



Depression: *Superman to the Rescue!*

by Dr. Kelly J. Gibas



Anxiety, depression...depression, anxiety...which comes first? It's the familiar chicken/egg dilemma; what seems clear is the intimate association between anxiety and depression. In fact, researchers from John Hopkins University were bold enough to assert that, "anxiety does not always lead to depression, but *all depression* stems from anxiety." Why the intimate connection between stress and feelings of hopelessness and despair? The secret may be in the brain's evolutionary protection mechanism during times of chronic, unresolved stress. The HPTA (hypothalamus, pituitary, thyroid, adrenal) axis holds the key to the biological mystery of stress and depression initiated by an irregular chemical cascade in the brain, adrenals, and thyroid (everydayhealth.com).

Countless studies have proven that chronic adrenal stress depresses hypothalamic and pituitary function; in fact, stress creates a 'code red' for many body systems including immunity, reproduction, digestion and metabolism (Medicine for the 21st Century, C. Kressler, 2012). Stress slows the body down for 'fight or flight' of the immediate stressor; the 'code red' does not create an immediate biological problem, if the stressor is resolved in a timely manner. However, if the stress turns into chronic anxiety, the slow down wrecks havoc leading to physiological and psychological compromise. Unresolved fight or flight stimulates the adrenal cortex to release cortisol, the most powerful adrenal hormone. Prolonged cortisol elevation caused by chronic stress prompts the brain to repress the normal expression of thyroid hormones. This becomes extremely significant as hypothyroidism, or low thyroid, is a primary cause of depression (Kressler, 2012). In a healthy response, the hypothalamus triggers the pituitary gland to release TSH (thyroid stimulating hormone) based on the person's perceived activity level; TSH then speaks to the thyroid gland directing the amount of thyroid hormone to be release into the bloodstream. It is a feedback loop; the more thyroid hormone that is needed, the higher the TSH. With less demand for thyroid, the TSH level falls. The thyroid gland, at the direction of TSH, produces the thyroid hormones T4 (80%) and T3 (20%); T4 is a biologically *inactive* hormone, T3 is the *active* hormone. At the cellular level, key enzymes and carefully calculated levels of cortisol, convert the inactive T4 into active T3 in the tissue. This assimilation (T4 into T3) ensures proper hormone control in the cell, not just the blood. But, elevated cortisol changes this delicate feedback loop by blocking the conversion of T4 into T3.

When cortisol remains elevated, as with anxiety, the brain's sympathetic response is chronically triggered much like a key getting stuck on a keyboard. Excess cortisol alerts the brain to slow the metabolic rate by interrupting the usual thyroid loop of TSH, T4 to T3. With too much cortisol, T4 begins to convert into an inactive form of T3 called *reverse T3*, *rT3*. Reverse T3 is a normal compound, but under chronic stress the body produces too much resulting in what's known as '*reverse T3 dominance*.' This forces the body into a metabolic holding pattern until the stressor has been resolved; the brain makes an executive decision, 'I don't want to waste energy while in crisis, so I'll make rT3!' Reverse T3 is a mirror image of T3, but it is biologically inactive; rT3 *plugs* the receptor sites and functionally blocks the metabolism by stopping the action of the T3 hormone. RT3 is similar to the plastic strip covering the battery on a new clock; the plastic strip preserves the life of the battery until the clock is purchased. Once the clock is bought from the store, the owner pulls the strip and the battery begins to work. Unfortunately, for many people with chronic anxiety, the 'plastic strip' doesn't get pulled and their thyroid function is repressed indefinitely by excess reverse T3.

What does all of this have to do with depression? T3 is not only a powerful thyroid hormone regulating metabolism; it is also a key neurotransmitter facilitating serotonin uptake at the synapse receptor. T3 functions in a dual role like Clark Kent/Superman; it's a thyroid hormone by day and a neurotransmitter by night! When excess cortisol blocks T3 from the tissue (reverse T3), it is also blocking T3 from the brain, resulting in secondary depression (*Stop the Thyroid Madness*, J. Bowthorpe).



The problem? Most thyroid blood panels don't calculate reverse T3 or free T3 values; and, when the feedback loop of thyroid is disturbed due to excess cortisol, it is disturbed in the *middle of the chain*, T4 to T3. Meaning, TSH and T4 (the traditional markers for low thyroid) are usually always normal. The 'normal' thyroid lab leaves the depressed person without answers...and the familiar story goes on, and on, and on! *The answer?* Request additional thyroid tests, ask for a free T3 and a reverse T3; the ratio of free T3 to rT3 should be greater than 2. If not, supplementation with T3 can be extremely beneficial to alleviate depression (*Stop the Thyroid Madness*).

Traditional medications for low thyroid are synthetic T4 only, which are problematic if the root issue is anxiety. Levothyroxine type medications, like Synthroid, leave many hypothyroid patients with minimal relief; because, T4 is not what the body needs. However, natural T3 supplements, like Cytomel, deliver T3 directly to the tissue which challenges the metabolic 'plug' of reverse T3 and add more '*Superman*' in the synapse to unlock the serotonin receptors. ***Superman to the rescue!***

~Kelly