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Transcranial Direct Current Stimulation in the Treatment of Autism: Literature Review

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1. ABSTRACT

Transcranial Electrical Stimulation (tES) is a non-invasive brain stimulation technique used to modulate neuronal activity through the application of weak electrical currents (1mA to 2 mA) to the scalp. Unlike Transcranial Magnetic Stimulation (TMS), tES is administered during task execution, as it more specifically influences the neurons involved in the task, thereby altering responses. The method includes various stimulation protocols such as Transcranial Direct Current Stimulation (tDCS), Transcranial Alternating Current Stimulation (tACS), and Transcranial Random Noise Stimulation (tRNS), each differing in the type of current delivered. tDCS, the most commonly used protocol, applies a continuous current, inducing depolarization (excitatory effects) with anodic stimulation or hyperpolarization (inhibitory effects) with cathodic stimulation. This technique is widely used for cognitive enhancement and produces both short-term and long-term effects on synaptic plasticity. tACS, on the other hand, utilizes alternating currents to synchronize neuronal oscillations, acting specifically on brain regions at defined frequencies. Lastly, tRNS delivers randomized currents within a frequency range, leveraging stochastic resonance to enhance cortical excitability and improve performance in concurrent motor or cognitive tasks. tRNS is also associated with prolonged aftereffects and is less discomforting for subjects. Safety guidelines for tES include limiting current density, electrode type, and duration (maximum 2 mA for 20 minutes) to prevent tissue damage. Exclusion criteria apply to individuals with metallic implants, neurological conditions (e.g., epilepsy), or certain medications. This study highlights the mechanisms, protocols, applications, and safety parameters of tES, offering insights into its potential for modulating brain activity and enhancing cognitive and motor performance.

2. INTRODUCTION

Autism spectrum disorders are pervasive developmental disorders characterized by atypical behavioural features. The term “spectrum” refers to the distribution and variability of problematic behaviours over time and intensity, meaning that autism encompasses individuals with heterogeneous clinical characteristics. Each person with autism is unique, as there are multiple combinations of this syndrome, presenting similar structural manifestations but differing in intensity, quality, and quantity of symptoms.

In autism, non-invasive brain stimulation techniques focus on symptom remission and are used to modulate neuronal activity in specific brain areas responsible for atypical behaviours. These methods include transcranial magnetic stimulation and transcranial electrical stimulation.

3. DEFINITION OF AUTISM SPECTRUM DISORDERS

Autism Spectrum Disorders (ASD) are recognized as among the most prevalent neurodevelopmental disorders. Since the 1960s, epidemiological studies on ASD have increased, highlighting a substantial rise in these disorders in the general population, largely attributable to the use of more precise diagnostic tools. ASD has an incidence of 1% to 1.5% among children, making it a frequent condition in child neuropsychiatry [1].

These disorders typically arise around the third year of life, tend to impair the individual's functioning across social and communication domains, and are characterized by a repetitive, limited, and stereotyped repertoire of activities and interests [2]. They are defined by developmental deficits that cause impairments in personal, social, academic, or occupational functioning. ASD belongs to neurodevelopmental disorders-conditions that arise during the developmental period and tend to manifest before school age.

ASD is biologically determined and diagnosed based on behavioural symptoms that appear within the first three years of life, with a wide and varied presentation, especially evident in children before or after two years of age. The range of developmental deficits varies from very specific learning or executive function limitations to global impairments of social skills or intelligence. ASD frequently co-occurs with other disorders; for example, individuals with ASD often have intellectual disability (developmental intellectual disorder).

In some disorders, the clinical picture includes both excess symptoms and deficits or delays in reaching expected developmental milestones. For instance, ASD is diagnosed only when the characteristic deficits in social communication are accompanied by excessively repetitive behaviours and a restricted range of interests.

It is important to emphasize that, although these disorders are structurally similar, they differ in quality, quantity, and intensity.

Given that autism is a heterogeneous disorder with multiple causes and varying symptom severity, three different severity levels are specified. Severity is based on the degree of impairment in social communication and restricted, repetitive behaviour patterns (DSM-5):

- Level 1 (Requiring Support): The individual has difficulty initiating social interactions and shows clear atypical or unsuccessful responses to social overtures. There is reduced interest in social interaction; without support, deficits in communication are noticeable. Restricted interests and repetitive behaviours interfere significantly in one or more contexts, with difficulty switching between activities and problems with organization and planning that impede personal autonomy.
- Level 2 (Requiring Substantial Support): Marked deficits in verbal and nonverbal social communication skills are apparent even with support. Social interaction is limited, and responses to social approaches are reduced or abnormal. There are frequent fixed rituals and/or repetitive behaviours that interfere with functioning in various contexts, behavioural inflexibility, and significant difficulty coping with change. Stress and frustration increase when routines are interrupted, and redirecting attention is challenging.
- Level 3 (Requiring Very Substantial Support): Severe deficits in verbal and nonverbal social communication skills cause major impairments in functioning. Social initiation is very limited, and

responses to others are minimal. Concerns, fixed rituals, and repetitive behaviours markedly interfere with functioning in all areas. Stress is heightened when rituals or routines are interrupted, making it very difficult to redirect attention or action from a fixated interest.

The etiopathogenesis of these disorders is heterogeneous and multifactorial, involving various factors that primarily affect the central nervous system during development. Prenatal factors include infectious diseases such as congenital rubella (causing brain damage in 5% to 16% of ASD cases), congenital syphilis, cytomegalovirus infection, or toxoplasmosis [3]. Perinatal factors may include brain trauma or hypoxic-haemorrhagic syndrome. Postnatal factors can be infectious or traumatic.

Although autism is behaviourally defined, it is now well recognized as the endpoint of various organic etiologies, including prenatal insults like rubella infection, untreated metabolic disorders such as phenylketonuria, anticonvulsant use during pregnancy, localized lesions (e.g., tuberous sclerosis), and postnatal infections like encephalitis. However, a specific medical cause is found in only a minority of people with autism (6% to 10% depending on the study), more frequently in those with pronounced learning difficulties.

4. NON-INVASIVE BRAIN STIMULATION (NIBS) TECHNIQUES

It is possible to stimulate the brain without the need for deep electrodes [4]. Non-invasive brain stimulation methods modulate the activity of specific areas by activating or inhibiting neuronal excitability thresholds or discharge [5]. These techniques influence neuronal states by altering the membrane potential. Normally, the membrane potential is stable at rest, but NIBS can modify it using electric currents that facilitate or inhibit action potential generation. All these methods can shift cellular charges, modifying membrane potential.

Among the most used techniques are Transcranial Magnetic Stimulation (TMS), which is based on neuronal membrane depolarization *via* electromagnetic induction to trigger action potentials in the stimulated area, and transcranial electrical stimulation (tES), which includes all NIBS methods using weak transcranial currents to modulate brain neuronal activity. These low-intensity stimulations induce changes in membrane potential, alter ionic flows, and modulate neuronal response thresholds, thus modulating neuronal response [6].

TMS and tES differ in that TMS is a neurostimulation technique capable of evoking action potentials (depending on whether stimulation is supra- or sub-threshold), while tES are neuromodulation techniques, modulating postsynaptic potentials but not directly triggering action potentials.

To precisely localize the target area, neuronavigation systems have been introduced [6], guiding TMS based on individual MRI data. The coil is positioned over the target area according to the patient's anatomical images. In the absence of individual data, some systems use a template-a model of an average brain obtained by digital image manipulation (warping). Neuronavigation allows for precise targeting and reproducibility in repeated or prolonged stimulation sessions [6].

5. TRANSCRANIAL ELECTRICAL STIMULATION

So far, we will discuss later in part II about magnetic stimulation techniques. Now we will focus on electrical stimulation methods. Transcranial Electrical Stimulation (tES), unlike Transcranial Magnetic Stimulation

(TMS), is administered during the execution of a task because it more significantly influences the neurons involved in that task, thereby altering the response. tES encompasses all methods of Non-Invasive Brain Stimulation (NIBS) that utilize weak transcranial electrical currents (approximately 1 to mA 2 mA) applied to the scalp for a duration of a few minutes (around 5-20 minutes) through the use of a pair of electrodes known as the anode (positive), which delivers the current, and the cathode (negative), which serves as the return electrode. These currents generate an electric field that modulates neuronal activity based on the application protocol. In this context, we refer to electricity delivery parameters that can be continuous (tDCS, Transcranial Direct Current Stimulation), alternating (tACS, Transcranial Alternating Current Stimulation), or randomized (tRNS, Transcranial Random Noise Stimulation) [5,6]. Guidelines regarding safety must be followed to perform stimulation *via* tES, aimed at avoiding damage to the stimulated tissues. For instance, it is important to limit the duration of stimulation using a minimal current density and surface electrodes that do not cause skin damage. A safe stimulation can be achieved by using a protocol with a stimulation intensity of 2 mA for a maximum duration of 20 minutes. There are also exclusion criteria that prevent stimulation of individuals with metallic implants in the brain, those who have undergone neurosurgical interventions, or those exhibiting signs of epilepsy or using antiepileptic medications, as well as individuals with febrile seizures. Additionally, subjects taking neuroleptics, benzodiazepines, antidepressants, or those suffering from migraines are also excluded. Stimulation techniques are often associated with the tDCS protocol, as it is the most commonly used, including for cognitive enhancement. tDCS involves the application of currents of 1 mA to 2 mA in a continuous manner, always following the same direction. The current delivery device determines the intensity and duration of the delivery. Depending on the polarity of the electrodes, depolarization of the targeted area can be induced through anodic current, while cathodic current hyperpolarizes the neuronal membrane, resulting in inhibition of the underlying cortex. The application of anodic and cathodic currents produces Meta plasticity, which refers to activity-dependent changes in neural functions that modulate subsequent synaptic plasticity, such as Long-Term Potentiation (LTP) and Long-Term Depression (LTD). The long-term effects depend on synaptic efficacy, while the short-term effects of tDCS depend on the polarization of the neuronal membrane [6,7]. The excitability of an area or its response threshold can increase or decrease depending on whether anodic or cathodic current parameters are used. Neurons involved in task execution while being stimulated by tDCS are more readily activated with anodic current [6]. with constant stimulation, phenomena of neurophysiological homeostasis may arise, returning neural activity to a normal functional level. Depending on the task involving the stimulated neural network, reactions will be influenced by their activation state [6]. The difference between tDCS and tACS lies in the fact that tDCS is non-specific, meaning that the applied currents diffuse in all directions following the path of least resistance. It is a stimulation in which current is delivered from one pole to another, where one pole is excited and the other inhibited. In contrast, tACS involves two poles that are out of phase, being continuously alternated. Compared to tDCS, tACS has a more potent effect as it acts specifically, facilitating or interfering with a particular area only at a specific oscillation frequency of that area, with a frequency range varying from 0.1 Hz to 1000 Hz, thereby inducing alterations in brain oscillations and modifying neuronal activity in that area along with related behaviours. These current oscillations produce an entrainment phenomenon in the underlying cortex, meaning a synchronization that induces neurons to oscillate at the same frequency. The excitatory or inhibitory effect thus depends on the frequency applied and how that area is polarized at that moment. The last paradigm of tES is tRNS, where the oscillation frequency changes

randomly within an oscillation spectrum between 0.1 Hz and 1000 Hz. Using the phenomenon of stochastic resonance (the phenomenon whereby an optimal background noise allows for the amplification of some signals that are too weak, even sub-threshold) [5], the functioning of the cortex is modulated by exciting the stimulated area. A current is applied randomly with variable intensity and within a certain frequency range. When applied during the execution of a concurrent motor or cognitive task, tRNS enhances performance by increasing cortical excitability, with an effect similar to anodic tDCS. In this case, the aftereffect of tRNS persists after the end of stimulation, thus producing a long-term change in synaptic efficacy. Furthermore, tRNS is found to be less uncomfortable for the subject.

6. CONCLUSION

Autism Spectrum Disorders (ASD) are neurodevelopmental disorders characterized by difficulties in social interaction and communication, accompanied by atypical behaviours such as stereotypies, restricted interests, and other symptoms that compromise quality of life. Deficits may manifest as early as infancy and can present a wide range of manifestations, from specific limitations in learning or executive function control to global impairment of social skills or intelligence. As previously noted, each individual is unique, and while presenting with autism spectrum disorder, no two individuals will be alike, as the disorder presents with qualitative, quantitative, and intensity differences. The studies conducted [8-59] report positive outcomes from Non-Invasive Brain Stimulation (NIBS) treatments in patients with autism spectrum disorder, regardless of the severity of the disorder, the patient's gender, or the area stimulated. In reaching these conclusions, various brain regions were stimulated based on the cerebral deficits associated with autism, with particular focus on the stimulation of the Dorsolateral Prefrontal Cortex (DLPFC). Researchers have concentrated on this brain area because it is defined as a critical cognitive control centre, essential for goal-directed behaviours and decision-making processes. Furthermore, the DLPFC has multiple functions that relate to significant executive functions, such as inhibitory processes, action selection, and the implementation of behavioural patterns.

7. REFERENCES

1. Autism and Developmental Disabilities Monitorig Network Surveillance Year 2008 Principal Investigators; Center for Disease Control and Preventions. Prevalence of autism spectrum disorders-Autism and Developmental Disabilities Monitoring Network, 14 sites, United States, 2008. MMWR Surveill Summ. 2012;61(3):1-19.
2. Vicari S, Valeri G, Fava L. L'autismo. Dalla diagnosi al trattamento. Il Mulino: Bologna; 2012.
3. Roccella M. Neuropsichiatria dell'infanzia e (II ed). Piccin. 2020.
4. Dehaene S. Vedere la mente. Il cervello in 100 immagini. Raffaello Cortina Editore, 2022.
5. Rossi S. Il Cervello Elettrico. Le sfide della neuromodulazione. Raffaello Cortina Editore, 2020.
6. Bolognini N, Vallar G. Stimolare il cervello. Manuale di stimolazione cerebrale non invasiva. Il Mulino. 2015.
7. Gazzaniga MS, Ivry RB, Mangun GR. Neuroscienze cognitive. Zanichelli. 2020.

8. Gomez L, Vidal B, Maragoto C, Morales LM, Berrillo S, Cuesta HV, et al. Non-Invasive Brain Stimulation for Children with Autism Spectrum Disorders: A Short-Term Outcome Study. Behav Sci (Basel). 2017;7(3).63.
9. Khaleghi A, Zarafshan H, Vand SR, Mohammadi MR. Effects of Non-invasive Neurostimulation on Autism Spectrum Disorder: A Systematic Review. Clin Psychopharmacol Neurosci. 2020;18(4):527-52.
10. Abujadi C, Croarkin PE, Bellini BB, Brentani H, Marcolin MA. Intermittent theta-burst transcranial magnetic stimulation for autism spectrum disorder: an open-label pilot study. Braz J Psychiatry. 2018;40(3):309-11.
11. American Psychiatric Association. Manuale diagnostico e statistico dei disturbi mentali - Quinta edizione. DSM-5. Tr.it. Raffaello Cortina: Milano; 2015.
12. Baird G, Cass H, Slonims V. Diagnosis of autism. BMJ. 2003;327(7413):488-93.
13. Barahona-Correa JB, Velosa A, Chainho A, Lopes R, Oliveira-Maja AJ. Repetitive Transcranial Magnetic Stimulation for Treatment of Autism Spectrum Disorder: A Systematic Review and Meta-Analysis. Front Integr Neurosci. 2018;12:27.
14. Bisiacchi P, Vallesi A. Il cervello a lavoro. Nuove prospettive in neuropsicologia. Il Mulino. 2017.
15. Carradori G, Sangiorgi A. L'analisi funzionale del comportamento. Principi, metodi e tecniche. Erickson. 2017.
16. Casanova MF, Sokhadze EM, Casanova EL, Li X. Transcranial Magnetic Stimulation in Autism Spectrum Disorders: Neuropathological Underpinnings and Clinical Correlations. Semin Pediatr Neurol. 2020;35:100832.
17. Cohen LG, Celnik P, Pascual-Leone A, Corwell B, Falz L, Dambrosia J, et al. Functional relevance of cross-modale plasticity in blind humans. Nature, 1997;389(6647):180-3.
18. Cole EJ, Barraclough NE, Enticott PG. Investigating Mirror System (MS) Activity in Adults with ASD When Inferring Others' Intentions Using Both TMS and EEG. J Autism Dev Disord. 2018;48(7):2350-67.
19. Cottini L. Educazione e riabilitazione del bambino autistico, Carrocci editore, Roma. 2015.
20. Courchesne E, Karns CM, Davis HR, Ziccardi R, Carper RA, Tigue ZD, et al. Unusual brain growth patterns in early life in patients with autistic disorder. An MRI Study. Neurology. 2001;57(2).
21. Desarkar P, Rajji TK, Ameis SH, Daskajakis ZJ. Assessing and Stabilizing Aberrant Neuroplasticity in Autism Spectrum Disorder: The Potential Role of Transcranial Magnetic Stimulation. Frontiers in Psychiatry. 2015;6:124.
22. Enticott PG, Fitzgibbon BM, Kennedy HA, Arnold SL, Elliot D, Peachey A, et al. A Double-blind, Randomized Trial of Deep Repetitive Transcranial Magnetic Stimulation (rTMS) for Autism Spectrum Disorder. Brain Stimul. 2014;7(2):206-11.
23. Faggioli R, Lorenzo JS. Dentro l'autismo. Franco Angeli. Milano. 2014.
24. Filippetti M, Amico AP, Mazzucchelli M, Perin C, Cornaggia CM, Megna M, et al. Tecniche di stimolazione cerebrale non invasiva: la stimolazione elettrica transcranica. Revisione narrativa della letteratura. Springer HealtCare Communications. Giornale Italiano di Medicina Riabilitativa. 2021;35(3):23-9.

25. Foxx R. Tecniche base del metodo comportamentale. Sviluppare le abilità e ridurre i comportamenti problema. Erickson. 2021.
26. Franciosi F. La Regolazione Emotiva nei Disturbi dello Spettro Autistico. Verso un modello operativo in ambito clinico e psicoeducativo. Edizioni ETS, Pisa. 2017.
27. Greenspan SI, Wieder S. Engaging Autism: Using the Floortime Approach to Help Children Relate, Communicate, and Think. Da Capo Lifelong Books: Cambridge; 2006.
28. García-González S, Lugo-Marín J, Setien-Ramos I, Gisbert-Gustemps L, Arteaga-Henríquez G, Díez-Villoria E, et al. Transcranial direct current stimulation in autism spectrum disorder: A systematic review and meta-analysis. Eur Neuropsychopharmacol. 2021:48:89-109.
29. Guiot G, Meini C, Sindelar MT. Autismo e musica. Il modello Floortime nei disturbi della comunicazione e della relazione. Edizione Centro Studi Erickson S.p.A. 2012.
30. Han YMY, Chan MMY, Shea CKS, Lai OL, Krishnamurthy K, Cheung MC, et al. Neurophysiological and behavioral effects of multisession prefrontal tDCS and concurrent cognitive remediation training in patients with autism spectrum disorder (ASD): A double-blind, randomized controlled fNIRS study. Brain Stimul. 2022;15(2):414-25.
31. Hyman SL, Levy SE, Myers SM. Identification, Evaluation, and Management of Children with Autism Spectrum Disorder. Clinical Report. Pediatrics. 2020;145(1):e20193447.
32. Hollister Sandberg E, Spritz BL. Autismo: Piccola guida a tutti gli interventi terapeutici, Il Castello srl, Milano. 2016.
33. Kim AK, Taghva A, Peterson KA, Liu CY. Improved autism behaviors after noninvasive cerebral transmagnetic stimulation using customized frequency modulation: follow-up mean 24 months. American Association of Neurological Surgeons. 2014.
34. Kleinhans NM, Richards T, Weaver K, Johnson LC, Greenson J, Dawson G, et al. Association between amygdala response to emotional faces and social anxiety in autism spectrum disorders, Neuropsychologia. 2010;48(12):3665-70.
35. Kosslyn SM, Pascual-Leone A, Felician O, Camposano S, Keenan JP, Thompson WL, et al. The role of area 17 in visual imagery: Convergent evidence from PET and rTMS. Science. 1999;284(5411):167-70.
36. Làdavas, E., Berti, A. Neuropsicologia. Quarta edizione. Il Mulino. 2020.
37. Lefaucheur JP, Andre-Obadia N, Antal A, Ayache SS, Baeken C, Benninger DH, et al. Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS). Clin Neurophysiol. 2020;131(2):474-528.
38. Mazza M, Valenti M. Cognizione sociale e autismo. Modelli comportamentali, implicazioni cliniche e valutazione. Franco Angeli. 2019.
39. Miniussi C, Harris JA, Ruzzoli M. Modelling non-invasive brain stimulation in cognitive neuroscience. Neurosci Biobehav Rev. 2013;37(8):1702-12.
40. Minshew NJ, Keller TA. The nature of Brain Dysfunction in Autism: Functional Brain Imaging Studies. Curr Opin Neurol. 2010;23(2):124-30.
41. Ni H-C, Hung J, Wu C-T, Wu Y-Y, Chang C-J, Chen R-S, et al. The Impact of Single Session Intermittent Theta-Burst Stimulation over the Dorsolateral Prefrontal Cortex and Posterior Superior Temporal Sulcus on Adults with Autism Spectrum Disorder. Front Neurosci. 2017;11:255.

42. Oberman LM, Enticott PG, Casanova MF, Rotenberg A, Pascual-Leone A, McCracken JT, et al. Transcranial magnetic stimulation in autism spectrum disorder: Challenges, promise, and roadmap for future research. Autism Res. 2016;9(2):184-203.
43. Paslely BN, Allen EA, Freeman RD. State-Dependent Variability of Neuronal Responses to Transcranial Magnetic Stimulation of the Visual Cortex. Neuron. 2009;62(2):291-303.
44. Paulus W. Transcranial electrical stimulation (tES – tDCS; tRNS, tACS) methods. Neuropsychol Rehabil. 2011;21(5):602-17.
45. Razmkon A, Maghsoodzadeh S, Abdollahifard S. The effect of deep brain stimulation in children and adults with autism spectrum disorder: A systematic review. Science Direct. 2022;22.
46. Roccella M. I disturbi dello spettro autistico. Carbone Editore, Palermo. 2015.
47. Ruzzoli M, Marzi CA, Miniussi C. The neural mechanisms of the effects of transcranial magnetic stimulation on perception. J Neurophysiol. 2010;103(6):2982-9.
48. Touchette PE, Howard JS. Errorless learning: reinforcement contingencies and stimulus control transfer in delayed prompting. J Appl Behav Anal. 1984;17(2):175-88.
49. Davis III TE, White SW, Ollendick TH, editors. Handbook of Autism and Anxiety. Autism and Child Psychopathology Series. Spinger: New York; 2014.
50. Venuti P. Intervento e riabilitazione nei Disturbi dello Spettro Autistico, Carrocci Editore, Roma. 2014.
51. Walsh V, Rushworth MA. primer of magnetic stimulation as a tool for neuropsychology. Neuropsychologia. 1999;37(2):125-35.
52. Xiao L, Huo X, Wand Y, Li W, Wang C, Wang F, et al. A bibliometric analysis of global research status and trends in neuromodulation techniques in the treatment of autism spectrum disorder. BMC Psychiatry. 2023;23(1):183.
53. Mandala G, Capone D, Ajovalasit D. Early Diagnosis of Cognitive Impairment in Mild TBI. J Clin Stud Med Case Rep. 2023;10:0156.
54. Giorgio M, Dalila C, Sofia M. Prevention project of the chronicity of cognitive-behavioral complications of mild head traumatic brain injury. AJMCRR. 2024;3(1):1-9.
55. Mandala G, Sanguedolce G, Angileri V, Maltese S, Lupo F, Spataro R. et al. Rehabilitation of motor and cognitive functions in patients with right cerebral hemisphere stroke. AJMCRR. 2023;2(8):1-4.
56. Guger C, Spataro R, Pellas F, Allison BZ, Heilingher A, Ortner R, et al. Assessing command-following and communication with vibro-tactile P300 brain computer interface tools in patients with unresponsive Wakefulness syndrome. Front Neurosci. 2018;12:423.
57. Mandala' G, La Mantia, V. Alien Hand: bibliographic review and description. Academia Letters. 2022;5584.
58. Spataro R, Xu Y, Xu R, Mandalà G, Allison BZ, Ortner R, et al. How brain-computer interface technology may improve the diagnosis of the disorders of consciousness: A comparative study. Front. Neurosci. 2022;16:959339.
59. Mandalà G, Sarullo FM, Ficile S, Mandalà S Maltese S, Lupo F, et al. Cognitive impairment in cardiovascular diseases: Preliminary data. AJMCRR. 2023;2(11):1-2.