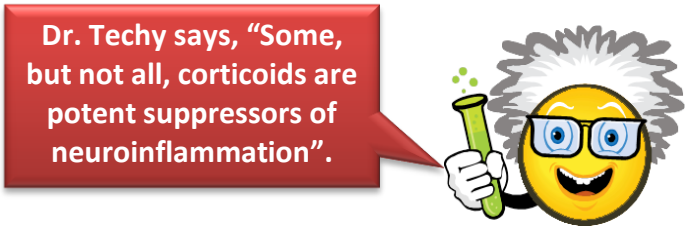
Hormones & Pain Care Information Network

JULY, 2016
BULLETIN 56



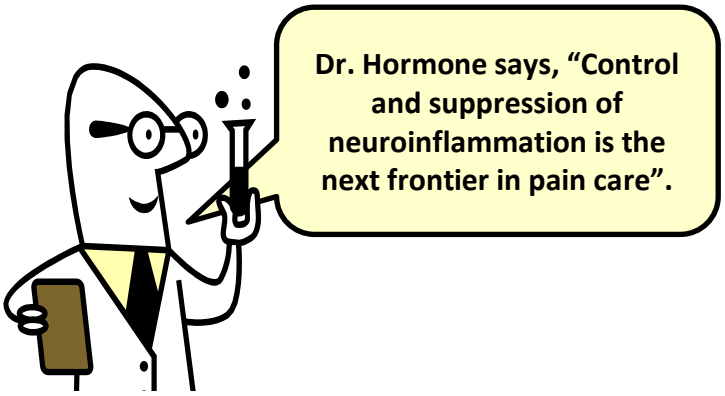
CORTISOL AND CONTROL OF NEUROINFLAMMATION

- ✓ It is now clear that activation of the microglial cell in the CNS is the major cause of neuroinflammation that may lead to central sensitization, neuroplasticity, and severe, chronic pain. Neuroinflammation is also responsible for multiple symptoms, impairments, and morbidities that may accompany pain including fatigue, depression, mental dysfunction, and overstimulation of the autonomic nervous and endocrine systems.

- ✓ At this time there is a concerted effort among some pain care investigators to develop clinical protocols to control microglial cell over-activation and neuroinflammation. We have made the development of such a protocol our top developmental priority.

- ✓ While several nutritional supplements, therapeutic agents, and neurohormones such as pregnenolone, DHEA, estradiol, and progesterone may all suppress microglial cells to some extent, cortisol that originates in the adrenal glands and migrates into the CNS is likely the most critical and potent hormone which controls microglial cell activation and neuroinflammation.

We now believe that severe centralized pain and neuroinflammation states such as arachnoiditis, RSD/CRPS, and post-viral syndromes require use of some specific corticoids to reduce pain and neuroinflammation. The next several bulletins will detail our discoveries and our protocol using some specific corticoids to control neuroinflammation.



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