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Editorial Article

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An open Letter to the Editors of Nature Magazine Regarding their Involvement in a PR Action in Favor of a Dubious Publication about Advances in the Treatment of Alzheimer's Disease

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Recently, on August 6, 2025, Nature journal published a report on the preliminary results of a study (conducted at Harvard University) on the effect of Lithium on the formation and development of amyloid plaques (which are considered the primary cause of Alzheimer's disease) [1-3].

The study itself is not something entirely new or extraordinary - there are currently 182 studies being conducted in the United States related to the causes of occurrence and development of Alzheimer's disease [4].

Scientists still do not fully understand the mechanisms that lead to Alzheimer's disease. Therefore, most of the studies focus on exploring the highly noticeable changes in the brain - these are 1) the accumulation of fragments of the beta-amyloid protein in the interneuronal space (clumps of which are called beta-amyloid plaques) and 2) the accumulation of an abnormal form of the tau protein inside neurons (called tau tangles). The studies published in Nature relate to the first type of the phenomena - studying the effect of lithium on the processes of accumulation of fragments of the beta-amyloid protein in the interneuronal space.

As already mentioned above, there is nothing special about this study (published in Nature on August 6, 2025) – an ordinary and routine research work. But the explosive wave of excited reprints that immediately tailed this publication causes shock and confusion: why is this happen? for what reason? in what aim?

Indeed, the range of responses (to the Nature publication) is surprising and astounding in its multiplicity and diversity. Look what you can to meet there:

Science magazines: Science (USA), The Scientist (G. Britain), Science & Vie (France), New Scientist (London), ScienceAlert (USA), Newswise (USA), Nature (USA);
Newspapers: The Washington Post (USA), The Harvard Gazette (USA), Newsweek (New York City), Courthouse News Service (USA), The Independent (British), The Hindu (India), Libération (France), El País (Madrid), The Jerusalem Post (Israel), The Boston Globe (USA), The Daily Telegraph (UK), Diari ARA (Barcelona), New Indian Express (India).

Media: CNN, YouTube, Instagram, WCVB Channel 5 (Boston), Yahoo! News (UK), Straight Arrow News (USA), Science Media Centre España (Spain), Life Science Network (Brazil).

Professional publications: The National Institutes of Health (USA), Medical News Today (UK), Baptist Health (South Florida), Genetic Engineering & Biotechnology News (GEN USA), Chemical & Engineering News (C&EN USA), Chemistry World (UK), Psychiatry Redefined (USA), Parsemus Foundation (San Francisco), ResearchGate (Berlin), MedicalBrief (Africa's weekly medical news), BioWorld MedTech (Asia), KFF Health News (California), Piedmont Orthopedics (Georgia.), Being Patient (USA), The American Dental Association (USA), Pharmacy Times (USA), MedPage Today (USA), Pharmazeutischen Zeitung (Germany)

(The list is far from being complete - I managed to find more than 200 responses to the Nature publication).

The titles of the publications are also astonishing: "Lithium May Combat Alzheimer's Disease", "Lithium deficiency identified as key Alzheimer's trigger", "Lithium may reverse Alzheimer's disease". And, although one of the titles resolutely rejected suspicions of conspiracy or intent (Lithium for Alzheimer's? A Hype-Free Explainer), such suspicions naturally arise - all the publications appeared, literally, on the same day, August 6-7,

2025 (with some exceptions - August 9, 2025). All the publications were unanimous in recognizing the remarkable prospects of using lithium to treat Alzheimer's disease.

The authors of the article published in Nature acknowledge that "In mouse models of Alzheimer's disease (AD), endogenous Li protects against amyloid deposition, tau hyperphosphorylation, neuroinflammation, and loss of synapses, axons, and myelin," [1],

At the same time, they share the generally accepted view that amyloid deposits are the primary cause of AD. "AD is defined by specific brain abnormalities—amyloid β plaques and neurofibrillary tau tangles—which are thought to actively influence the neurodegenerative process," [5].

However, in the current scientific literature, the connection between beta-amyloid tangles and Alzheimer's disease is considered to be rather tentative and unproven. It is already known that "Trials of anti-amyloid β have highlighted the limitations of this approach and suggested that amyloid-β may not play such an important role in neurodegeneration compared to other factors contributing to it" [6].

And more "the mechanisms leading to the accumulation of plaques and tangles are unknown, and removing amyloid-β has not halted neurodegeneration", [6]. And more "AD is more akin to a syndrome than to a traditional disease, with its pathological manifestation representing a convergence of pathogenic pathways", [6].

In this context, the hype-presentation of Li as a panacea for all problems (associated with AD) looks a bit inappropriate and irresponsible. The international hype campaign around the publication in Nature thus looks like a highly doubtful undertaking.

I personally, have long been irritated and outraged by all this hype around the possible regulation of the processes of accumulation of amyloid plaques. I have my own, and special, opinion on this matter – everyone talks about the possibilities of managing the processes of accumulation of amyloid plaques, but at the same time no one talks about the possible causes of the process. And without this, the entire battle against amyloid plaques turns into "monkey business" - you destroy one generation of amyloid plaques (without eliminating the causes of their formation), but a new generation of amyloid plaques immediately appears to replace them.

Many years ago, I proposed my own (specific) approach to the study and understanding of the neurophysiology of the brain, which I call "Information Neurophysiology" [7]. Within the framework of this approach, a new theory of the appearance and formation of amyloid plaques naturally arises. Subsequently, the possibility of theoretical opposition to the processes of their formation and development appears [8-10]. (That is, a theory of possible therapy for AD emerges. Today, this problem is not even discussed. In the best case, today we are only talking about slowing down the processes of AD development, if they can be diagnosed at an early stage of the disease).

I am not going to take advantage of the moment and force you to discuss my ideas – the list of articles that I have published in recent years is given at the end of this letter. (And everyone who is interested can explore it). My problem is not that my ideas seem unacceptable to someone. My problem is that my ideas are published in journals with a very low Impact factor (from 1.5 to 2.9) – which hinders the process of their dissemination and use (compare these figures with the impact factor of Nature (50.5), Science (45.8), Cell (42.5), or other popular publications). There is no place for a normal discussion of new ideas under such circumstances.

Therefore, I dare to propose you: not to republish in Nature my old articles, but to give them a detailed critical review. (You accompanied the publication on the effect of lithium on the formation of amyloid plaques with two very favorable reviews which, in many ways, contributed to the hype that arose around these publications) [1-3]. I do not expect to gain support or success from your review. I hope that such a review could contribute to the beginning of a serious discussion about the false underpinnings of today's discussion of AD problems, and pave the way for the development of a new modern approach to the topics of the wake up, development, and treatment of AD, [11].

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