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ANTIDEPRESSANTS APPLICATION PROSPECTS AND RELEVANCE OF THE SEARCH FOR HIGH-ACTIVITY ANTIDEPRESSANTS

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Abstract: Treatment strategies that present a rapid improvement of depressive symptoms—within hours or even a few days—and whose effects are sustained would have a huge impact on public health. Current depression therapies typically take weeks to months to achieve response and remission, which frequently results in significant morbidity and disruption in personal, professional, family, and social life, as well as a risk for suicidal behavior. This article reviews the published data related to various aspects of rapid improvement of depressive symptoms. Antidepressant responses can be produced by some investigational treatments far more quickly than by current drugs. Knowing the molecular underpinnings of these experimental interventions is probably going to result in better treatments rather than just increasing our understanding of the conventional antidepressants that are now on the market. Walking or jogging, yoga, and strength training are more beneficial than other forms of exercise, especially when done intensely, when it comes to treating depression. When compared to other treatments, yoga and strength training were well received. People with and without comorbidities, as well as those with varying baseline degrees of depression, seemed to benefit similarly from exercise. Future research could try to blind staff and participants in order to lessen expectation effects. In addition to psychotherapy and medicines, these types of exercise could be regarded as essential treatments for depression.

Key words: 5-hydroxytryptamine, or 5-HT, non-severe depression, serotonin, treating depression, adverse reactions.

Introduction. For many years, there has been a prevalent belief that depression stems from irregularities in brain chemicals, specifically serotonin (5-hydroxytryptamine, or 5-HT), which serves as a significant rationale for the use of antidepressants. First proposed in the 1960s, a connection between depression and decreased serotonin became generally known in the 1990s when selective serotonin reuptake inhibitors (SSRIs) were available. Even if it has been challenged more lately, the serotonin theory of depression is still widely accepted, supported by top experts, a large body of empirical research, and principal English language textbooks. According to surveys, at least 80% of the general population now think that the "chemical imbalance" that causes depression is known to exist [1,2,3,4]. Though the serotonin theory of depression has been so influential, no thorough review has yet synthesized the relevant evidence. It is commonly believed that the effects of antidepressants demonstrate that depression must be at least partially caused by a brain-based chemical abnormality, and that the apparent efficacy of SSRIs shows that serotonin is implicated. Other explanations for the effects of antidepressants have been proposed, such as the idea that they work via an amplified placebo effect or their ability to restrict or blunt emotions in general [5,6,7]. Tricyclic antidepressants (TCAs), selective serotonin reuptake inhibitors (SSRIs), selective serotonin and norepinephrine reuptake inhibitors (SNRIs), monoamine oxidase inhibitors (MAOIs), and atypical antidepressants, which include unclassified medications like bupropion, mirtazapine, and vortioxetine, are the five classes of antidepressants. Repurposing some antidepressants to treat bacterial and fungal infections has been experimentally tested17. Ipramine hydrochloride, sertraline hydrochloride, amitriptyline hydrochloride, and paroxetine hydrochloride have all recently been studied for their antiviral

properties against Marburg Virus18 and SARS-CoV-2, respectively [8,9,10,11]. Therefore, we conducted a systematic review and network meta-analysis to determine the comparative effectiveness of exercise and antidepressants on depressive symptoms in adults with non-severe depression. We also looked at the effect of combination treatment versus either treatment alone to explore the potential synergistic action of exercise and antidepressants. Finally, we set out to compare the drop-out rates of participants among interventions as a measure of treatment acceptance. Comparing the effects of exercise and antidepressants is crucial to clear up whether exercise is a suitable non-pharmacological treatment approach to manage non-severe depression and to inform current international treatment guidelines regarding the protective role of exercise in depression [12,13,14,15]. Rapid reaction, antidepressant, time to, glutamate, sleep, therapies, latency, and depression were among the keywords and phrases used to search the medline database (1966–2007) for literature for this review. The collected data was arranged based on the following topics: new technology to better understand rapid antidepressant activities; therapies demonstrating quick response and its possible neurological basis; and clinical relevance and time course of antidepressant action [7-11].

Materials and Methods

The methodology of this study involved a systematic review and network meta-analysis to evaluate the comparative effectiveness of exercise and antidepressants on depressive symptoms in adults with non-severe depression. A comprehensive literature search was conducted using databases such as Medline, focusing on keywords including rapid reaction, antidepressants, serotonin, non-severe depression, and therapies. The selection criteria included randomized controlled trials, observational studies, and clinical reports that analyzed the efficacy of antidepressants and exercise interventions. Data extraction was carried out systematically, ensuring the inclusion of studies that provided measurable outcomes on depression severity, treatment duration, and dropout rates. The methodological approach also considered the effect of combination therapy, analyzing the potential synergistic benefits of integrating antidepressants with structured exercise programs. The impact of various exercise modalities, such as strength training, yoga, and aerobic activities, was assessed based on their intensity and reception among individuals with different baseline levels of depression. Statistical analyses included meta-analytical techniques to compare effect sizes and treatment response rates, ensuring the reliability of findings. Special attention was given to biases associated with expectation effects by reviewing blinding techniques in included studies. The methodology further examined the time course of antidepressant action, investigating the onset of symptom improvement and its clinical relevance. Overall, the methodological framework aimed to provide a comprehensive assessment of current treatment approaches, contributing to the optimization of depression management strategies through pharmacological and non-pharmacological interventions.

Results and Discussion

This article's aim is to review research on the following topics: 1) the time it takes for current antidepressant treatments to start, which usually happens within a few weeks; 2) interventions that significantly reduce core depressive symptoms in a matter of hours to days; and 3) the cellular mechanisms thought to be involved in the quick onset of antidepressant actions. We hope that this article will inspire new directions in medication development research on treatments that can be implemented in a matter of hours.

Reasons for needing rapid antidepressant action: major depressive disorder's high morbidity during latency. Jick and colleagues found that the first month of antidepressant treatment was associated with a higher risk of suicide, especially during the first nine days; individuals exhibited similar rates of vulnerability regardless of the chemical class of their antidepressant. It is noteworthy that the higher risk of suicide and other intentional acts of self-harm during the first month of treatment is not uncommon and when it occurs, it has been hypothesized to be due to a mismatch in symptom improvement, i.e., physical energy improves first, while the resolution of depressive mood and negative thoughts occurs more gradually [1,5,8,9]. Secondary psychosocial losses may also be linked to a delayed onset of antidepressant effects. Since depression has been shown to reduce quality of life,

which impairs the skills needed to work, build and maintain relationships, be productive, and function in many other areas, severe depressive episodes should be considered an emergent condition that requires a rapidly effective intervention to limit the time spent in this state; this kind of thinking is typically observed in many other medical disorders. Rapid therapeutic effects have been shown in recent years to significantly alter the human and financial costs associated with many medical illnesses. For example, triptans, which have been shown to produce maximum therapeutic effects for migraine within minutes or hours, have transformed the treatment of migraine. These findings make the urgent need to study and create novel antidepressants that act quickly to eradicate the early morbidity and mortality that arise from depressive episodes a top public health priority [11,-18].

Time course of antidepressant action and clinical variables. The timing of antidepressant response has been a well-debated topic in the psychiatric literature over the last twenty years. A number of large-scale studies and meta-analyses have provided evidence that some current antidepressant treatments can have some initial positive effects within the first week, challenging the conventional wisdom that standard antidepressants have a delayed onset of at least two weeks. Other research, however, indicates that the typical duration for the onset of antidepressant action with conventional antidepressants is approximately two weeks; this duration increases to twenty days when response criteria are taken into account [1,11,12,14,17]. The observed inter-individual variation in outcomes is only marginally addressed by current hypotheses regarding the potential mechanisms underlying antidepressant response. Research on the onset of antidepressant effects may preferentially involve two major aspects: first, it will be crucial to prospectively determine associations between the time required for antidepressants to produce a significantly greater therapeutic effect in overall symptoms compared to placebo associated with other outcomes like response and remission; and second, it will be crucial to determine the timing of improvement of individual depressive symptoms and constructs, based on findings that show that certain symptoms or clusters of symptoms may tend to remit more quickly than others and may produce clinically relevant predictors that may directly correlate short- and long-term outcomes [5-12].

In order to better understand the rapid antidepressant actions, new technologies have been developed. In addition to the potentially novel approaches in the treatment of depression described above, new investigational tools have been proposed to present validity for predicting rapid improvement and may represent potential endophenotypes associated with faster antidepressant response. Since we are currently unable to predict who will respond to a particular treatment more quickly, evaluating characteristics observed in studies using useful technologies like structural and functional imaging, physiological studies, and genetic studies may improve our understanding of the neurobiological basis involved in the rapid improvement and may enable the identification of surrogate outcomes and molecular targets for the next generation of faster-acting antidepressants. The more we understand these individual differences, the more likely it is that clinicians will be able to identify subgroups of depressed subjects who will respond better to specific treatments, reducing the risks of treatment-resistance and lack of efficacy. As a whole, these encouraging pharmacogenetic findings support a central role for genes regulating these systems in the rapid response and improved outcome in depression [2,7,1,14,15].

Improving the results of the healthcare team. Many patients do not obtain proper treatment, despite the fact that antidepressants are helpful in treating depression and its various indications. An interprofessional team-centered approach is necessary for a clinician to effectively detect and diagnose depression, educate patients, use evidence-based medication, closely monitor patients for compliance, identify side effects, and assess the efficacy of treatment. Research indicates that a variety of factors influence how well patients take their antidepressants. In general, adherence was predicted by worries about adverse drug reactions. Comorbidities among patients may also influence their adherence to antidepressant drugs. Conditions that affect a person's cognitive state in particular may cause non-compliance. Lower adherence was predicted by factors such as alcohol or drug addiction, cardiovascular illness, metabolic diseases, youth, low-income inhabitants, and the use of depressive medications from older generations, especially in in the acute phase [22-28]. The pharmacist and

nursing staff are also members of the healthcare team who must help with antidepressant care: Psychiatric specialty nurses are best suited to identify treatment failure, counsel patients on the medication, monitor adverse events, and assess compliance; pharmacists can confirm agent selection and dosage and perform medication reconciliation for drug interactions; both nurses and pharmacists need open access to the prescriber in case of concern; emergency department physicians should quickly stabilize the patient in the event of an antidepressant overdose, ensuring adequate breathing, circulation, and airway; medical toxicologists should be consulted for severe poisoning; and a psychiatrist should be consulted for a deliberate overdose. As previously mentioned, the patient undergoing antidepressant therapy is cared for by a multidisciplinary team consisting of clinicians, specialists, pharmacists, nurses, and other healthcare professionals. By working together, the team can optimize patient outcomes by maximizing efficacy and minimizing adverse reactions [1,15,19,21,22].

Discussion. Redefining our understanding and definition of the clinically significant ideas related to depression therapy is essential, as the data discussed in this paper highlights. The current model has concentrated on evaluating existing pharmacological treatments on a weekly basis; these evaluations primarily reveal minor variations among drugs that are known to have low capacity to provide quick antidepressant effects. The discovery of novel, effective treatments for depression is hampered by the need for a quicker and longer-lasting antidepressant response, which could also avoid the negative neurobiological and psychosocial repercussions of recurrent or persistent depressive episodes. Our ultimate objective can and should be to have an antidepressant that acts quickly—within hours or days as opposed to weeks or months. As we learn more about the genetics of depression, we can use that knowledge to make better decisions about which patients are likely to respond to which therapeutic approach. This new paradigm in the therapeutics of depression is expected to include not only the development of new and improved therapeutics, but also the development of tools that allow us to evaluate antidepressant efficacy within hours or days of first administration; many other medical specialties, including cardiology, neurology, oncology, and endocrinology, presently have the tools necessary to evaluate therapeutic onset quickly and reliably [1-11]. It should be noted that many substances can produce short-term euphoria and hyperactivity (including psychomimetic effects) that are restricted to the half-life of the compound being administered. However, these effects cannot be classified as a genuine improvement of core depressive symptoms when it comes to the development of novel medicines. Hence, drugs that induce antidepressant effects that are both quick and long-lasting (in terms of core depressive symptoms and constructs) may be a crucial characteristic of novel pharmaceutical treatments that have the ability to produce antidepressant activity quickly. Rapid antidepressant-inducing therapies may quickly repair damaged neural circuitry, hence enhancing quality of life, functional well-being, and symptom relief. Therefore, finding new antidepressants that produce antidepressant response and remission faster should be a top priority in mood disorder research. Preclinical and clinical studies have been conducted in this area, looking for genes, signaling pathways, and/or neurochemical circuits that may be involved in these therapeutic effects. Common targets for future research in this area may include both glutamatergic modulation, the neuro-biomolecular basis for sleep, wakefulness, SD, and electrical activity in limbic-cortical circuits. Current evaluative strategies are inadequate in terms of tools that measure early improvement; for example, if ratings are only collected once a week, early response and identification of important symptoms/clusters predictive of ultimate response—possibly occurring within hours or days of administration—will be neglected [6,7,8,9,14,17,20]. The creation of antidepressants that take effect quickly—within hours or days—should be the second main priority. This objective is achievable, as demonstrated by a wealth of evidence from various medical specialties. For instance, a migraine attack, pain, or elevated blood pressure or blood sugar can all be prevented in a matter of hours. To put it another way, we should expect and be able to quantify appropriately antidepressant treatments that may work within hours or days, rather than presuming that patients will respond within weeks or months. Our current expectations surrounding antidepressant treatments are excessively low. Thankfully, there is a lot of promise in the current research on antidepressant tactics. We anticipate that this research will set the standard for creating the upcoming generation of faster-acting and more effective antidepressants to better treat this devastating illness [29-34].

Conclusions. Compared to current drugs, certain investigational treatments can produce an antidepressant response significantly more quickly. Rather than only expanding our understanding of the existing standard antidepressants, a better understanding of the molecular basis of these experimental interventions is likely to result in the development of better therapies.

Depression can be effectively treated with exercise, and strength training, yoga, and jogging or walking are more beneficial than other forms of exercise, especially when done vigorously. Comparing yoga and strength training to other treatments, they were well tolerated. People with varying baseline levels of depression and those without comorbidities seemed to benefit equally from exercise. In order to reduce expectancy effects, future research could try to blind staff and participants. These types of exercise could be viewed as essential treatments for depression, in addition to psychotherapy and antidepressants.

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