researchers screened at every stage of the review, with a third researcher resolving any conflicts. Though a full systematic review outlining the current literature on the complete mechanisms of action of psilocybin on depression was conducted, this abstract will focus specifically on the nine papers that included human subjects, disregarding the five animal models. PROSPERO registration number: 282710.

Results: After removing duplicates, the search identified 2193 papers and forty-nine were selected for full text review. Out of nine papers outlining the mechanisms of action of psilocybin use in human subjects, three papers investigated psilocybin's effect on serotonin or glutamate receptor activity, two found an increase in synaptogenesis in regions such as the medial frontal cortex and hippocampus. Four found variation in blood flow to the amygdala, two found altered blood flow to the prefrontal cortex, and one found a reduction in delta power during sleep. Four papers found changes in functional connectivity or neurotransmission, most commonly in the hippocampus or prefrontal cortex.

Conclusions: Overall, the exact mechanism of psilocybin's potential antidepressant effect remains unclear. Multiple pathways may be involved, including alterations in serotonin and glutamate receptor activity, as well as shifts in amygdala activity, neurogenesis, and functional connectivity in various brain regions. The relative lack of studies, and the variety of neurobiological modalities and endpoints used challenged the consolidation of data into consensus findings. Further studies are needed to better characterize psilocybin's mechanism of action and to better understand the clinical effects of the use of psilocybin in the treatment of depression.

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EPP0600

Is the most really the best: a review for the most selective SSRI concept three decades later

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Introduction: Pharmaceutical slogans presuming a particular antidepressant molecule being the best solely based on a core concept could be proved "not accurate" especially following patients' actual exposure to the antidepressant for longer than the usual six or twelve weeks' trials

Objectives: Reviewing the current situations of **SSRI induced anhedonia** recognition and its management. Distinguishing anhedonia as a core symptoms of depression from SSRI induced anhedonia and the combination of both.

Methods: Review of literatures including theses related to the same topic

Results: Research suggests that, SSRIs might be more effective at treating some symptoms than others. More specifically, it has been suggested that, SSRIs might be more effective at improving symptoms such as low mood and anxiety but not anhedonia *(Argyropoulos et al. psych.pharmaology, 2013; 27(10), 869-877).* It has been proposed that, catecholaminergic antidepressants might be more effective treatments for anhedonia and emotional blunting in MDD than SSRIs *(McCabe et al. Biological psychiatry, 2010; 67(5), 439–445.* The primary effect of SSRIs is reduced processing of negative stimuli rather than increased positive stimuli. Emotional blunting is related to SSRI dose and possibly serotonergic effects on the frontal lobes and/or serotonergic modulation of midbrain dopaminergic systems projecting to the prefrontal cortex (PFC). By enhancing serotonergic transmission, SSRIs can activate the inhibitory Gamma Aminobutyric Acid (GABA) interneurons, thereby dampening the noradrenergic and dopaminergic input *(Blier. Int J Neuropsychopharmacol., 2014; 17:997–1008).*

Management of SSRI induced anhedonia includes lowering the current SSRI dose. Adding non SSRI antidepressant to the current SSRI dose or to a lowered SSRI dose. Gradual discontinuation of the SSRI and switching to another antidepressant with a different profile (SNRI) that might improve the patient's emotional response *(Koenigs. Behav Brain Res., 2009;201:239–43).*

Bupropion is an antidepressant with less possibility to give rise to **emotional blunting.** (Tomoko et al. Neuroscience Letters., 2021; 749, 135716. agomelatine(Thome et al. Journal of neural transmission., 2015; 122(1), 3-7.Vortioxetine and others (Bing et al. frontiers in psychiatry., Jan, 2019; 10-17. are of inrerest in this regard.

Image 2:

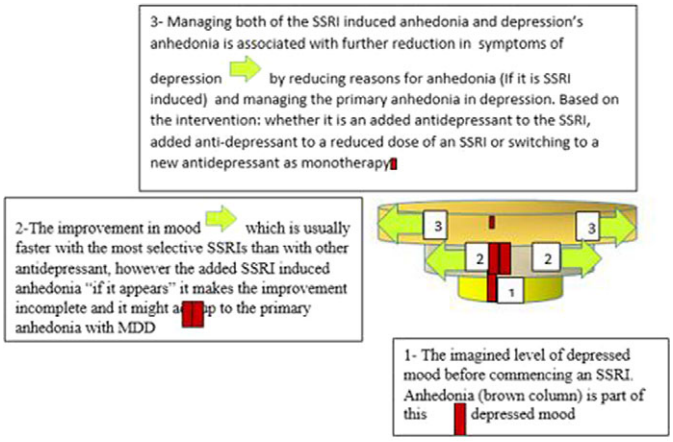

Figure (1) The depressed mood at base line then managed by an SSRI and managing the SSRI induced anhedonia successfully.

This figure is made fully by Dr Mohammed Allam, – however It can be cited without prior consent from Mohammed Allam.

Conclusions: The **most selective SSRI** concept assumes that most selective means less affinity to other receptors or secondary binding sites which might suggest less side effects and perhaps being the most efficacious. Not only serotonin but multiple neurotransmitters are in action at the downstream part of a cascade of events underpinning the etiology of MDD. MDD has heterogeneous etiology and this explains why patients respond differently. **SSRI induced anhedonia** could be tackled and we need to explore how many patients would benefit from that now and have not yet.

Disclosure of Interest: None Declared