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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
psychotropic 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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TACHYPHYLAISIS: 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 oestriadiol produce
reduced clinical effect despite increased doses often being used.
This can lead to oestriadiol 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 oestriadiol 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.