Persistent pain involves a heightened sensitivity to pain, lowered thresholds and occasions of spontaneous pain; non-painful stimulus may trigger pain. These changes arise due to alterations within the nervous system due to what is termed ?plasticity'.

The pain-modulating pathways in the brain and spinal cord involve the neurotransmitters serotonin and norepinephrine, which, as we have seen, are the chemicals also involved with depression. This is why antidepressant medication can be helpful in relieving pain.

The Tripartite Model of Depression

Clark and Watson ([1]) have described depression as being divided depression into 3 clusters of symptoms: serotonin related, catecholamine related, and general distress related.

Serotonergic (5HT)-related symptoms include tachycardia, profuse sweating (diaphoresis), rapid breathing, gastrointestinal upset, chest discomfort, etc. and are most commonly seen in anxious depressed patients.

Catecholaminergic (norepinephrine) symptoms from reduced activation include poor concentration, low energy, low motivation, and apathy. General distress, including fatigue, tension, restlessness, worry, and hopelessness, could be due to depletion of either of these neurotransmitters.

Patients tend fall into one of these categories, serotonin deficiency or norepinephrine deficiency or a combination of both.

Treating only one part of this tripartite problem is unlikely to be successful of there are

depressive symptoms related to the other neurotransmitter.

The symptoms of general distress can be treated from either end of the spectrum.

The majority of patients experience a constellation of symptoms that reflect a dual problem with the neurotransmitters, which means that medication that addresses both deficiencies is more likely to produce better results. (See below).

---

1. Clark LA, Watson D. Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. J Abnorm Psychol. 1991; 100:316-336.

