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Can psychopathy be treated?

D. Durães*, R. Borralho
Centro Hospitalar Barreiro-Montijo, Mental Health and Psychiatry Department, Barreiro, Portugal
* *Corresponding author.*

Introduction Psychopaths are incapable of feeling empathy and guilt, being responsible for most violent crimes. To date, confinement has been the option of choice to minimize the harm they inflict. However, a deeper understanding of the neurobiology of psychopathy may lead to new insight on possible treatment approaches.
Aims This work aims to review the current knowledge in psychopathy treatment.
Methods A literature search of MEDLINE (2000-present) was conducted using the search terms "psychopathy" + "treatment" and "drug therapy".
Results Defects in the amygdala and the prefrontal cortex have been implicated in the pathological basis of psychopathy. The most affected areas are the ventromedial prefrontal cortex (VMPC) and the associated anterior cingulated cortex. Alterations in connectivity between the amygdala and the VMPC with other areas of the brain have been demonstrated and seem to be responsible for the non-empathetic, unemotional, and amoral features of psychopaths. Also, they present an increase in dopamine turnover and metabolism and a serotonin dysregulation.
As not all individuals with the biological substrate for psychopathy become violent, it seems that plasticity in forebrain circuits may allow the development of more prosocial responses, especially in youth. Some authors emphasize the need to address other behaviours that can be responsible for violent actions, namely, impulsive aggression. Some drugs have shown efficacy in controlling impulsive aggression.
Conclusions Pharmacological approaches to treating psychopathy have been disappointing. A more reasonable goal would be to focus on impulsive aggression, for which treatment effectiveness has been demonstrated. Additional research is needed if we hope to design rational therapeutic strategies for this disorder.
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Investigating misophonia: A review of the literature, clinical implications and research agenda reflecting current neuroscience and emotion research perspectives

M. Erfanian[1,*], J. Jo Brout[2], M. Edelstein[3], S. Kumar[4], M. Mannino[5], L.J. Miller[6], R. Rouw[7], M.Z. Rosenthal[8]
[1] *Maastricht University, Neuroscience and Psychology, Maastricht, The Netherlands*
[2] *International Misophonia Research Network, Misophonia, New York, USA*
[3] *University of California, Brain and Cognition, San Diego, USA*
[4] *Newcastle University, Neuroscience, Newcastle, United Kingdom*
[5] *Florida Atlantic University, Complex Systems and Brain Sciences, Boca Raton, USA*
[6] *STAR Institute for Sensory Processing Disorder, Sensory Processing Disorder, Greenwood Village, USA*
[7] *Amsterdam University, Brain and Cognition, Amsterdam, The Netherlands*
[8] *Duke University, Psychiatry and Behavioral Science, Durham, USA*
* *Corresponding author.*

Misophonia is a complex neurobehavioral syndrome phenotypically characterized by heightened autonomic nervous system arousal and negative emotional reactivity, in response to specific sounds [1–3]. Research from basic and applied fields are synthesized with studies explicitly designed to investigate misophonia in an effort to more specifically conceptualise this syndrome. The purpose of this study is to review the emerging misophonia research and to integrate cross-disciplinary research in order to inform conceptualisation of this recently defined syndrome. Recently published case studies, descriptive studies, and laboratory-based psycho-physiological and neurobiological research are reviewed within a transdiagnostic and multi-disciplinary perspective. Finally, a brief discussion of updated neuroscience paradigms of emotion, including defence/fear circuitry related to the amygdala, is included to help more clearly contextualise findings from previous research and inform future studies investigating misophonia. From this perspective misophonia may be considered a central nervous system dysfunction associated with threat cue responding. Clinical implications should first stress coping skills, as there is no evidence-based treatment for misophonia. Ideally, clinicians would work together in cross-disciplinary teams to assist in individualizing coping skills plans for patients. However, for each clinician understanding the neurophysiological, emotional and behaviour manifestations of misophonia is essential, as a practitioner cannot simply apply one specific known therapy at this point, or haphazardly integrate what is known without up-to-date in depth knowledge of the research in so far as it is currently understood, as well as the impact on individual's lives and that of their families.
Disclosure of interest The authors have not supplied their declaration of competing interest.
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Psychogenic polydipsia: A case report

G. Erzin*, K. ozdel , H. Karadağ
Dıskapı Yıldırım Beyazıt Education and Research Hospital, Psychiatry, Ankara, Turkey
* *Corresponding author.*

Introduction Psychogenic or primary polydipsia characterized by excessive thirst and compulsive water drinking is a common problem among psychiatric populations, affecting 6% to 20% of patients. It is frequent in chronic psychiatric diseases, particularly schizophrenia. We report a patient with excessive thirst and diagnosed as PIP syndrome.
Case A 54-year-old, married, female patient had normal vital signs. She has excessive water intake (10–12 L/day). She did not have edema, signs of dehydration or fever. The neurological examination, CT, MRI, and EEG was normal. The laboratory tests were normal. She had started using sertraline 100 mg, 7 months ago due to anxiety disorder. There is not any disease except the anxiety disorder, which is in remission due to the treatment. A total of, 2 μg desmopressin I.M. is applied in fluid restriction test. The urine density is determined as 1.008 mg/dL initially, 1.011 mg/dL one hour later, and 1.013 mg/dL two hours later in the urinary test. The diagnosis is psychogenic polydipsia (primary) according to patient history, the clinical examination, and the test results. The patient is recommended to continue the sertraline 100 mg treatment, and also assigned with fluid restriction behaviour.
Conclusion Since excess water intake periods are correlated with psychotic exacerbations; psychosis and polydipsia might have similar physiopathologic mechanisms. Polydipsia might be due to anti-cholinergic side effect of some psychiatric drugs. The physiopathology of the polydipsia and polyuria is not totally enlightened in the psychiatric disorders. In some cases, the fluid intake occurs completely voluntary. Therefore, we decided to present this case.