We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Personalized Treatment of Antisocial Personality Disorder with Inherent Impulsivity and Severe Alcoholism: A Brief Review of Relevant Literature

Roope Tikkanen

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/intechopen.73586>

Abstract

Antisocial personality disorder (ASPD) is a persistent psychiatric disorder. Behaviors and emotions deviate from the norm. Inherent impulsivity, comorbid alcohol dependence, and violation of laws cause severe challenges at individual and societal levels. Both environment and heritability alter the risk for ASPD. Research shows that specific biologic changes predispose to this disorder. Biological factors may lead to novel possibilities to treat and alleviate symptoms with medications or nutritional means. However, treatment of ASPD meets particular challenges due to the inherent symptoms of the disorder, and firm evidence-based personalized treatments are scant. This chapter describes the disorder and associated adverse outcomes in life such as recurrent violent behavior and increased mortality. Moreover, treatment possibilities are discussed covering risk assessment, medication, psychotherapy, and nutrition.

Keywords: antisocial personality disorder, ASPD, alcoholism, impulsivity, violent behavior

1. Introduction

Antisocial personality disorder (ASPD) is a psychiatric disorder, which despite being probably heavily under diagnosed has at least a prevalence of 1 in 100 persons [1]. The golden standard of treatment of psychiatric disorders is a combination of pharmacological treatment and psychotherapy. To date, there are no specific golden standards of pharmacological or psychotherapeutic treatments of ASPD, but in the future, treatment results may improve

after careful selection of whom to treat. ASPD forms a patient group, which has a poor treatment compliance in general due to inherent diagnostic symptoms of unplanned lifestyle and impulsive decision-making.

2. What is ASPD: what to treat

The core symptoms of ASPD are antisocial attitudes that lead to generally destructive behavior in part due to the inherent impulsive behavior and severe alcoholism (early-onset type II alcoholism) associated with this disorder. ASPD is diagnosed among adults, but it is preceded by conduct disorder symptoms before age 15, symptoms such as running away from home, initiation of physical fights, usage of weapons in fights, forcing others into sexual activity, cruelty to animals, destruction of other persons' property, deliberately engaged in fire-setting, frequent lying, and stealing. An extensive description of conduct disorder, however, is beyond the scope of this article.

Diagnostic symptoms of ASPD have not changed much over time. The symptom cluster includes the following type of items [2]:

- Inability to sustain consistent work or studies due to lack of motivation, repeated absence from work, impulsive abandonment of several jobs without realistic plans of future jobs or education.
- Failure to conform to social norms with respect to lawful behavior, as indicated by repeatedly performing acts that are grounds for arrest (whether arrested or not), e.g., destroying property, harassing others, stealing, and pursuing an illegal occupation.
- Irritability and aggressiveness, as indicated by repeated physical fights or assaults.
- Repeatedly failing to honor financial obligations, as indicated by defaulting on debts or failing to provide child support on regular basis.
- Impulsive behavior and inability to plan ahead, as indicated by, e.g., traveling from place to place without a prearranged job or clear goal for the period of travel or clear idea about when the travel will terminate and lack of a fixed address for a long period.
- No respect for the truth as indicated by repeated lying, use of aliases, and "conning" others for personal profit or pleasure.
- Recklessness regarding own or others' safety, as indicated by driving while intoxicated or recurrent speeding.
- If being a parent or guardian, lacks ability to function as a responsible parent, as indicated by, for instance, malnutrition of child, child's illness resulting from lack of minimal hygiene, child's dependence on neighbors or nonresident relative for food or shelter, failure to arrange for a caretaker for your child when parent is away from home, and repeated squandering on personal items or of money required for household necessities.

- Never sustained a totally monogamous relationship for more than 1 year.
- Lacks remorse (feels justified in having hurt, mistreated, or stolen from others).

Realistic foci of treatment for persons diagnosed with ASPD are (i) modulation of attitudes, (ii) decrease of impulsive behavior, and (iii) treatment of alcoholism.

3. Personalized focus and selection of whom to treat

As patient care resources and skills to treat specifically ASPD are limited, there is a strong rationale to carefully choose the target groups of individuals suffering from ASPD, whom should persistently be encouraged and motivated to treatment. For this, two separate ways of selective approach may be adapted, each focusing on different criteria. One alternative for selection of whom to treat in this patient group would be a focus in primary prevention when the first symptoms appear and accumulate (early interventions). Another focus is secondary prevention and treatment when the first tangible severe consequences of having ASPD appear.

Some of the more obvious measurement tools that could be considered for the decision-making on whether to start an intervention or not would be presence of persistent alcohol consumption and conduct disorder symptoms appearing at an early age (age 10–14) or acts leading to a prison sentence. An act of violence that leads to a prison sentence is unfortunately frequent among ASPD patients in early adulthood preceded by several smaller violations of the law not leading to incarceration. However, in the incarcerated environment, a meaningful secondary prevention effort can be arranged. As 50% of prison populations are diagnosed with ASPD [3], it is unrealistic to provide treatment for all ASPD patients, but a good rationale for focus would be those individuals who are at an increased risk for reoffenses.

Decisions for offering treatments are dichotomous but symptoms and risk are often measured on a continuum from mild to severe. However, scales of symptom and risk severity can easily be constructed, and often a meaningful cutoff point, depending on the size of the selection population, is the 75–90% percentile. Continuums such as alcohol consumption quantity, trait impulsivity, and accumulation of acts of violent can be transformed into categories of high-low expected efficacy of treatment for the individual or high-low expectancy of benefit for society. Both interests are often in line with each other.

4. Risk assessment for recurrent violent behavior or premature death

The tools for assessing risk for future acts of violence in previously violent populations have been criticized as an inexact science [4]. Many attempts of constructing assessment tools have been made, but their accuracy is far from satisfactory. For instance, the risk assessment tools HCR-20 and PCL-R rely on psychological assessments and historical data on life events, which may be difficult to assess. Moreover, application of such tool needs highly trained

professionals. The accuracy of such assessments suffers from several sources of bias such as underlying biologic individual variation [5]. Likewise, childhood maltreatment may also have ambiguous inter-rater variations and compromised data source reliability. However, childhood maltreatment has been unambiguously assessed to increase the risk for antisocial behavior, recurrent violent behavior, and premature death at the age of 40, especially in combinations with biological variations (e.g., different monoamine oxidase A genotypes) leading to alteration of risk [6–8]. Alcohol consumption has shown a positive correlation with increase in risk for recurrent acts of violence, whereas aging has shown to have a negative correlation to this outcome measure [9]. The biologic field of research may have most new tools to contribute to risk assessments in the future.

The biological research in violent reconvictions may reveal new possibilities to pharmacological treatments of ASPD and support decisions on whom to treat. The method for obtaining reliable scientific evidence of causalities leading to recurrent violent behavior is conducting long follow-up study settings in large cohorts. Both ASPD and severe alcoholism are considered hereditary. A consensus approximation of the hereditary component of alcoholism is 50% [10]. Impulsivity, composed of a persistent tendency to act on the spur of the moment and locomotive restlessness, is also partly hereditary [11]. Detection of genetic causalities underlying ASPD, severe alcoholism, and impulsivity has to date resulted in a handful of robust findings mainly through utilization of violent alcoholics in a young Finnish founder population. Genetic research in founder population allows an increased power to detect specific genetic risk for complex disorders due to a relatively homogenous gene pool caused by geographic isolation [12]. Six functional genetic loci associated with ASPD, impulsivity, alcoholism, and violent behavior, have been detected in the Finnish founder population. These genes comprise the serotonin 2B receptor (*HTR2B*) [13], tryptophan hydroxylase 2 (*TPH2*) [14], serotonin 1B receptor (*HTR1B*) [15], serotonin 3B receptor (*HTR3B*) [16], monoamine oxidase A (*MAOA*) [7, 9, 17], and T-cadherin [17].

Apart from specific genes, variation in glucose and insulin metabolism, which could be regarded as biomarkers, has also been shown to robustly predict recurrent violent behavior [18, 19]. Likewise, low serotonin levels have also been robustly associated with impulsive-aggressive behavior [20, 21].

Detection of individuals with high risk, especially due to biologic reasons, may raise ethical issues whether to treat or isolate individuals with increased risk for recurrent violent behavior. However, for medical professionals, the importance of treatment efforts of the psychiatric disorder ASPD, alcoholism, and overt impulsivity is clear as treatment is offered for other diseases with underlying biologic causalities such as diabetes or strokes, as well.

5. Medication

Impulsivity and alcoholism frequently coexist. Consequently, treatment of either symptom or disorder alleviates the other. The treatment of alcoholism is more advanced compared with treatment of impulsivity. At best, specific genetic data may reveal who will benefit from a

specific treatment in a dichotomous manner. A prime example of this is that individuals having a specific gene variant (Asp40) in the μ -opioid receptor (OPRM1) seem to clearly benefit from naltrexone treatment of alcoholism, whereas individuals with another genetic variant do not benefit from this medication [22]. Although there is a paucity of studies with such clear evidence of one gene's effect on a particular pharmacological treatment, this is the direction research should move into. Kupila et al. [23] recently found that the severity of alcoholism (Cloninger's type I vs. type II) alters pharmacological binding to the glutamate system, which is associated with addictions. Such findings give clues for meaningful study-setting which could contribute to future evidence-based personalized medical management. The severity of alcoholism has also been examined as a predictor in some pharmacological studies. It seems that patients with severe alcohol dependence (type II), which is associated with ASPD, may react differently to medications as compared with patients with the less severe form of alcoholism (type I), which is the type of alcohol dependence that the majority of "alcoholics" suffer from. For instance, antagonism of the serotonin 3 receptor with ondansetron has shown beneficial in the treatment of early onset severe alcoholism (type II), whereas no considerable therapeutic effect appears among patients with less severe alcoholism [24].

Moreover, carriers of a loss-of-function point mutation allele of the serotonin 2B receptor (*HTR2B* Q20*) may benefit from focused treatment of alcoholism and impulsivity as this mutation has been shown to make the carriers of this mutation more susceptible for problem-behavior especially while under the influence of alcohol, but also while sober [25]. However, this point mutation in the serotonin 2B receptor has only been found among Finns at this point, but this may serve as an example of a functional genetic discovery, which may lead to development of personalized treatments. Widely used serotonin selective reuptake inhibitors (SSRIs), such as fluoxetine and citalopram, do not seem to decrease depression or suicide rates among human *HTR2B* Q20* carriers or *Htr2b* knockout mice [26, 27]. Therefore, SSRIs likely show no effect on *HTR2B* Q20* ASPD carriers, but new pharmacological strategies are being currently developed [28].

Due to a lack of highly efficient pharmacological treatment of impulsivity and aggression, a vast variety of psychotropic medications have been used for treatment of impulsivity and aggression. However, recent research both in animal study settings [29] and large human follow-up cohorts [30] suggests that lithium could be the drug of choice for preventative treatment of impulsive behavior, due to the associated lower risk of suicide attempts and significantly decreased suicide mortality, in comparison to valproic acid and benzodiazepines, in high-risk bipolar patients [30].

6. Psychotherapy

Psychotherapy aims at alleviating various symptoms, modulate thought constructs, and to gain control of behavior such as antisocial attitudes, drinking alcohol, or acting impulsively. A slight pessimism regarding the efficacy of psychotherapy in treating ASPD is frequent among clinicians, which is partly justified as antisocial thought constructs and impulsive behavior decrease treatment compliance. Persons suffering from personality disorders tend

to have difficulties to realize or admit that they have a mental health problem that would need treatment. This is especially true for ASPD. However, this pessimism for the efficacy of treatment is not justified for all ASPD patients. For instance, group-based cognitive and behavioral interventions focused on reducing offending and other antisocial behaviors in an optimistic and trusting context have shown some good results [31]. It is also important not to have to ambitious goals, but to aim at alleviating some symptoms such as impulsive behavior, decrease in alcohol consumption, and decrease in disrespect of the rights of others. Clinical experience in the field of forensic psychiatry suggests that life events such as coming into religious faith and commitment to a strong-willed spouse would represent a “natural” psychotherapy altering thought constructs and behavior, and it has proved to be helpful for some ASPD patients. Consequently, simple psychotherapeutic themes in treatment of ASPD would be discussing religious interests and the benefits of being in a stable long-term relationship.

7. Nutrition

There is no convincing robust scientific evidence for treating ASPD with specific diets alone, but the inherent impulsivity and lack of persistence in ASPD certainly cause poor dietary habits. Many vitamins and other compounds received through food are vital for the proper biologic and physiologic functioning of the body. Thus, it is likely—and a fair hypothesis—that specific diets and supplements may show beneficiary for the treatment of ASPD with associated symptoms in the future as a part of the treatment strategy. One randomized placebo-controlled study suggests that the influence of supplementary vitamins, minerals, and essential fatty acid would decrease antisocial behavior and violence in an incarcerated context [32]. These kinds of study settings should be replicated and performed in outpatient study settings.

Some general advice, although speculative, on what diet to recommend for ASPD individuals could be mentioned. As low serotonin levels have been coupled with impulsivity and aggression [20, 21], a tangible means of ensuring a sufficient supply of tryptophan in the diet would be consumption of cheese and peanuts. When an alcoholic is being in a prolonged relapse period of drinking, it may be a good advice to make sure to eat glucose-rich food, as low glucose levels have been associated to violent and irritable behavior [20]. On the other hand, a glucose-rich diet may be harmful in the long run as it increases the risk for type 2 diabetes, and high insulin levels have been associated with increased risk for acts of violence [18].

8. Conclusions and future direction

As most of the acts of violence in the western world are attributable to individuals having ASPD, enhancement of the treatment of ASPD will have tangible effects in terms of reduction of violence in society. Most new ground is expected to be gained through an increasing understanding of biological causalities underlying ASPD will help to personalize treatments, including the therapeutic areas such as redox state and diet. Combination of several treatment strategies likely amounts in best treatment results. As treatment is challenging, professionals

should be trained to treat this specific patient group. Furthermore, clinicians—including general practitioners—would need education in recognizing symptoms associated with ASPD and encouragement to refer these patients to specialized treatment, although the problem remains that ASPD patients rarely seek treatment due to the inherent nature of the disorder. Conclusively, it should be noted that research supporting evidence-based personalized treatment of ASPD is sparse. This review therefore reflects the clinical and theoretical expertise of the author, which hopefully could initiate further research.

Author details

Roope Tikkanen

Address all correspondence to: roope.tikkanen@helsinki.fi

Department of Psychiatry, University of Helsinki and Laboratory of Neurogenetics,
National Institute on Alcohol Abuse and Alcoholism, National Institute of Health,
Rockville, MD, USA

References

- [1] Lenzenweger MF, Lane MC, Loranger AW, Kessler RC. DSMIV personality disorders in the national comorbidity survey replication. *Biological Psychiatry*. 2007;**62**(6):55364
- [2] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3rd revised ed. Washington, DC: American Psychiatric Press; 1987
- [3] Fazel S, Danesh J. Serious mental disorder in 23000 prisoners: A systematic review of 62 surveys. *Lancet*. 2002;**359**(9306):54550
- [4] Doyle M, Dolan M. Violence risk assessment: Combining actuarial and clinical information to structure clinical judgements for the formulation and management of risk. *Journal of Psychiatric and Mental Health Nursing*. 2002;**9**(6):64957
- [5] Tikkanen R, AuvinenLintunen L, Ducci F, Sjoberg RL, Goldman D, Tiihonen J, Ojansuu I, Virkkunen M. Psychopathy, PCLR, and MAOA genotype as predictors of violent reconvictions. *Psychiatry Research*. 2011;**185**(3):3826
- [6] Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R. Role of genotype in the cycle of violence in maltreated children. *Science*. 2002;**297**(5582):8514
- [7] Tikkanen R, Ducci F, Goldman D, Holli M, Lindberg N, Tiihonen J, Virkkunen M. MAOA alters the effects of heavy drinking and childhood physical abuse on risk for severe impulsive acts of violence among alcoholic violent offenders. *Alcoholism: Clinical and Experimental Research*. 2010;**34**(5):85360
- [8] Tikkanen R, Holli M, Lindberg N, Tiihonen J, Virkkunen M. Recidivistic offending and mortality in alcoholic violent offenders: A prospective follow-up study. *Psychiatry Research*. 2009;**168**(1):1825

- [9] Tikkanen R, Sjoberg RL, Ducci F, Goldman D, Holm M, Tiihonen J, Virkkunen M. Effects of MAOA genotype, alcohol consumption, and aging on violent behavior. *Alcoholism: Clinical and Experimental Research*. 2009;**33**(3):42834
- [10] Ducci F, Goldman D. The genetic basis of addictive disorders. *The Psychiatric Clinics of North America*. 2012;**35**(2):495519
- [11] Bevilacqua L, Goldman D. Genetics of impulsive behaviour. *Philosophical Transactions of the Royal Society of London Series B: Biological Sciences*. 2013;**368**(1615):20120380
- [12] Lim ET, Wurtz P, Havulinna AS, Palta P, Tukiainen T, Rehnstrom K, Esko T, Magi R, Inouye M, Lappalainen T, et al. Distribution and medical impact of loss-of-function variants in the Finnish founder population. *PLoS Genetics*. 2014;**10**(7):e1004494
- [13] Bevilacqua L, Doly S, Kaprio J, Yuan Q, Tikkanen R, Paunio T, Zhou Z, Wedenoja J, Maroteaux L, Diaz S, et al. A population specific HTR2B stop codon predisposes to severe impulsivity. *Nature*. 2010;**468**(7327):10616
- [14] Zhou Z, Roy A, Lipsky R, Kuchipudi K, Zhu G, Taubman J, Enoch M, Virkkunen M, Goldman D. Haplotype-based linkage of tryptophan hydroxylase 2 to suicide attempt, major depression, and cerebrospinal fluid 5hydroxyindoleacetic acid in 4 populations. *Archives of General Psychiatry*. 2005;**62**(10):110918
- [15] Lappalainen J, Long JC, Eggert M, Ozaki N, Robin RW, Brown GL, Naukkarinen H, Virkkunen M, Linnoila M, Goldman D. Linkage of antisocial alcoholism to the serotonin 5HT1B receptor gene in 2 populations. *Archives of General Psychiatry*. 1998;**55**(11):98994
- [16] Ducci F, Enoch M, Yuan Q, Shen P, White KV, Hodgkinson C, Albaugh B, Virkkunen M, Goldman D. HTR3B is associated with alcoholism with antisocial behavior and alpha EEG power: An intermediate phenotype for alcoholism and comorbid behaviors. *Alcohol*. 2009;**43**(1):7384
- [17] Tiihonen J, Rautiainen M, Ollila HM, RepoTiihonen E, Virkkunen M, Palotie A, Pietilainen O, Kristiansson K, Joukamaa M, Lauerma H, et al. Genetic background of extreme violent behavior. *Molecular Psychiatry*. 2015;**20**(6):78692
- [18] Ojala KP, Tiihonen J, RepoTiihonen E, Tikkanen R, Virkkunen M. Basal insulin secretion, PCLR and recidivism among impulsive violent alcoholic offenders. *Psychiatry Research*. 2015;**225**(3):4204
- [19] Virkkunen M, Rissanen A, FranssilaKallunki A, Tiihonen J. Low nonoxidative glucose metabolism and violent offending: An 8 year prospective follow-up study. *Psychiatry Research*. 2009;**168**(1):2631
- [20] Virkkunen M, Linnoila M. Brain serotonin, type II alcoholism and impulsive violence. *Journal of Studies on Alcohol Supplement*. 1993;**11**:163-169
- [21] Virkkunen M, Rawlings R, Tokola R, Poland RE, Guidotti A, Nemeroff C, Bissette G, Kalogeras K, Karonen SL, Linnoila M. CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic, violent offenders, fire setters, and healthy volunteers. *Archives of General Psychiatry*. 1994;**51**(1):207

- [22] Anton RF, Oroszi G, O'Malley S, Couper D, Swift R, Pettinati H, Goldman D. An evaluation of mu-opioid receptor (OPRM1) as a predictor of naltrexone response in the treatment of alcohol dependence: Results from the combined pharmacotherapies and behavioral interventions for alcohol dependence (COMBINE) study. *Archives of General Psychiatry*. 2008;**65**(2):13544
- [23] Kupila J, Karkkainen O, Laukkanen V, Hakkinen M, Kautiainen H, Tiihonen J, Storvik M. [3H]Ifenprodil binding in postmortem brains of Cloninger type 1 and 2 alcoholics: A whole hemisphere autoradiography study. *Psychiatry Research*. 2015;**231**(3):197201
- [24] Johnson BA. Update on neuropharmacological treatments for alcoholism: Scientific basis and clinical findings. *Biochemical Pharmacology*. 2008;**75**(1):3456
- [25] Tikkanen R, Tiihonen J, Rautiainen MR, Paunio T, Bevilacqua L, Panarsky R, Goldman D, Virkkunen M. Impulsive alcohol-related risk-behavior and emotional dysregulation among individuals with a serotonin 2B receptor stop codon. *Translational Psychiatry*. 2015;**5**:e681
- [26] Diaz SL, Narboux-Neme N, Boutourlinsky K, Doly S, Maroteaux L. Mice lacking the serotonin 5-HT_{2B} receptor as an animal model of resistance to selective serotonin reuptake inhibitors antidepressants. *European Neuropsychopharmacology*. 2016;**26**(2):265-279
- [27] Rahikainen AL, Majaharju S, Haukka J, Palo JU, Sajantila A. Serotonergic 5HTTLPR/rs25531 s-allele homozygosity associates with violent suicides in male citalopram users. *American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics*. 2017;**174**(7):691-700
- [28] Maroteaux L, Ayme-Dietrich E, Aubertin-Kirch G, Banas S, Quentin E, Lawson R, et al. New therapeutic opportunities for 5-HT₂ receptor ligands. *Pharmacology & Therapeutics*. 2017;**170**:14-36
- [29] Halcomb ME, Gould TD, Grahame NJ. Lithium, but not valproate, reduces impulsive choice in the delay discounting task in mice. *Neuropsychopharmacology*. 2013;**38**(10):193744
- [30] Toffol E, Hatonen T, Tanskanen A, Lonnqvist J, Wahlbeck K, Joffe G, Tiihonen J, Haukka J, Partonen T. Lithium is associated with decrease in all-cause and suicide mortality in high-risk bipolar patients: A nationwide registry-based prospective cohort study. *Journal of Affective Disorders*. 2015;**183**:15965
- [31] Kendall T, Pilling S, Tyrer P, Duggan C, Burbeck R, Meader N, Taylor C, Guideline Development Groups. Borderline and antisocial personality disorders: Summary of NICE guidance. *BMJ*. 2009;**338**:b93
- [32] Gesch CB, Hammond SM, Hampson SE, Eves A, Crowder MJ. Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners. Randomised, placebo controlled trial. *British Journal of Psychiatry*. 2002;**181**:228