

Diagnostic Methodology and Prevention in Internal Medicine and Common Neurobehavior: Target of Prevention of Cardiovascular Events and Metabolic Syndrome

Edson Detrigiachi^{1,2}, Jan Pagenotto Sukorski³, Marcelo Tadeu Marques de Oliveira⁴, Luciana Alencar Lima⁵ and Mario Luiz Furlanetto Junior^{6,7*}

¹Psychiatrist, University of São Paulo, Brazil

²Specialist in psychoanalytic theories and techniques, Paulista-Marília State University, Brazil

³Faculty of Medicine of Marília, General surgeon, University of Marília, Brazil

⁴Graduated in Physical Education, University of Marília, Brazil

⁵Nurse, Specialist in Urgency and Emergency, University of Marília, Brazil

⁶Doctor, General Surgeon, Faculty of Medicine of Marília, University of Marília, Brazil

⁷CEO and clinical director of Stakehold ZXSJ, Brazil

Citation: Luiz Furlanetto Junior, et al. (2023) Diagnostic Methodology and Prevention in Internal Medicine and Common Neurobehavior: Target of Prevention of Cardiovascular Events and Metabolic Syndrome. *Arch Cardiol* 2023;1(1): 5-11.

Received: 22 November, 2023; **Accepted:** 01 December, 2023; **Published:** 04 December, 2023

***Corresponding author:** Mario Luiz Furlanetto Junior, Doctor, General Surgeon, Faculty of Medicine of Marília, University of Marília, Brazil; CEO and clinical director of Stakehold ZXSJ, Brazil, E-mail: furlanettojr@hotmail.com

Copyright: © 2023 Luiz Furlanetto Junior, et al., This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

1. Introduction

Cardiovascular disease (CVD) continues to be the leading cause of global morbidity and mortality, which is fundamental to the ongoing need to identify new targets and complementary instruments for the prevention of new cardiovascular events (CVE)¹⁻³.

Over the past five years, the pooling of multiple data from large studies has accelerated progress in research on stress as a risk and prognostic factor for cardiovascular disease¹⁻⁴.

Currently, many studies of Adverse Childhood Experiences (CAE) and post-traumatic stress disorder (PTSD) in childhood have shown correlations in presenting and/or increasing the risk of multiple chronic conditions in adulthood, such as obesity, chemical dependence, immunoinflammatory diseases and even neoplasms¹⁻⁷.

Compared with childhood stress and the classic risk factors of adulthood, such as smoking, obesity, systemic arterial hypertension (SAH), dyslipidemia, and diabetes mellitus (DM), the harmful effects of stress in adulthood are generally less pronounced months¹⁻⁸. However, it is a diagnostic determinant,

as a trigger in many cases of CVE, in the presence of a high burden of atherosclerotic plaque triggering cerebrovascular accident (CVA) and acute myocardium infarction (AMI)¹⁻⁸.

Several mechanistic studies have corroborated previous laboratory observations of pathophysiological changes related to stress and Sd X, which are associated with the unfolding of the CVE, such as reduction of the arrhythmic threshold, increased activation Cortisol elevation, which cause sudden increases in blood pressure, and pro-inflammatory and pro-coagulant responses⁴⁻⁸.

Some metabolic and inflammatory syndromes and disorders present correlations of common factors, which potentiate their pathophysiology in Sd X, Polycystic Ovary Syndrome and Z Syndrome, the latter being referred to in association from obstructive sleep apnea (AOS) to metabolic syndrome⁶⁻⁸.

AOS is defined as a sleep disorder characterized by recurrent episodes of narrowing or collapse of the airways and pharyngeal during sleep, despite continuous respiratory efforts, and has also been shown to be a risk factor for cardiovascular consequences. It is usually found in association with various components of Sd X⁶⁻⁸.

Sd Z is little studied, it began in the late 90's, and was named by Wilcox, et al. The association of AOS risk factors with cardiovascular consequences is well established, as central obesity is a risk factor for both conditions. In the review by Carneiro, et al. they provided evidence that OSA is an independent risk factor for obesity, glucose intolerance, and insulin resistance⁶⁻¹⁰.

The mechanisms implicated in Sd Z result from the activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, activation of pro-inflammatory factors such as IL-6 and TNF- α ; and decreased adiponectin levels mediated mainly by intermittent hypoxemia related to apneas⁶⁻¹¹.

Common mental health disorders, such as depression and anxiety, and especially chronic stress, are highly prevalent and have a clear correlation with risk factors for CAD, since smoking is a psychiatric disease, and in many cases of cancer, the psychological factor is predominant and is the cause of refractoriness, in the treatment of bariatric surgeries and clinical treatments⁶⁻¹².

2. ZX Syndrome and Neuropsychological Component

Understanding the grouping of risk factors is of fundamental clinical importance in order to avoid underestimation of severity and for risk stratification of CVE. Although some guidelines already use stress as a prevention target for people at high risk of CVE or in known cardiovascular disease. Each component of the syndrome has significant effects on the cardiovascular system, so it is important to treat each individual component to reduce morbidity and mortality¹⁰⁻¹³.

In 2022, a description with the same term of Syndrome Z was published for the first time in a case report, to a clinical picture of NCCC (with only four neuro dysfunctions at the time), which has a causal link to substance use disorders, and other addictions, being secondary to the in some situations and/or simultaneous in others. In this case, the Z lens is referred to due to the zigzag format, which is observed in people with a clinical diagnosis, in three family generations, in the application of dialectical family neurobehavioral assessment¹³⁻¹⁶.

Although acquired mutations have historically been a focus of genomics in several chronic diseases, recent technological advances in phenotypic traits and regions of the Genome (GWAS), Blum, et al, with the fundamental development of Reward System Dysfunction Syndrome (RDS), and its various discoveries of polymorphisms of reward genes and addictions³⁻⁸.

Currently, many clinical investigations of genetics and cardiovascular disease focus on inherited genetic mutations, with little effective observation of the interactions between genes and the environment. When we focus on the environment, the neurobehavioral factor can be pathological due to the premise of being a triggering factor of refractoriness in some chronic diseases, especially when there is associated stress¹³⁻¹⁶.

The basic dissection that can be performed clinically, individualizing the neuroadaptation behaviors, which are all predominantly tonsillar in the family environment, is the same as in the professional environment, with the association of activation of the same tonsillar neuron system and mirror neurons, and Therefore, real situations of subtle stress are at work¹⁷⁻²³.

In behavioral and cognitive psychology, Young specifically described them as Family Schemas, and we perform clinical construction, analogous to Professional Schemas, when the

same neuro activations occur in an individual and a professional (family role), and then clinically express the same pictures of toxic and pathological neuro behaviors producing irrational behaviors, but which in clinical fact have the background of Family Schema, or NAA¹⁷⁻²⁴.

3. Clinical-Neurological Development

A Neurological Dysfunction common to all human brains, which presents behavioral, cognitive and epigenetic effects, clinically evidences the quality of a human relationship, is the bio behavior of Family Synchrony, in which genes and disorders of Oxytocin, Vasopressin, Melatonin and Cortisol were detected, which are associated with it bio behavior of Family Synchrony and functional deficits of parenting and the Newborn (NB)²¹⁻²⁹.

In addition to the fundamental role of uterine contraction and milk ejection, oxytocin participates in cardiovascular effects. The most well-known effects are the reduction of blood pressure (caused by natriuresis and release of atrial natriuretic peptides), negative inotropic and chronotropic effect due to parasympathetic neuromodulation, anti-stress effect and vasodilation mediated by activation of the nitric oxide pathway²³⁻³³.

The cases of hypertensive crises, and the acute and chronic complications caused by stress, whose pathophysiology is the cortisol disorder, are classic, and there are many neuropsychological cases of stress that complicates with cardiovascular events, such as cerebrovascular accident without detectable primary cause, and instability In the case of atherosclerotic plaques, in which Interleukin-6 (IL-6) is an active product both in stress and in plaque events, we must not forget that IL-6 acts on a different anatomical site, even if it has not been produced in the same anatomical region in which there is an EVC²⁴⁻³⁷. Post-traumatic stress disorder (PTSD) is a maladaptive and debilitating psychiatric disorder characterized by re-experience, avoidance, disturbances of emotional oscillations, especially in relation to fear, with distortions of negative thoughts, and central and peripheral neuronal hyperarousal, in the following months and even years, after exposure to severe trauma, and similar in the affected mechanism with loss and bereavement²⁴⁻³⁷.

The risk of developing PTSD due to the influence of hereditary genes is up to 40%, and the risk after severe trauma is determined by several factors, such as the presence of Adverse Childhood Experiences (AA), the presence of tonsillar neuroadaptations (NAA), and a history of Subclinical Parental Neglect (SPN), and we may suspect PTSD or SPL in the identification of ONCs²⁴⁻³⁷.

A large amount of evidence suggests that PTSD is a disorder that is associated with dysregulation of neurobiological fear disorders. The neural circuit underlying the behavior and learning related to fear and threat, is the prefrontal cortex system, with interference and maintenance of the immune system. behavioral and translational neuroscience³²⁻⁴³.

We hypothesize that we observe in the clinical practical environment, combined with the new findings of post-Covid-19 neurobiology studies, on themes of fear and family disaffection, that the initial dysfunction is the DNAF and triggers in response to all ONC, which are hormonal, genetic, and epigenetic, neuro inflammatory disorders, NAA dysfunctions during child development, which initially occur in the family environment and produce NCCC in the specific environments of the family and professional environments⁴¹⁻⁴⁹.

In the gestational phase, some maternal disorders and complications of childbirth may contribute as aggravating factors, associated with the sum of specific factors in childhood, such as AEI and PTSD, because they have the same neuro pathophysiology with clinical expression of causal link⁴¹⁻⁴⁹.

1. NFDNA due to the absence of neuro maturation, caused by the absence of stimulation from the biological parents.
2. Behaviors of NAA, which participates in the biological regulation of fear, through the tonsillar and limbic neurological systems (clinically observed by YOUNG's Family Schemas);
3. Pituitary axis dysfunction with disorders of Cortisol, Vasopressin, and Melatonin, which are responsible for the clinical changes in stress.
4. Dysfunction of family roles related to psychodynamics, with inversion, excess or absence of function of roles in the family system, which simultaneously reproduce the reality of family asynchrony inherited from parents, through mirror neurons and schema activation;
5. Dysfunctions of dopaminergic receptors specific to Reward System Dysfunction Syndrome (RDS) and their influencing genes described by Blum, et al.
6. Hypodopaminergic status oscillatory, chronic due to dysfunction of the primitive neurodopaminergic system SEEKING, which presents momentary symptoms, triggered by causal and aggravating factors such as loss, grief, EAI, PTSD and stress situations (Covid-19 for example)
7. Simultanagnosia secondary to hostile caregiver behaviors, caused by neuronal commissurotomy in the uncinate fasciculus.
8. States of Emotional Reactivity such as Alexithymia and Anosognosia secondary to pathological activated neurobehaviors, such as family schemas, automatisms, fear and stress.

Updating the clinical classification, individualizing the current RDS, which is previous in the sense of hierarchical etiopathogenesis to ADHD and ASD, presenting differences in clinical, genetic, and neuro pathophysiology. There are three segments of different pharmacological targets, therapies with different approaches, different evolution, although very similar⁴¹⁻⁴⁹.

NAA produces secondary *simultanagnosia* in childhood (inability to identify more than four objects in the same second) due to neuronal disconnection in the uncinate fascicle, caused by dysfunctional behavior of the parents in the child, mainly by psychological abuse carried out with verbal acts *alexithymia* (inability to identify effective self-observation), *Anosognosia* (inability to observe others effectively)⁴¹⁻⁴⁹.

In the ZXSYS Stakehold Diagnostic Methodology, the clinical presence of the set of eight (eighth) common neurodysfunctions (ONCs) is used as the initial and central connector, because they express typical reductionist NCCC, with a causal link to the clinical stress and fear, in addition to presenting common biological factors studied by Precision Medicine. The letter Z is used for ONCs, X for Metabolic SD, Y for associated psychiatric disorders and the letter S for associated immunoinflammatory diseases such as up too sclerosis, dermatitis and others⁴¹⁻⁴⁹.

4. Objective

The functional objective of this mini-review is to briefly bring to light an empirical finding of a new diagnostic construction to help other professionals, in which we did not present the initial objective of conducting scientific studies. After the finding of Sd Z or ONC in the practical field of addictions, the same patients with smoking addiction and obesity were identified.

5. Methodology

This study has ethnographic methodology drawn from the cardiovascular clinical environment and from the psychiatry of addictions, in which the clinical descriptions of pathological neuro behaviors, which were allied to the theories of behavioral and cognitive psychology, family neuro psychodynamics, schema therapy, dialectical behavioral therapy, Beck's cognitive therapy, and Freud's psychoanalytic technique.

The review of studies was selected for the convenience of several articles in the digital libraries PubMed, Way os Science, Scielo, where we captured 3221 scientific articles, and 2,299 articles were excluded, for example. did not present clinical data on neuro behavior and neuro genetics, totaling 112 articles, initiated in the period from December 10, February 20, 20 to 30, 2023.

We used the terms attachment, parental neglect, PTSD, dopamine, neurobiology, addictions, obesity, EAI, and for convenience the studies by Blum, et al. on RDS.

The self-comparison without judgment is technical and sequential, initially evaluating the behaviors that predominate the objective factor of the brain, to the psychological factor, because only in this way was it possible to individualize the methodology of neuro-reductionist clinical evaluation in the family environment, in which the individualization of the objective (neurological) intimate component of the brain is evident. subjective (personal, intimate, essential). Initially, we classified it as dialectical family neurobehavioral assessment.

This study was not planned as the scientific tradition, but we believe it has value of paramount importance, because it is evident that we do not performs ophisms, guesswork, selective or malicious feedback bias or misinterpretation of data, because ONCs is a clinical and cognitive neurobehavioral truth, present in a large portion of the world population, and the intention is to evidence a new medical perspective, to assist the various specialists

6. Discussion

Currently, there are several concepts, terms and similar disorders that cause confusion and limitation in the medical and non-medical areas, in which the objects are a disease and a patient, and the clinical importance of such objects is varied by conceptual disorganization, alliance deficits or effective interlocution between human scientific knowledge⁴³⁻⁵⁴.

By applying the common dialectical neurobehavioral assessment observing the behavioral problems of the hypodopaminergic antecedents, in the case described below in a study by Blum, et al. using the term "Generational Family Syndrome", it is possible to make a clinical diagnosis based on pathophysiology³⁻⁴⁹:

The family consists of a mother, father, son and daughter. The mother had problems with focus, memory, anger, and

*a motivational syndrome. Her father had weight problems and depression. The son experienced heavy drinking, along with some drug abuse and anxiety. The daughter experienced depression, lethargy, brain fog, trouble focusing, and anxiety, among others*³⁻⁴⁹.

The mother presented hypo dopaminergic decompensation, and the father developed symptoms and diseases secondary to Sd Z, such as depression and obesity or Sd ZX. The daughter repeats her mother's pattern, showing an asynchronous family relationship, such as Sd Z, as the final effect⁴⁹⁻⁵⁹.

A systematic review and meta-analysis of a study of the prevalence of Sd X in adolescents in Brazil found high heterogeneity among the studies. Kuschner, et al. He attributed it to different eating habits and lifestyles, since these are the main factors in the genesis of obesity, in addition to representing a central component in the diagnosis, it is a disease that increases in several countries⁶⁰⁻⁷².

However, lifestyle is directly associated with the personality molded, consciously or not, at home, by the parents. And the family relationship shapes psychologically on foundations of beliefs and values, but concomitantly organically on epigenetic changes. Neuroadaptations, neuroplasticity and some authors show processes of inflammation⁶⁰⁻⁷³.

The family habit is directly related to mood, relief from moments of distress, that hypo dopaminergic state, or a psychological factor is present. And the habit crystallizes into addiction, as evidenced by studies of food and sugar addictions, which is a problem for diabetics⁷³⁻⁷⁷.

Recently, pre-addiction has been hypothesized for diabetes, without developmental evolution, and certainly ONC are the beginning for new studies of prevention, treatment of diabetes mellitus with emotional decompensation and obesity refractory to clinical treatment, being an important ally to the current treatments of Relapse for Obesity and Maintenance of diets negatively influenced by the emotional factor⁷³⁻⁷⁷. Years This study does not present a classic scientific clinical methodology, so the justification of little psychological rigor should not be underestimated, since syndrome Z is literally an extension of Metabolic DS, or vice versa in many clinical cases⁷³⁻⁷⁷.

7. Conclusion

We ignore the fact that patients with metabolic SD should be evaluated for the presence of ONC or ZX syndrome, especially in cases of childhood obesity. In the professional spheres of mental health and pediatrics, clinical screening for Sd X is extremely important. The identification of ONCs that express NCCC in a universal way can generate protocols in several dimensions that help improve the economy, crime, domestic violence, control and prevention of chronic diseases, new pharmacological targets, acceleration of new scientific studies, public health policies, and technological and social advances⁷⁷⁻⁹⁶.

However, longitudinal population-based studies are needed to prove the causal relationship of ONC in relation to metabolic and inflammatory disorders of Sd Z and Sd X, as well as multicenter randomized, well-controlled sampling studies considering common clustering of ONC, and confirmation of the beneficial effect of Reductionist psychotherapy⁹³⁻¹⁰¹.

8. References

1. Kivimäki M, Steptoe A (2018) Effects of stress on the development and progression of cardiovascular disease. *Nat Rev Cardiol*, 15: 215-229.
2. Ressler KJ, Berretta S, Bolshakov VY, et al. (2022) Post-traumatic stress disorder: Clinical and translational neuroscience from cells to circuits. *Nat Rev Neurol*, 18: 273-288.
3. Arciniegas H, Hernandez G, Suarez et al. (2017). Neuropsychological profile of young adult patient with Z syndrome. *Chilean Journal of Neuropsychology*, 12: 48-53.
4. Marcelo Febo, Kenneth Blum, Rajendra D Badgaiyan, et al. (2017) Dopamine homeostasis brain functional connectivity in reward deficiency syndrome. *Frontiers in Bioscience*, 22: 669-691.
5. K Blum, A Bowirrat, D Baron, et al. (2020) Biotechnical development of genetic dependence risk score (GARS) and selective evidence for inclusion of polymorphic allelic risk in substance use disorder (SUD). *J Syst Integr Neurosci*, 6: 15761.
6. Kenneth Blum, Marlene Oscar-Berman, Zsolt Demetrovics, et al. (2014) Genetic addiction risk score (GARS): Molecular neurogenetic evidence for predisposition to reward deficiency syndrome (RDS). *Mol Neurobiol*, 50: 765-796.
7. Kenneth Blum, Thomas J H Chen, B William Downs, et al. (2009) Neurogenetics of dopaminergic receptor supersensitivity in activation of brain reward circuitry and relapse: Proposing deprivation-amplification relapse therapy (DART). *Postgrad Med*, 121: 176-196.
8. Cicekliyurt MM, Dermenci B (2022) Relationship between oxytocin receptor gene polymorphism and hypertension in the Turkish population. *Rev Porto Cardiol*, 17:870-2551.
9. Venkateswaran S, Shankar P (2007) The prevalence of Z syndrome (the interaction of obstructive sleep apnea with metabolic syndrome) in a teaching hospital in Singapore. *Postgraduate Med J*, 83: 329-331.
10. Eric Afuseh, Caitlin A Pike, Ukamaka M Oruche (2020) Individualized approach to primary prevention of substance use disorder: Age-related risks. *Subst Abuse Treat Prev Policy*, 15: 58.
11. Volkow ND, Wang GJ, Fowler JS, et al. (2011) Addiction: Beyond dopamine reward circuitry. *Proc Natl Acad Sci U S A*, 108: 15037-15042.
12. Hossein Akbari, Mohsen Roshanpajouh, Keramat Nourijelyani, et al. (2019) Profile of drug users in the residential treatment centers of Tehran, Iran. *Health Promot Perspect*, 9: 248-254.
13. AL'ABSI, Mustafa, GINTY Annie T, LOVALLO, William R (2021) Neurobiological mechanisms of early life adversity, blunted stress reactivity and risk for addiction. *Neuropharmacology*, 188: 08519.
14. Carneiro G, FH Fonts, Togeiro SMGP (2010) Untreated metabolic consequences in OSAS. *J Bras Pulmonology*, 36: 1-61.
15. Alammehrjerdi Z (2018) Methamphetamine dependence in methadone treatment services in Iran: The first literature review of a new health problem. *Asian Journal of Psychiatry*, 31: 49-55.
16. Albrecht U, Kirschner NE, Grüsser SM (2007) Diagnostic instruments for behavioural addiction: An overview. *Psychosoc Med*, 4: 1-11.
17. Ray Alsuhaibani, Douglas Cary Smith, Richard Lowrie, et al. (2021) Scope, quality, and uniqueness of international clinical guidelines on mental health and substance abuse in relation to dual diagnosis, social and community outcomes: A systematic review. *BMC* 21: 209.

18. WA Anderson, C Burnett (1979) Reproductive tract peroxidases as endproducts of estrogen-specific gene expression. *J Histochem Cytochem*, 27: 1363-1364.
19. Helle Wessel Andersson, Anders D Forsmo Lauvsnes, Trond Nordfjærn (2021) Emerging adults on inpatient substance use treatment: A prospective cohort study of patient characteristics and treatment outcomes. *Eur Addict Res*, 27: 206-215.
20. Timothy R Apodaca, Richard Longabaugh (2009) Mechanisms of change in motivational interviewing: A review and preliminary evaluation of the evidence. *Addiction*, 104: 705-715.
21. Pietro Avanzini, Maddalena Fabbri-Destro, Riccardo Dalla Volta, et al. (2012) The dynamics of sensorimotor cortical oscillations during the observation of hand movements: An EEG study. *PLoS ONE*, 7: e37534.
22. Michael T Baglivio, Kevin T. Wolff, Alex R Piquero, et al. (2015) The relationship between adverse childhood experiences (ACE) and juvenile offending trajectories in a juvenile offender sample. *Journal of Criminal Justice*, 43: 229-241.
23. https://www.uniad.org.br/wp-content/uploads/2013/10/Addictions_Neurobiology_final.pdf
24. Maxime Billot, Maeva Daycard, Chantal Wood, et al. (2019) Reiki therapy for pain, anxiety and quality of life. *BMJ Support Palliat Care*, 9: 434-438.
25. Birine MT, Kooiker CL, Short AK, et al. (2020) Plasticity of the Reward Circuitry After Early-Life Adversity: Mechanisms and Significance. *Biol Psychiatry*, 87: 875-884.
26. Blanco C, Moreyra P, Nunes EV, et al. (2001) Pathological gambling: Addiction or compulsion? *Semin Clin Neuropsychiatry*, 6: 167-176.
27. Blodgett JC, Maisel NC, Fuh IL, et al. (2014) How effective is continuing care for substance use disorders? A meta-analytic review. *J Subst Abuse Treat*, 46: 87-97.
28. Bowen S, Witkiewitz K, Clifasefi SL, et al. (2014) Relative Efficacy of Mindfulness-Based Relapse Prevention, Standard Relapse Prevention, and Treatment as Usual for Substance Use Disorders: A randomized clinical trial. *JAMA Psychiatry*, 71: 547-556.
29. Braun TD, Kunicki ZJ, Blevins CE, et al. (2021) Prospective Associations between Attitudes toward Sweet Foods, Sugar Consumption, and Cravings for Alcohol and Sweets in Early Recovery from Alcohol Use Disorders. *Alcohol Treat Q*, 39: 269-281.
30. Brockie TN, Dana-Sacco G, Wallen GR, et al. (2015) The Relationship of Adverse Childhood Experiences to PTSD, Depression, Poly-Drug Use and Suicide Attempt in Reservation-Based Native American Adolescents and Young Adults. *Am J Community Psychol*, 55: 411-421.
31. Burns GL, Geiser C, Servera M, et al. (2019) Application of the Bifactor S - 1 Model to Multisource Ratings of ADHD/ODD Symptoms: An Appropriate Bifactor Model for Symptom Ratings. *J Abnorm Child Psychol*, 48: 881-894.
32. Cai C, Yuan K, Yin J, et al. (2015) Striatum morphometry is associated with cognitive control deficits and symptom severity in internet gaming disorder. *Brain Imaging Behav*, 10: 12-20.
33. Chambers H, Amos J, Allison S, et al. (2006) Parent and Child Therapy: An Attachment-Based Intervention for Children With Challenging Problems. *Australian and New Zealand Journal of Family Therapy*, 27: 68-74.
34. Chen J, Paul JM, Reeve R (2022) Manipulation of attention affects subitizing performance: A systematic review and meta-analysis. *Neurosci Biobehav Rev*, 139: 104753.
35. Chew HSJ (2022) The Use of Artificial Intelligence-Based Conversational Agents (Chatbots) for Weight Loss: Scoping Review and Practical Recommendations. *JMIR Med Inform*, 10: e32578.
36. Clark DA, Beck AT (2010) Cognitive theory and therapy of anxiety and depression: Convergence with neurobiological findings. *Trends Cogn Sci*, 14: 418-424.
37. Clark L, Boileau I, Zack M (2018) Neuroimaging of reward mechanisms in Gambling disorder: an integrative review. *Mol Psychiatry*, 24: 674-693.
38. Claudat K, Brown TA, Anderson L, et al. (2020) Correlates of co-occurring eating disorders and substance use disorders: a case for dialectical behavior therapy. *Eat Disord*, 28: 142-156.
39. Bethesda (2020) Common comorbidities with substance use disorders research report. national institutes on drug abuse.
40. Constant A, Moirand R, Thibault R, et al. (2020) Meeting of Minds around Food Addiction: Insights from Addiction Medicine, Nutrition, Psychology, and Neurosciences. *Nutrients*, 12: 3564.
41. Correa MF, Junior OFP (2019) Emotional affects according to Panksepp, compared with Damasio and observational materialism. In: ALVES MA (Org.). *Cognition, emotions, and action*. São Paulo: Cultura Acadêmica; UNICAMP; Center for Logic, Epistemology and History of Science, 84: 279-310.
42. Cossin T, Thon I, Lalanne L (2021) Workaholism Prevention in Occupational Medicine: A Systematic Review. *Int J Environ Res Public Health*, 18: 7109.
43. Donges US, Suslow T (2017) Alexithymia and automatic processing of emotional stimuli: a systematic review. *Rev Neurosci*, 28: 247-264.
44. Donohue B, Azrin NH, Bradshaw K, et al. (2014) A controlled evaluation of family behavior therapy in concurrent child neglect and drug abuse. *J Consult Clin Psychol*, 82: 706-720.
45. Durand-Moreau Q, Deun CLE, Lodde B, et al. (2018) The framework of clinical occupational medicine to provide new insight for workaholism. *Ind Health*, 56: 441-451.
46. Feng C, Li Z, Feng X, et al. (2015) Social hierarchy modulates neural responses of empathy for pain. *Soc Cogn Affect Neurosci*, 11: 485-495.
47. Flores-Bonilla A, Richardson HN (2020) Sex Differences in the Neurobiology of Alcohol Use Disorder. *Alcohol Res*, 40: 4.
48. Forster GL, Anderson EM, Scholl JL, et al. (2018) Negative consequences of early-life adversity on substance use as mediated by corticotropin-releasing factor modulation of serotonin activity. *Neurobiology of Stress*, 9: 29-39.
49. Forster M, Grigsby TJ, Rogers CJ, et al. (2018) The relationship between family-based adverse childhood experiences and substance use behaviors among a diverse sample of college students. *Addictive Behaviors*, 76: 298-304.
50. Garfinkel SN, Seth AK, Barrett AB, et al. (2015) Knowing your own heart: Distinguishing interoceptive accuracy from interoceptive awareness. *Biol Psychol*, 104: 65-74.
51. Gastfriend DR, Rubin A, Sharon E, et al. (2003) New constructs and assessments for relapse and continued use potential in the ASAM Patient Placement Criteria. *J Addict Dis*, 22: 95-111.
52. Ghouchani HT, Lashkardoost H, Saadati H, et al. (2021) Developing and validating a measurement tool to self-report perceived barriers in substance use treatment: the substance use treatment barriers questionnaire (SUTBQ). *Substance Abuse Treatment, Prevention, and Policy*, 16.
53. Gliksman Y, Henik A (2019) Enumeration and Alertness in Developmental Dyscalculia. *J Cognition*, 2: 5.
54. Goldstein RZ, Volkow ND (2011) Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nature Reviews Neuroscience*, 12: 652-669.
55. Gonzales R, Anglin MD, Beattie R, et al. (2012) Understanding Recovery Barriers: Youth Perceptions About Substance Use Relapse. *Am J Health Behav*, 36: 602-614.

56. Grant S, Colaiaco B, Motala A, et al. (2017) Mindfulness-based Relapse Prevention for Substance Use Disorders: A Systematic Review and Meta-analysis. *J Addict Med*, 11: 386-396.
57. Gregorowski C, Seedat S, Jordaan GP (2013) A clinical approach to the assessment and management of co-morbid eating disorders and substance use disorders. *BMC Psychiatry*, 13: 289.
58. Grummitt L, Kelly E, Barrett E, et al. (2021) Targets for intervention to prevent substance use in young people exposed to childhood adversity: A systematic review. *PLOS ONE*, 16: e0252815.
59. Hagedorn HJ, Noorbaloochi S, Simon AB, et al. (2013) Rewarding early abstinence in Veterans Health Administration addiction clinics. *J Subst Abuse Treat*, 45: 109-117.
60. Hakobyan S, Vazirian S, Lee-Chong S, et al. (2020) Concurrent Disorder Management Guidelines. Systematic Review. *J Clin Med*, 9: 2406.
61. Hallgren KA, Moyers TB (2011) Does readiness to change predict in-session motivational language? Correspondence between two conceptualizations of client motivation. *Addiction*, 106: 1261-1269.
62. Hamann S (2005) Sex Differences in the Responses of the Human Amygdala. *Neuroscientist*, 11: 288-293.
63. He Z, Jiang Y, Gu S, et al. (2021) The Aversion Function of the Limbic Dopaminergic Neurons and Their Roles in Functional Neurological Disorders. *Front Cell Deve Biol*, 9: 713762.
64. Herzmann G, Young B, Bird CW, et al. (2012) Oxytocin can impair memory for social and non-social visual objects: A within-subject investigation of oxytocin's effects on human memory. *Brain Res*, 1451: 65-73.
65. Hildebrandt MK, Dieterich R, Endrass T (2021) Neural correlates of inhibitory control in relation to the degree of substance use and substance-related problems - A systematic review and perspective. *Neurosci Biobehav Rev*, 128: 1-11.
66. Ikemoto S (2007) Dopamine reward circuitry: Two projection systems from the ventral midbrain to the nucleus accumbens-olfactory tubercle complex. *Brain Research Reviews*, 56: 27-78.
67. Ionio C, Ciuffo G, Landoni M (2021) Parent-Infant Skin-to-Skin Contact and Stress Regulation: A Systematic Review of the Literature. *Int J Environ Res Public Health*, 18: 4695.
68. Jansson LM, Patrick SW (2019) Neonatal Abstinence Syndrome. *Pediatr Clin North Am*, 66: 353-367.
69. Jaswetz L, Voogd LD, Becker ES, et al. (2022) No evidence for disruption of reconsolidation of conditioned threat memories with a cognitively demanding intervention. *Scientific Reports*, 12.
70. Jaynes KD, Gibson EL (2017) The importance of nutrition in aiding recovery from substance use disorders: A review. *Drug and Alcohol Dependence*, 179: 229-239.
71. Junyue J, Siyu C, Xindong W, et al. (2021) Complementary and Alternative Medicine for Substance Use Disorders: A Scientometric Analysis and Visualization of Its Use Between 2001 and 2020. *Front Psychiatry*, 12: 722240.
72. Kelly JF, Humphreys K, Ferri M (2020) Alcoholics Anonymous and other 12-step programs for alcohol use disorder. *Cochrane Database of Syst Rev*, 3.
73. Knight DK, Yang Y, Joseph ED, et al. (2021) Preventing opioid use among justice-involved youth as they transition to adulthood: leveraging safe adults (LeSA). *BMC Public Health*, 21: 2133.
74. Kobis NC, Verschuere B, Shalvi S, et al. (2019) Intuitive Honesty Versus Dishonesty: Meta-Analytic Evidence. *Perspectives on Psychological Science*, 14: 778-796.
75. Koehler S, Hasselmann E, Wustenberg T, et al. (2013) Higher volume of ventral striatum and right prefrontal cortex in pathological gambling. *Structure Brain and Function*, 220: 469-477.
76. Koob GF, Volkow ND (2016) Neurobiology of addiction: a neuro-circuitry analysis. *Lancet Psychiatry*, 3: 760-773.
77. Kube T, Rozenkrantz L (2020) When Beliefs Face Reality: An Integrative Review of Belief Updating in Mental Health and Illness. *Perspectives Psychol Sci*, 16: 247-274.
78. Kuntz A, Missonnier P, Prevot A, et al. (2021) Persistence of Neuronal Alterations in Alcohol-Dependent Patients at Conclusion of the Gold Standard Withdrawal Treatment: Evidence From ERPs. *Front Psychiatry*, 12.
79. Kuss DJ, Bridges HM, Griffiths MD (2018) Neurobiological Correlates in Internet Gaming Disorder: A Systematic Literature Review. *Front Psychiatry*, 9: 166.
80. Labar KS, Cabeza R (2006) Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience*, 7: 54-64.
81. Leibenluft E, Gobbin MI, Harrison T, et al. (2004) Mothers' neural activation in response to pictures of their children and other children. *BIOL PSYCHIATRY*, 56: 225-232.
82. Lennox R, Dennis ML, Scott CK, et al. (2006) Combining psychometric and biometric measures of substance use. *Drug and Alcohol Dependence*, 83: 95-103.
83. Revach D, Salti M (2022) Consciousness as the Temporal Propagation of Information. *Front Syst Neurosci*, 16: 759683.
84. Li MT, Zhang J, Zhang DC, et al. (2021) Development and Psychometric Properties of the Synthetic Drug Dependence Scale in a Chinese Sample. *Front Psychol*, 12.
85. Li X, Caprioli D, Marchant NJ (2015) Recent updates on incubation of drug craving: a mini-review. *Addict Biol*, 20: 872-876.
86. Li X, Weissman M, Talati A, et al. (2019) A diffusion tensor imaging study of brain microstructural changes related to religion and spirituality in families at high risk for depression. *Brain and Behav*, 9: e01209.
87. Liu Y, Bressel JRD, Yau JOY, et al. (2020) The Mesolimbic Dopamine Activity Signatures of Relapse to Alcohol-Seeking. *J Neurosci*, 40: 6409-6427.
88. Liu Y, McNally GP (2021) Dopamine and relapse to drug seeking. *Journal of Neurochemistry*, 157: 1572-1584.
89. Lyons-Ruth K, Elisa B, Gwendolyn A (1999) A relational diathesis model of hostile-helpless states of mind: Expressions in mother-infant interaction. In: Solomon J, George C (Eds.). *Attachment disorganization*. New York, Guilford Press, 33-70.
90. Marraudino M, Bonaldo B, Vitiello B, et al. (2022) Sexual Differences in Internet Gaming Disorder (IGD): From Psychological Features to Neuroanatomical Networks. *J Clin Med*, 11: 1018.
91. Mathers BM, Degenhardt L, Bucello C, et al. (2013) Mortality among people who inject drugs: a systematic review and meta-analysis. *Bull World Health Organ*, 91: 102-123.
92. Mccusker J, Vickers-Lahti M, Stoddard A, et al. (1995) The effectiveness of alternative planned durations of residential drug abuse treatment. *Am J Public Health*, 85: 1426-1429.
93. McGovern MP, Wisley BR, Drake RE (2005) Special Section on Relapse Prevention: Relapse of Substance Use Disorder and Its Prevention Among Persons With Co-occurring Disorders. *Psychiatr Serv*, 56: 1270-1273.
94. Mckay JR (2021) Impact of Continuing Care on Recovery From Substance Use Disorder. *Alcohol Res*, 41: 1.
95. Nassar MF, Younis NT, El-Arab SE, et al. (2010) Neuro-developmental outcome and brain-derived neurotrophic factor level in relation to feeding practice in early infancy. *Matern Child Nutr*, 7: 188-197.

96. Nestler EJ, Barrot M, Self DW (2001) DeltaFosB: A Sustained Molecular Change for Addiction. *Proc Natl Acad Sci U S A*, 98: 11042-11046.
97. Pando-Naude V, Toxtó S, Fernandez-Lozano S, et al. (2021) Gray and white matter morphology in substance use disorders: a systematic neuroimaging review and meta-analysis. *Transl Psychiatry*, 11:29.
98. Pettorruso M, Zoratto F, Miuli A, et al. (2020) Exploring dopaminergic transmission in gambling addiction: A systematic translational review. *Neurosci Biobehav Rev*, 119: 481-511.
99. Poncet M, Caramazza A, Mazza V (2016) Individuation of objects and object parts rely on the same neuronal mechanism. *Scientific Reports*, 6.
100. Prom-Wormley EC, Ebejer J, Dick DM, et al. (2017) The genetic epidemiology of substance use disorder: A review. *Drug Alcohol Depend*, 180: 241-259.
101. Kelly Quinn, Bartley C Frueh, Joy Scheidell, et al. (2019) Internalizing and externalizing factors on the pathway from adverse experiences in childhood to non-medical prescription opioid use in adulthood. *Drug Alcohol Depend*, 197: 212-219.