination from the bodies of pigs and humans (27).

The highest percentage of people in the researched group were people with primary and secondary education, which indicates impairment and neglect in the process of continuous and obligatory education in adolescence causally connected with the abuse of alcohol at the time of the intensive educational process. It can also be observed in other research, where the higher risk of committing a crime was among people of lower socio-economic status regardless of schizophrenia occurrence (31). Other risk factors of violence in patients with schizophrenia are, among other things, young age, abuse of alcohol, a premorbid tendency to aggressive behavior, a broken home, or an early loss of a father (18).

In one-third of the researched patients, the crime concerned the abuse of a close family member. Most frequently, people under the influence of intoxicants release their negative and aggressive emotions toward those in their immediate surroundings. It also involves children who experience physical damage, burns, and fractures due to abuse from their parents or caretakers intensively consuming alcohol (2).

An important fact is also the co occurrence of other diseases caused by alcohol abuse which may result in hospitalization at other wards than detoxification. Alcohol impairs the immune system, which is proved by a two times higher risk of oesophagus and colon cancer, and breast cancer in women abusing alcohol. Other disorders include haemorrhagic gastritis, pancreatitis, hepatic cirrhosis, and a decrease in mineral bone density (26). Chronic abuse of alcohol may cause alcoholic cardiomyopathy which if untreated, can progress into irreversible cardiac failure. However, low and moderate alcohol intake may have a beneficial influence by decreasing the risk of a heart attack or stroke because it improves lipid profile and some changes in the activity of blood coagulation (26).

There is also a dependence between cognitive dysfunctions and the intensification of heart and cardiovascular system ailments (significantly higher among patients with cognitive dysfunctions than among those without any dysfunctions) (16).

It shall be stressed that in patients of psychiatric wards, along with an increased risk of organ disorders, there is also an increased occurrence of psycho-social problems (37). People chronically abusing alcohol, who simultaneously take medicines, expose themselves to the risk of therapeutic failures because alcohol changes the effects of drug influence by interfering with metabolic processes not only in the cellular structures of the organs, but also in the functioning of the central and peripheral nervous system, along with changes in the metabolism of the drugs. The effects of negative alcohol influence on healing processes may last for a long time despite limiting or eliminating alcohol intake (4).

The results indicate the criminogenic role of alcohol – almost half of the researched men were under the influence of alcohol when committing the crime. This criminogenic influence also involves the way alcohol impacts human functioning. Alcohol weakens moral principles, impairs the control of own behavior, and often leads to the straightforward display of rage or anger, and impulsive or aggressive behaviors. According to the research, alcohol intake had a relevant influence on the discipline of taking medications in outpatient treatment, and it impairs the effects of the drugs. It is justified to increase the level of education on threats caused by alcohol addiction, which may influence the decrease in the number of crimes committed by those people.

References

1. *Caroni P., Donato F., Muller D.*: Structural plasticity upon learning: regulation and functions. Nature Reviews Neuroscience 2012, 13, 478-490.
2. *Chikritzhs T., Livingston M.*: Alcohol and the risk of injury. Nutrients 2021, 13, 2777.
3. *Childress A. R., Mozley P. D., McElgin W., Fitzgerald J., Reivich M., O'Brien C. P.*: Limbic activation during cue-induced cocaine craving. Am. J. Psychiatry 1999, 156, 11-18.
4. *Citkowska-Kisielewska A., Rutkowski K., Sobański J. A., Dembinska E., Mielimaka M.*: Anxiety symptoms in obsessive-compulsive disorder and generalized anxiety disorder. Psychiatria Polska 2019, 53 (4), 845-864.
5. *Collins J. J.*: Alcohol careers and criminal careers, [in:] Collins J. J.: Drinking and crime: Perspectives on the relationships between alcohol consumption and criminal behavior. Tavistock Publications London 1982, p. 152-206.
6. *Crabbe J. C., Harris R. A., Koob G. F.*: Preclinical studies of alcohol binge drinking. Ann. N. Y. Acad. Sci. 2011, 1216, 24-40, doi: 10.1111/j.1749-6632.2010.05895.x.
7. *Cunningham C. L., Gremel C. M., Groblewski P. A.*: Drug-induced conditioned place preference and aversion in mice. Nature protocols 2006, 1, 1662-1670.
8. *Curran G. M., Sullivan G., Williams K., Han X., Allee E., Kotrla K. J.*: The association of psychiatric comorbidity and use of the emergency department among persons with substance use disorders: an observational cohort study. BMC Emergency Medicine 2008, 8, 17.
9. *Dahl R. E., Allen N. B., Wilbrecht L., Ballonoff Suleiman A.*: Importance of investing in adolescence from a developmental science perspective. Nature 2018, 554, 441-450.
10. *Davies R. D.*: Adolescent substance abuse: Psychiatric comorbidity and high-risk behaviors. Am. J. Psychiatry 2009, 166, 117.
11. *Dexter J. D., Tumbleson M. E., Hutcheson D. P., Middleton C. C.*: Sinclair (S-1) miniature swine as a model for the study of human alcoholism. Ann. N. Y. Acad. Sci. 1976, 273, 188-193, doi: 10.1111/j.1749-6632.1976.tb52881.x.
12. *Di Chiara G.*: A motivational learning hypothesis of the role of mesolimbic dopamine in compulsive drug use. Journal of Psychopharmacology 1998, 12, 54-67.

13. *Dobkin P., Dongier M., Cooper D., Hill J. M.*: Screening for alcoholism in a psychiatric hospital. Canadian Journal of Psychiatry 1991, 36, 39-45.
14. *Fazel S., Lingstrom N., Hjern A., Grann M., Lichtenstein P.*: Schizophrenia, substance abuse, and violent crime. JAMA 2009, 301, 2016-2020.
15. *Fortuna L. R.*: Adolescent substance abuse: Psychiatric comorbidity and high risk behaviors. Psychiatr. Serv. 2009, 60, 128.
16. *Gardner J. D., Mouton A. J.*: Alcohol effects on cardiac function. Comp. Physiol. 2015, 5, 791-802.
17. *Kroenke C. D., Rohlfing T., Park B., Sullivan E. V., Pfefferbaum A., Grant K. A.*: Monkeys that voluntarily and chronically drink alcohol damage their brains: a longitudinal MRI study. Neuropsychopharmacology 2014, 39 (4), 823-830, doi: 10.1038/npp.2013.259. Epub 2013 Sep 27. PMID: 24077067; PMCID: PMC3924514.
18. *Kudumija Slijepcevic M., Jukic V., Novalic D., Zarkovic-Palijan T., Milosevic M., Rosenzweig I.*: Alcohol abuse as the strongest risk factor for violent offending in patients with paranoid schizophrenia. Croat. Med. J. 2014, 55 (2), 156-162.
19. *Läck A. K., Diaz M. R., Chappell A., DuBois D. W., McCool B. A.*: Chronic ethanol and withdrawal differentially modulate pre- and post-synaptic function at glutamatergic synapses in rat basolateral amygdala. J. Neurophysiol. 2007, 98, 3185-3196.
20. *Lisman J., Buzsáki G., Eichenbaum H., Nadel L., Ranganath Ch., Redish D.*: Viewpoints: how the hippocampus contributes to memory, navigation and cognition. Nat. Neurosci. 2017, 20 (11), 1434-1447.
21. *Mijakowska Z., Łukasiewicz K., Ziółkowska M., Lipiński M., Trąbczyńska A., Matuszek Ż., Łęski Sz., Radwanska K.*: Autophosphorylation of alpha isoform of calcium/calmodulin-dependent kinase II regulates alcohol addiction-related behaviors. Addiction Biology 2015, 22 (2), 331-341.
22. *O'Brien C. P., Childtess A. R., McLellan A. T., Ehrman R.*: Classical conditioning in drug-dependent humans. Annals of the New York Academy of Sciences 1992, 654, 400-415.
23. *Pfefferbaum A., Rohlfing T., Pohl K. M., Lane B., Chu W., Kwon D., Nolan Nichols B., Brown S. A., Tapert S. F., Cummins K., Thompson W. K., Brumback T., Meloy M. J., Jernigan T. L., Dale A., Colrain I. M., Baker F. C., Prouty D., De Bellis M. D., Voyvodic J. T., Clark D. B., Luna B., Chung T., Nagel B. J., Sullivan E. V.*: Adolescent development of cortical and white matter structure in the NCANDA sample: role of sex, ethnicity, puberty, and alcohol drinking. Cereb Cortex 2016, 26, 4101-4121, doi: 10.1093/cercor/bhv205.
24. *Rasia-Filho A. A., Londero R. G., Achaval M.*: Functional activities of the amygdala: an overview. J. Psychiatry Neurosci. 2000, 25 (1), 14-23.
25. *Roberto M., Schweitzer P., Madamba S. G., Stouffer D. G., Parsons L. H., Siggins G. R.*: Acute and chronic ethanol alter glutamatergic transmission in rat central amygdala: an in vitro and in vivo analysis. J. Neurosci. 2004, 1594-1603.
26. *Schuckit M. A.*: Alcohol-use disorders. Lancet 2009, 373 (9662), 492-501.
27. *Shin S. K., Kaiser E. E., West F. D.*: Alcohol induced brain and liver damage: Advantages of a porcine alcohol use disorder model. Front. Physiol. 2021, 7, 11:592950, doi: 10.3389/fphys.2020.592950. PMID: 33488396; PMCID: PMC7818780.
28. *Shnitko T. A., Liu Z., Wang X., Grant K. A., Kroenke C. D.*: Chronic alcohol drinking slows brain development in adolescent and young adult nonhuman primates. eNeuro 2019, 9, 6 (2): ENEURO.0044-19. 2019, doi: 10.1523/ENEURO.0044-19.2019. PMID: 30993181; PMCID: PMC6464511.
29. *Singleton N., Meltzer H., Gatward R., Cold J. W., Deasy D.*: Psychiatric morbidity among prisoners in England and Wales. HMSO. London 1998.
30. *Snowden P.*: Substance misuse and violence: the scope and limitations of forensic psychiatry's role. Adv. Psych. Treat. 2001, 7, 189-197.
31. *Stompe T.*: Family and social influences on offending in man with schizophrenia. Aust. N. Z. J. Psychiatry 2006, 40, 554-560.
32. *Squeglia L. M., Tapert S. F., Sullivan E. V., Jacobus J., Meloy M. J., Rohlfing T., Pfefferbaum A.*: Brain development in heavy-drinking adolescents. Am. J. Psychiatry 2015, 172, 531-542, doi: 10.1176/appi.ajp.2015.14101249.
33. *Tumbleson M. E., Dexter J. D., Middleton C. C.*: Voluntary ethanol consumption by female offspring from alcoholic and control Sinclair (S-1) miniature dams. Prog. Biochem. Pharmacol. 1981, 18, 179-189.
34. *Tumbelson M. E., Hutcheson J. D., Dexter J. D., Middleton C. C.*: Miniature swine as a model for the study of human alcoholism: the withdrawal syndrome, [in:] Gastineau C., Darby W., Turner T. (eds.): Fermented Food Beverages in Nutrition. Academic Press; Cambridge, MA 1979, 457-475, doi: 10.1016/b978-0-12-277050-0.50035-5.
35. *Vetreno R. P., Yaxley R., Paniagua B., Johnson G. A., Crews F. T.*: Adult rat cortical thickness changes across age and following adolescent intermittent ethanol treatment. Addict Biol. 2017, 22, 712-723, doi: 10.1111/adb.12364.
36. *Watanabe T., Nakagawa T., Yamamoto R., Maeda A., Minami M., Satoh M.*: Involvement of glutamate receptors within the central nucleus of the amygdala in naloxone-precipitated morphine withdrawal induced conditioned place aversion in rats. Jpn. J. Pharmacol. 2002, 88, 399-406.
37. *Wichniak A., Dudek D., Heitzman J., Kaplon-Cieslicka A., Mamcarz A., Samochowiec J., Szulc A., Bienkowski P.*: Metabolic risk reduction in patients with schizophrenia treated with antipsychotics: recommendations of the polish psychiatric association. Psychiatr. Pol. 2019, 53 (6), 1191-1218.

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