Fluoxetine

- Environmental risk assessment of selective serotonin reuptake inhibitors (SSRIs) after COVID-19 pandemic: a systematic review
- Fluoxetine substitution for deprescribing antidepressants: a technical approach
- Changes in Antidepressant Absorption After Metabolic Bariatric Surgery
- Fluoxetine and agomelatine mitigate anhedonic and hepatic changes in chronic restraint stress rat model
- Machine learning-based model for behavioural analysis in rodents applied to the forced swim test
- Retraction: miR-16 and fluoxetine both reverse autophagic and apoptotic change in chronic unpredictable mild stress model rats
- Investigating the potential of quercetin-loaded chitosan-coated lipid carriers as an oral nanoplatform for depression management
- Identifying Predictors of Serious Adverse Events in Antidepressant Treatment from a Decade-Long Nationwide Pharmacovigilance Study: Impact of Dementia and Parkinson's Disease Treatment

Fluoxetine, sold under the brand names Prozac and Sarafem among others, is an antidepressant of the selective serotonin reuptake inhibitor class. It is used for the treatment of major depressive disorder, obsessive–compulsive disorder, bulimia nervosa, panic disorder, and premenstrual dysphoric disorder

Fluoxetine in neurosurgery

- Long-Term Clinical Response to Medical Treatment, Behavioral Therapy, or Their Combination in Cats With Hyperesthesia Syndrome
- Medical management of cerebellar mutism syndrome at a quaternary children's hospital
- The Influence of Fluoxetine on the Sensorimotor Function of the Sciatic Nerve in Wistar Rats after Axonotmesis: An Experimental Animal Model
- Neuroprotective agents ineffective in mitigating autonomic dysreflexia following experimental spinal cord injury
- Ethyl acetate extract of Nymphaea candida Presl: A potential anti-depressant and neuroprotective treatment strategy
- Jingqianshu granules mitigates premenstrual depression by regulating orexin signaling
- Medical management for cerebellar mutism syndrome following posterior fossa surgery: A systematic review
- Evaluation of safety of fluoxetine for cerebellar mutism syndrome in children after posterior fossa surgery

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Cerebellar mutism syndrome (CMS) occurs in 8-29 % of children undergoing posterior fossa tumor surgery. Its main symptoms are mutism and emotional lability. Although it is always transient, recovery time can be lengthy with long-term cognitive sequelae. There is no approved drug treatment for CMS, but some drugs are used in everyday medical practice. One of these is fluoxetine, which has been used for many years in the Department of Neuropediatrics and Pediatric Neurosurgery, Angers University Hospital, Angers.The main objective of this study was to establish the safety profile of

fluoxetine in this condition.

The records of patients admitted to the pediatric intensive care unit after brain surgery at Angers University Hospital from 2010 to 2020 were reviewed. Children aged 2 years and older who underwent a posterior fossa tumor surgery and were diagnosed with CMS were included. Data on patient characteristics, prescription of fluoxetine treatment, side effects if any, and complete mutism duration were collected.

Among 246 patients admitted to the pediatric intensive care unit for brain surgery during the study period, 23 had CMS and eight were prescribed fluoxetine. No serious adverse event related to fluoxetine was reported. Complete mutism duration did not differ significantly between the fluoxetine group and the non-fluoxetine group($p = 0.22$). However, the treatment was initiated after recovery from complete mutism in half of the treated patients.

This study suggests a positive safety profile of fluoxetine used in postoperative CMS. It does not answer the question of whether the treatment is effective for this indication. A randomized controlled trial based on a syndrome severity scale should be conducted to provide a more reliable assessment of the efficacy and safety of fluoxetine [1)]

Hosseindoost et al. aims to examine the impact of fluoxetine on HSV-TK/GCV gene therapy in human GBM cells using human olfactory ensheathing cells (OECs) as vectors. The effect of fluoxetine on Cx43 levels was assessed using the western blot technique. GBM-derived astrocytes and OECs-TK were Cocultured, and the effect of fluoxetine on the Antitumor effect of OEC-TK/GCV gene therapy was evaluated using MTT assay and flow cytometry. Our results showed that fluoxetine increased Cx43 levels in OECs and GBM cells and augmented the killing effect of OECs-TK on GBM cells. Western blot data revealed that fluoxetine enhanced the Bax/Bcl2 ratio and the levels of cleaved caspase-3 in the coculture of OECs-TK and GBM cells. Moreover, flow cytometry data indicated that fluoxetine increased the percentage of apoptotic cells in the coculture system. This study suggests that fluoxetine, by upregulating Cx43 levels, could strengthen the Antitumor effect of OEC-TK/GCV gene therapy on GBM cells [2)]

Fluoxetine is a well-studied serotonin selective reuptake inhibitor (SSRI). However, its role in apoptosis has not been clearly understood. The present investigation assessed the effects of Fluoxetine in apoptosis and the potential Notch1/ASK1/p38 MAPK signaling pathway in EBI after SAH. Adult C57BL/6 J mice were subjected to SAH. Study mice (56) were randomly divided into 4 groups: the surgery without SAH (sham (n = 8), SAH+ vehicle; (SAH+V) (n = 16), surgery+ Fluoxetine (Fluox), (n = 16) and SAH+ Fluoxetine (n = 16). Various parameters were investigated 12, 24, 48, and 72 h after induction of SAH. Western blot analysis, terminal deoxynucleotidyl transferase-mediated deoxyuridine triphosphate nick-end labeling (TUNEL) staining, Immunohistochemistry (IHC), and flow cytometry were carried out in every experimental group. According to the findings, the SAH downregulated NOTCH1 signaling pathway, Jlk6 inhibited Notch1, Notch1 inactivation increased apoptotic protein expression and suppressed Bax, and cytochrome C. Fluoxetine reversed the effects of notch1 inhibition in SAH. The Neuroprotective Fluoxetine effects involved suppression of apoptosis post-SAH. In summary, early Fluoxetine treatment significantly attenuates apoptosis and the expression of apoptosis-related proteins after 72 h post-SAH. Fluoxetine may ameliorate early brain injury after subarachnoid hemorrhage through anti-apoptotic effects and Notch1/ASK1/p38 MAPK signaling pathway [3)].

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