Hormones, Neurotransmitters and Brain Function

Telling you about hormones is not as simple as it used to be. You may be familiar with the hormones estrogen, testosterone, and progesterone. You may even be aware that estrogen is the main female hormone, testosterone is the predominant male hormone, and progesterone is the hormone of pregnancy. It used to be relatively cut and dry. But hormones clearly have more impact than just making females female and males male. The jokes about women and their cycles have validity. The evidence for this becomes more pronounced when hormones are out of balance, as they are in premenstrual syndrome and at menopause. Hormones have emotional impacts, impacts on your mood and brain. How does this come about?

Your nervous system uses a vast system of communication molecules called neurotransmitters (NTs) to carry messages to every part of your body. NTs communicate between the cells in your brain, and they are also communication molecules for the parts of your nervous system outside of your brain, your spinal cord and all of the nerves going to your internal organs and limbs. They impact your hormone secretions by their actions at their receptor sites in your brain and on your endocrine organs, among others the thyroid and adrenal glands, the ovary and the testes.

NTs are protein molecules which function like a key for a lock. The receptor for the neurotransmitter (NT) is embedded in the wall of the cell. It is like the lock. When the NT fits into the receptor, it induces a cellular response that impacts your body’s function. Every biologic function, every brain function, every cell function, is under nervous system control via this system of NTs. NT receptor sites are present in every organ or system. There are receptor sites for NTs located in your gastrointestinal (GI) tract, on your heart and circulatory system, your endocrine organs, and your immune system, among others.

The important intersection of your endocrine system and your nervous system is in the hypothalamus in your brain. The hypothalamus sends hormonal signals to another area of your brain, the anterior pituitary gland, located near it. When your body gets low on thyroid hormone, for example, the thyroid receptor sites on your hypothalamus become under stimulated. Your hypothalamus sends a hormone message to your anterior pituitary called thyroid releasing factor (TRF). TRF then stimulates its receptor sites on the anterior pituitary, which sends its hormonal messenger, thyroid stimulating hormone (TSH), out of your brain to the thyroid gland in your neck.

Scientists have known for some time about the hormone receptors on your hypothalamus and anterior pituitary, but what they did not know until more recently, is that there are also receptor sites for NTs on your hypothalamus that have a direct effect on hormone excretion. Serotonin is an important NT. Without adequate stimulation of hypothalamic serotonin receptor sites, your ability to make TSH is blunted. If you have too little serotonin, you may need thyroid hormone, and your doctor may not be able to tell because of current thyroid testing practices.

Many doctors use only the level of TSH as an indicator of thyroid hormone levels. If the TSH is high, your thyroid hormone is thought to be low. If your TSH is within normal limits, your thy-
roid hormone level is thought to be normal. Your doctor makes the assumption that your body would be making higher levels of TSH if it needed thyroid hormone. However, if you are low in serotonin, the ability of your anterior pituitary to make TSH is blunted, so you may be low in serotonin, TSH and thyroid hormone.

Serotonin and thyroid hormone potentiate one another. So when one or the other gets low, it brings down the activity of both. Beyond that, estrogen potentiates the activity of both thyroid and serotonin, so when your levels of estrogen drop, as they do around menopause, you loose serotonin and thyroid activity. When your thyroid function is diminished, your adrenal gland function is reduced as well. So now you are low thyroid, low serotonin, low estrogen and low adrenal. In all, you feel terrible.

You go to your doctor. Your physical examination and lab tests are mostly normal. Your doctor tells you that you are fine. Your TSH is within normal range, so he assumes your thyroid hormone level is normal. Estrogen drop at menopause is normal. He has done no tests for serotonin levels or adrenal function. You tell him that you do not feel fine. You tell him this on repeated visits. He tells you that you are depressed and gives you Lexipro.

The Lexipro may help a bit because Lexipro may increase serotonin. But it is not a solution. The cause of your problem has neither been identified nor corrected. You still do not feel well, you do not feel like your old self. In the course of subsequent visits, your doctor tells you that you are not your old self, that you are older, and must expect decline in well being as you age. He refers you to a psychiatrist. It takes the practice of medicine time to catch up with science, but this common scenario is really an affront to your intelligence, not to mention your well being. You are inclined to look into this yourself. So, here you are reading this.

Serotonin is the ‘first among equals’ of the NTs. It functions as a modulator of all the others. It alters their communications rather than eliciting activity itself. It is an inhibitory NT. Serotonin acts with gamma-amino-butyric-acid (GABA), another significant inhibitory NT. Serotonin acts by binding sites on GABA. GABA then binds to receptor sites on nerve cell bodies that damp the transmission of excitatory impulses coming through the cell, which effects the inhibition. Neither serotonin nor SSRI medications like Lexipro, work well without adequate GABA in the body. GABA is the most important and widespread inhibitory NT in the brain. It induces relaxation, calmness and aids sleep.

An explosion of research on NTs in the second half of the twentieth century uncovered the role of serotonin depletion in causing depression and anxiety. This lead to the 1987 release of the first selective serotonin reuptake inhibitor (SSRI), the anti-depressant Prozac. By delaying the reuptake of serotonin into the axon of the nerve cell, Prozac and other medications in this class, increase the levels of serotonin in your body. Low serotonin has been associated with depression, anxiety, panic attacks, memory loss, lack of concentration, premenstrual syndrome, eating disorders, insomnia, obsessive compulsive issues, migraines, addictions, reactive hypoglycemia, insulin resistance and other syndromes.

Serotonin acts to inhibit the release of the catecholamines, epinephrine and nor-epinephrine, sometimes called adrenalin and nor-adrenalin. The catecholamines are your excitatory NTs, those NTs that cause ‘fight or flight’ over excitation of your sympathetic nervous system. Balance among your NTs, and between your excitatory and inhibitory NTs, is essential for feeling well.
When your inhibitory NTs, serotonin and GABA, are low, your catecholamines lack suppression and get high. Over excitation occurs that can leave you sleepless, restless, anxious, panicky, or wired yet tired. It is not optimal to attempt to suppress the effects of nor-epinephrine and epinephrine with pharmaceuticals. The more effective solution is to increase the inhibitory NTs, to increase serotonin and GABA, by using dietary and nutritional interventions. This option may remove the symptoms and leave you feeling like yourself.

Glutamate is the most potent of all excitatory NTs. It is present in the addictive food additive Monosodium Glutamate (MSG). Glutamate is necessary for brain function. In modulated quantities, it enhances alertness and arousal. Excess glutamate in your brain induces neuronal damage or death. It over stimulates your brain cells. Glutamate over stimulation of neurons can be compared to turning a light switch on and off rapidly and repeatedly until it stops functioning.

Glutamate can induce irritability, restlessness, anxiety, sleeplessness, panic attacks or even seizures. It is inhibited by serotonin and GABA. Many seizure medications are at least partly GABA enhancers or mimics, as are the sleep medications, and the benzodiazepenes, the minor tranquilizers Ativan, Xanax and Valium. GABA inhibits thyroid function, as thyroid hormone is very stimulating. It works by inhibiting TRH production in your hypothalamus. Most NT effects on your endocrine system act directly on your hypothalamus.

Glutamate is released with any inflammatory condition in your brain. It is present and significantly elevated in chronic neurodegenerative disorders such as Alzheimer’s Disease, Amyotrophic Lateral Sclerosis (ALS or Lou Gehrig’s Disease), and Parkinson’s Disease. Elevated levels of aluminum have been found in the brain cells of patients in all three of these diseases. Aluminum is a toxic metal that causes brain inflammation. Aluminum inhibits the production of acetylcholine, another important NT, in your brain. Low acetylcholine is one of the main features of Alzheimer’s and aluminum is implicated in this matter. Acetylcholine is important for brain function, nerve impulse transmission, attention and memory. Impacting the progression of any of these disorders is not a simple matter.

Dopamine is a central catecholamine NT. It is the least well understood of all the NTs, but it is important for cognition, focus, attention, memory, motor function and motivation. It has tremendous clinical significance. In excess, it causes you to act in an overly fixed and brittle manor. In extreme excess, it may produce psychotic thought disorder. The major anti-psychotic medications act to suppress dopamine.

Dopamine deficiency produces indifference, attention deficits, and rapid decay of motor ability. Parkinson’s Disease is associated with low dopa and dopamine levels, dopa being the immediate precursor to dopamine. In Parkinson’s Disease, the acetylcholine to dopamine balance may be important, in addition to the individual levels of dopamine and acetylcholine themselves. Parkinson’s disease is associated with low dopamine and high acetylcholine levels.

Estrogen maintains the integrity of your brain. Women are twice as likely to suffer from depression as men. Estrogen is implicated in this circumstance. Women lose brain function when their estrogen diminishes. Estrogen selectively enhances the hippocampus, the main site of memory in the brain. It also has a protective effect on the substantia nigra.

The dopamine motor system known to be involved in Parkinson’s. Low estrogen may lead to muscle twitches and restless legs. Drugs used for high cholesterol and other conditions in-
terfere with hormone production. Cholesterol-inhibiting drugs, like Lipitor, inhibit an enzyme, HMG-CoA-reductase, that is necessary for cholesterol production in the brain. HMG-CoA-reductase inhibitors cause dementia faster than normal aging, because cholesterol is a precursor needed to make estrogen, progesterone and testosterone.

Tamoxifen is also well documented to cause brain damage and memory loss by blocking estrogen reception. Raloxifene for osteoporosis, or Arimidex to reduce the conversion of testosterone and other adrenal hormones to estrogen, both increase your chances of developing brain malfunction. The drugs for high cholesterol, osteoporosis, or cancer that are prescribed to you are documented to cause brain injury, sometimes in multiple ways. The brain is 60 percent lipid (fat) by dry weight. Interference with lipid metabolism in the brain should not be undertaken lightly.

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