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HORMONES AND LIBIDO

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Libido, a latin word that means "desire", indicates an activated, unsatisfied mental state of variable intensity, created by external -via the sensory modalities- or internal stimuli -hormones, fantasy, memory, cognition...- that induces a feeling of a need or want to partake of sexual activity to satisfy the need. Three main components: **biological,** both endocrine and neurochemical, **motivational** and **relational,** contribute to the individual, variable levels of libido. Hormones are the necessary but **not** sufficient factors to maintain a satisfying human libido. In women, estrogens prime the Central Nervous System, acting as neurotrophic and psychotrophic factors during the female life. They prime as well the sensory organs that are the key receptors for external sexual stimuli. Sensory organs transmit the basic information that, mixed with emotional and affective messages, contribute to the structuring of core sex identity and self image, so relevant for the personal perception of being an "object of desire" and for the direction of the libido itself. The interplay between estrogens and the dopaminergic system is the key process in determining the **appetitive** side of sexual behaviour, which can be definitely thrilled by androgens. Prolactin has an inhibiting effect on libido. Progestins act as sedatives. Hypothyroidism may inhibit libido, whilst hyperthyroidism does not have a specific positive effect on sexual desire. Hormones seem to control the **intensity** of libido and sexual behaviour, rather than its direction. This is a major challenge in therapy, even in post-menopausal women.

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TACHYPHYLAXIS: A NOVEL APPROACH TO MANAGEMENT

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A syndrome of tachyphylaxis in response to oestrogen implant therapy can occur in which increasing doses of oestradiol produce reduced clinical effect despite increased doses often being used. This can lead to oestradiol levels four to five times normally accepted levels. Management has generally involved oestrogen therapy cessation, however hitherto no specific treatment has been reported. We report a series of five women who presented with a syndrome characterised by irritability and lability of mood, fatigue, sleep disturbance and stress sensitivity in addition to symptoms consistent with high oestrogen levels (oedema and nasal congestion). All had been treated with repeated oestrogen implants and had markedly elevated serum oestradiol levels. None had a history of preexisting mood disturbance and their symptoms commenced following implant therapy. Three of the five subjects showed a marked response to the selective serotonin reuptake inhibitor paroxetine, with one subject responding to the use of a tricyclic agent (doxepin). These findings suggest that tachyphylaxis may be mediated via serotonergic pathways in the central nervous system.