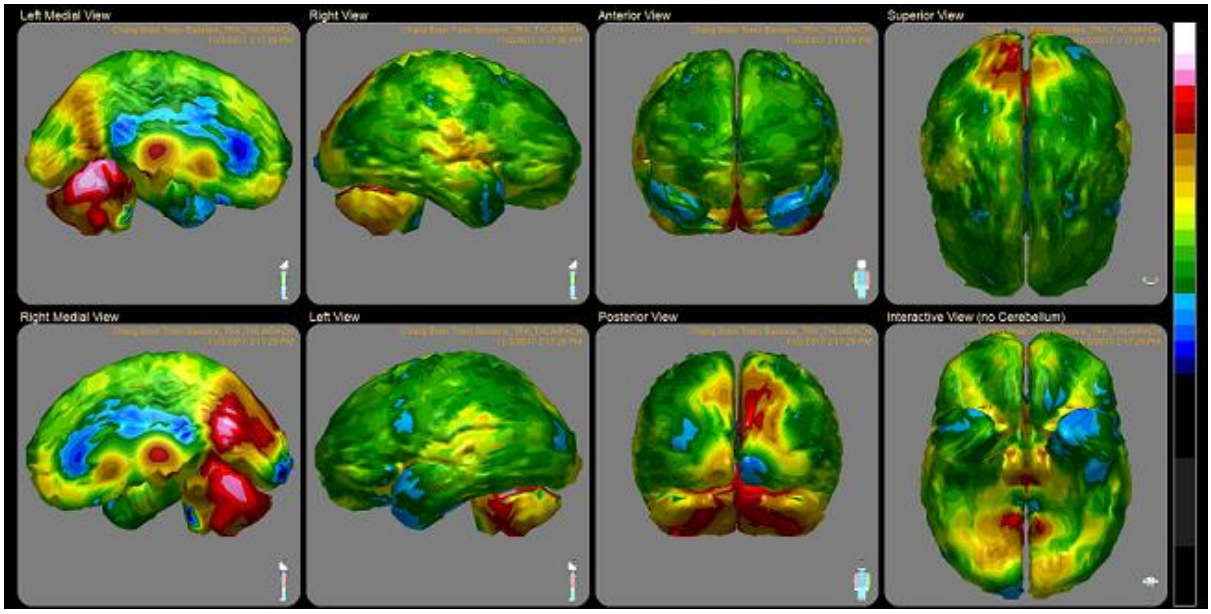
PE1651/TTT
Anonymous submission of 28 December 2017

I have been permanently damaged by the SSRI Escitalopram after a failed reinstatement of it in December 2015. I was recently diagnosed with Neurotoxicity (Toxic Encephelopathy) as a result. I am mostly bedridden, unable to drive, work, watch TV or play with my kids. I can't support them because up until recently no Dr would admit the med did this. I consider this horrific that thousands are in Facebook groups sick from SSRIs but doctors deny that it can happen. It's almost a crime and I do know some day they will be taken off the market. To deny a med that is known to by toxic caused brain damage is wrong and shameful. My story has been featured in blogs and I started a face blog group on neurotoxicity which are mostly med poisoned people. Below is one of the articles titled "My Brain after Long Term Lexapro: Chemically Induced TBI". I've had brain scans that proved my injury. My life is destroyed. Hundreds of others are as well. Please stop these Meds from being produced. Educate doctors. Help us. We cant support our families when we get sick from them if no dr will admit it and we can't get on disability.

Thank you for your consideration.

My Brain after Long Term Lexapro: Chemically Induced TBI



In December 2015, I was given Lexapro. I had taken Lexapro previously for 12 years, but because of abnormal blood chemistry and building side effects, my doctor suggested that I cease taking the medication. After five months without the drug, I was improving slightly, but because of life events, my doctor and I decided to begin taking Lexapro again. During the reinstatement of the drug, I immediately began to experience serious neurological reactions that I have come to describe as a brain kindling of sorts. It felt like my head was on fire chemically and electrically. Some of

the symptoms that developed included: fatigue, confusion, eye pain, leg weakness, headaches, visual processing disturbances, coordination difficulties, lack of concentration, and short-term memory problems.

Within a few weeks of taking the medication, these symptoms were worsening but my physician was not concerned. In fact, he suggested what I was experiencing was normal and that I continue taking the medication. I did, for a while, until the side effects were so bad that I decided to stop. I published my story on Hormones Matter a year ago. You can read it here: A Kindled Brain: Long Term Lexapro Use Reactions. A year and dozens of doctors later, I have become completely disabled. This is a follow up to my story, told with much help from the editor of Hormones Matter.

Two Years Post Lexapro Damage

I no longer take Lexapro or other medications but the damage was done. I still cannot work, watch TV, read, drive or walk more than a few feet. I have had what seems like a two year long headache and severe vision processing issues. It is going to be another great Christmas at my house.

Though I received diagnoses of Chronic Fatigue Syndrome and Fibromyalgia they really were just symptoms of something bigger. In the summer of 2017, I was finally given a provisional diagnosis of Lexapro induced neurotoxicity. Just recently, the extent of the brain damage was shown on both on a quantitative electroencephalograph (QEEG) and single-photon emission computed tomography (SPECT) brain scans. Both neurotoxicity and brain injuries were identified by both tests and later confirmed again by additional physicians who reviewed the results independently.

QEEG Results

The QEEG is an EEG with a visual mapping component. It measures the electrical activity or signal transduction between electrodes placed in specific locations on the brain. My test results showed that in certain areas of the brain there was very little organized electrical activity and other areas where there was unusual amount of high activity. What that means is that in some areas of my brain, the signals are very low and essentially not sufficiently strong for the activities that need to be performed but in other areas of my brain, there was way too much electrical activity. Specifically, the report said:

> “The patient was found to have a significant amount of low frequency (8-9 Hz) in the right posterior temporal (T6) area. There is an extreme amount of low beta (12-15 Hz) predominantly in the prefrontal and frontal areas that may interfere with executive functioning, organizing, decision making and may also account for fatigue. There is a very unusual amount of high activity for all measured frequencies (1-50 Hz) in the right posterior (O2) area, specifically in

> the right inferior occipital gyrus (Broadman areas 17, 18, and 19). This may result in sub-optimal functioning in visual processing of color, form, movement, visual perception and spatial processing."

This was the first confirmation of the cognitive dysfunction that I have been living with for the last few years. Doctor after doctor told me that my symptoms were not real and somehow made up (and some still do, as the QEEG is used mainly in research circles and not always accepted in conventional medicine). The QEEG clearly showed the abnormal electrical activity underlying my symptoms. Next up, the SPECT scan, a test I had to fight for, because again, no one seems to believe that a drug like Lexapro could cause such damage.

SPECT Scans

SPECT imaging shows brain activity (blood flow, which represents local brain metabolism and energy use). The technology uses radiolabeled isotopes that are then reconstructed with algorithms to display in color 3-D images of brain activity. The images below are some of my brain scans. Regions displayed in blue represent a state of very low activity called hypoperfusion and those in red represent hyperperfusion – excessive activity.

Figure 1. SPECT color coding scale.

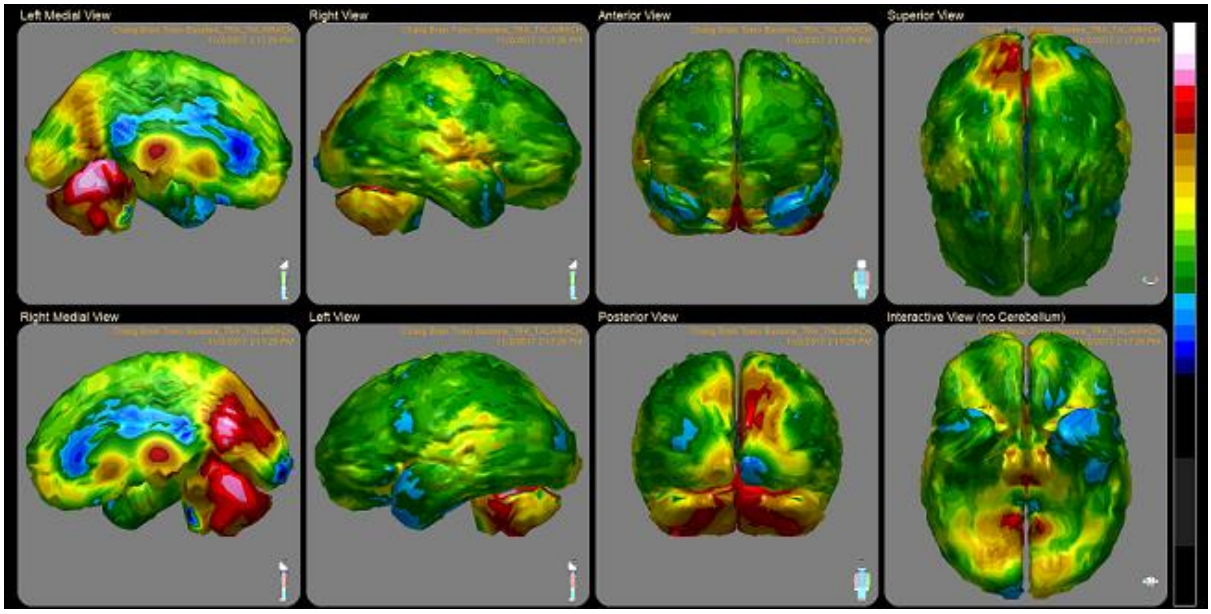


Figure 2. Changes in brain activity after long term Lexapro use.

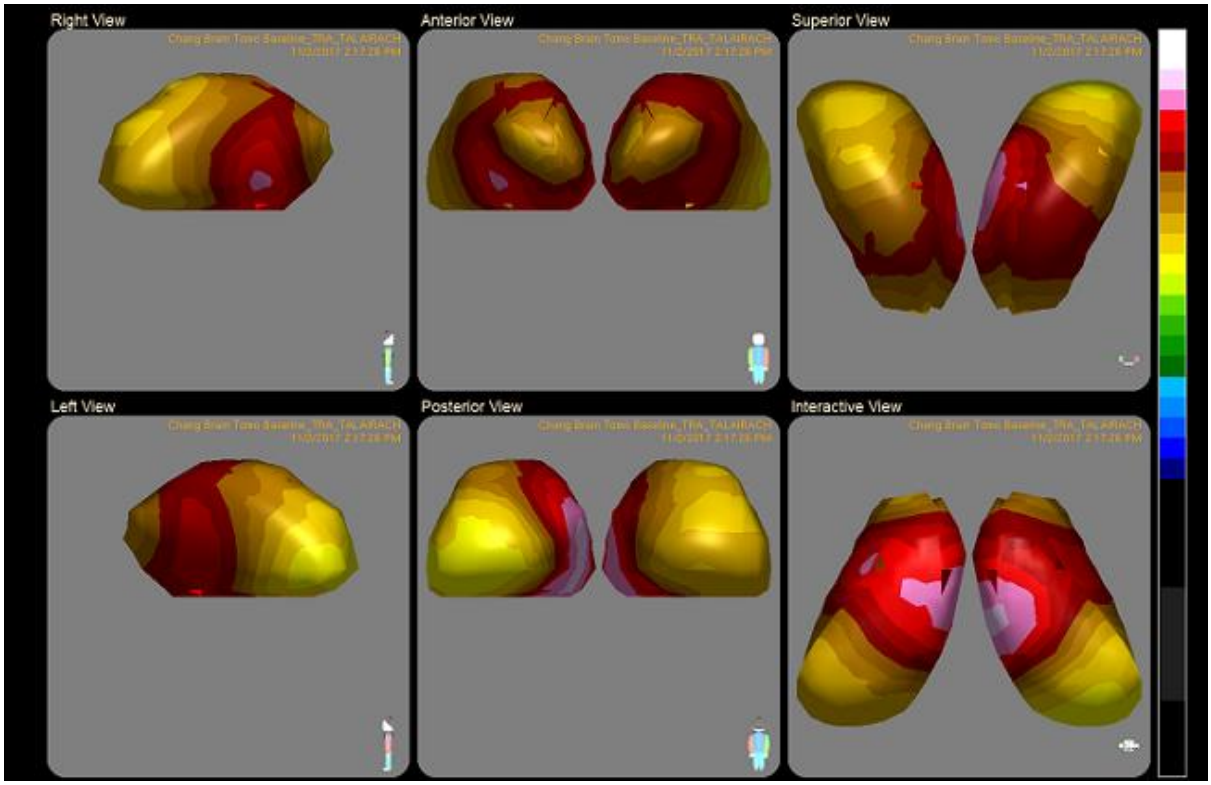


Figure 3. Post Lexapro thalamic activity.

From my images, you can see several areas of both abnormally low and abnormally high activity with very little normal brain function anywhere. From the report:

- At rest, the overall cortical activity was reduced in a diffuse, decreased, patchy pattern.

- Focal areas of abnormal cortical hypoperfusion were noted in the bilateral anterior frontal (L>R), left posterolateral frontal, left orbitofrontal, right dorsolateral prefrontal, bilateral anterior and medial temporal (L>R), bilateral superior parietal and bilateral occipital areas.

- Focal areas of abnormal subcortical hypoperfusion were noted in the anterior aspect of the pontine portion of the brainstem, bilateral caudate and right lentiform areas.

- Focal areas of abnormally increased cortical perfusion were not noted.

- Focal areas of abnormally increased subcortical perfusion were noted in the bilateral thalamic and left lentiform areas.

The doctors conclude:

> “The nature (diffuse, patchy), location (cortical and subcortical), and pattern (involving all lobes of the brain) of these abnormalities is primarily consistent

> with the scientific [literature] pertaining to a toxic/hypoxic/neuroinflammatory process and the patient's clinical history, as obtained, of a medication reaction which was received after the blind review. These results agree in large part with outside EEG data showing hypofrontality and NM report showing left frontal abnormality, likely not to be artefactual."

What This Means

My brain is a mess. According to the Hormones Matter editor, the areas of low activity in the various regions of the prefrontal cortex would explain my difficulties executive function, things like planning, decision-making and lack of concentration, while the reduced activity in the temporal and occipital lobes would explain my memory and vision difficulties, respectively. The reduced brainstem activity would connect to my walking and balance difficulties, but also, poor to autonomic regulation (the autonomic nervous system controls all automatic bodily functions including things like breathing, heart rate, temperature control, digestion, sleep/wake cycles, etc.). The areas of my brain on hyperdrive may reflect compensatory reactions, last ditch efforts to kick start some of the underactive regions. The thalamus (plural thalami), in particular, acts as a relay station between the brainstem and the rest of the brain for motor, sensory signals and 'emotional' signals via its connections to the limbic system. Mine seem to be screaming 'wake up' to the rest of the brain.

Please be careful using these medications, particularly psych meds and especially when prescribed for minor issues like a fear of flying or work-related stage fright. If I knew then what I know now, I would have never taken Lexapro.

**TBI: traumatic brain injury*