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The American Psychiatric Publishing textbook of mood disorders. Targeting peptide and hormonal systems / Stuart N. Seidman hormonal systems / Stuart N.

In addition, acute infections lead to a marked increase in cytokines. While this may, in part, be due to small amounts crossing the bloodâ€"brain barrier Banks et al. This glial interleukin-1, in turn, results in decreased release of acetylcholine and cognitive impairment. Cytokines may also play a role in the pathogenesis of dysphoria associated with physical illness. Hormones and aging As shown in Table 1. This has led to an enthusiasm for hormone replacement as a modern-day fountain of youth. Unfortunately, with the exception of the data for estrogen and testosterone replacement, the controlled studies in humans have failed to match the promise of animal studies. Dehydroepiandrosterone DHEA and its sulfate show the largest decline of any of the hormones with aging. The function of this adrenal cortical hormone remains elusive. Replacement studies in middle-aged persons suggested an improved mood Morales et al. In men, but not in women, high doses of DHEA mg daily have increased muscle mass and strength Morales et al. However, while some epidemiological studies have suggested that DHEA may be correlated with the decline in functional status with aging Morrison et al. Melatonin is produced by the pineal gland. Descartes originally suggested that the pineal was the seat of the soul. Continued studies on the potential role of melatonin on mental function are warranted. The recent availability of orally active growth hormone secretagogues may allow replacement with more physiological levels of growth hormone. Arginine vasopressin AVP levels increase during the day but the nocturnal surge is attenuated with aging. This is responsible for the well-known nocturia that occurs with aging. In addition, AVP has been demonstrated in animals to enhance memory. In rodents the decline in AVP that occurs with aging is related to the decline in testosterone see below and can be restored with testosterone replacement. Cortisol levels show no change or a small increase with aging. This is most probably predominantly due to the decrease in cortisol production rate that occurs with aging. Thyroxine levels remain stable throughout the lifespan as the decrease in thyroxine production rate is balanced by a decrease in thyroxine clearance. Triiodothyronine levels decline slightly in persons beyond 80 years of age. Norepinephrine increases in the young old and epinephrine in the oldâ€"old. This is due predominantly to a postreceptor defect. This postreceptor defect leads to a decreased catecholamine responsiveness with aging. This may result in altered stress responses in older individuals. Estrogen and cognitive dysfunction The rapid fall in estrogen in women at the time of the menopause is the most dramatic hormonal change that occurs with aging. These include an increase in choline acetyl transferase, an increase in cholinergic neuron survival and an increase in axonal sprouting and dendrite spine formation. Estrogen stimulates nerve growth factor. A number, but not all, cross-sectional studies have suggested that estrogen use is associated with better cognitive function than non-estrogen use. Morley One controlled study in nursing home residents suggested improved function in those residents receiving estrogen Birge, Other small controlled trials have found some improvement in cognition with estrogen use in postmenopausal women. Progesterone, which is usually co-administered with estrogen, is amnestic Farr et al. Testosterone and behavior There are now multiple cross-sectional studies demonstrating that testosterone, free testosterone and bioavailable testosterone albumin plus free testosterone decline with aging Morley et al. This decline is due primarily to a failure of the gonadotropin-releasing hormoneâ€"pituitary unit, and thus luteinizing hormone fails to rise as testosterone declines. Testosterone replacement reverses the memory and learning defects seen in these mice. Cross-sectional studies have suggested that bioavailable testosterone is a major factor in age-related cognitive decline Morley et al. Two interventional studies in older males have shown that testosterone replacement results in enhanced visualâ€"spatial cognitive function Janowsky et al. Testosterone has also been clearly demonstrated to increase libido Hajjar et al. Testosterone replacement results in increased strength Sih et al. Androgen deficiency in aging males Questionnaire 1. Do you have a decrease in libido sex drive? Do you have

a lack of energy? Have you lost weight? Are your erections less strong? Have you noted a recent deterioration in your ability to play sports? Are you falling asleep after dinner? Has there been a recent deterioration in your work performance? Depression and the hypothalamicâ€"pituitaryâ€"adrenal axis Some of the most important advances on the understanding of hormones and behavior in the last decade have come from the increased recognition of the role of the hypothalamicâ€"pituitaryâ€"adrenal axis in depression Chapter 3. Stress activates this axis through the actuation of hypothalamic corticotropin releasing factor CRF. CRF levels are elevated in the majority, but not all, persons with depression. CRF appears to play a particularly important role in the genesis of the vegetative signs of depression. These include sleep disturbance, decreased locomotion, anorexia, and weight loss. Older persons with depression are more likely to have anorexia and weight loss than young depressives Fitten et al. In addition, the elevated glucocorticoid levels in depression lead to hippocampal neuronal loss and impaired cognition the pseudodementia syndrome of depression in older persons. The elevated cortisol levels also increase the likelihood of older depressed women to develop osteoporosis Michelson et al. Alcoholism is associated with direct activation of the hypothalamicâ€"pituitaryâ€"adrenal axis Willenbring et al. This could, in turn, result in some of the depressive symptoms seen in older alcoholics. An interesting therapeutic development in the treatment of late life depression has been the use of electromagnetic brain stimulation EBS. Morley The potential of the treatment of vegetative symptoms, such as weight loss, in older depressives with CRF antagonists is enormously exciting. Anorexia of aging and hormones Food intake declines throughout the lifespan, and beyond the age of 70 years, there is a declining in body mass Morley, In addition to this there is a decrease in the ability of the gastric fundus to relax to accept normal sized meals. Leptin is a peptide hormone produced by fat cells Morley et al. It plays a role in decreasing intake through inhibiting neuropeptide Y in the central nervous system. A number of neurotransmitters within the hypothalamus drive food intake. Dynorphin an opioid peptide increases fat intake and neuropeptide Y increases carbohydrate intake. Both of these peptides decline with aging. Levels of the mRNA for nitric oxide synthase also decline with aging Morley et al. Depression is the most common cause of anorexia and weight loss in older persons Wilson et al. The potential relationship of this condition to sex hormone changes in the elderly is in need of exploration. Late life paranoia can also be associated with refusal to eat because of fear that the food is poisoned. Dementia is most often associated with failure to eat, apraxia of swallowing, and weight loss. In the midstages of dementia some patients develop hyperphagia. Ingestion of unpalatable objects, e. The endocrinology of disturbances of food intake and metabolism associated with aging represents one of the cutting edge issues in geriatric psychiatry. Conclusions As illustrated in Fig. In older persons hormonal alterations can lead to depression, delirium, dementia, and panic attacks. Depression or a decline in food intake can lead to 11 Endocrine basis of geriatric psychiatry Figure 1. The interaction of hormones and behavior in the aging brain. Aging can result in a decline in hormonal levels making the aging brain more vulnerable to developing cognitive defects. Cytokines produced by the immune system can directly alter both hormones and behavior. With the increased computer power in the next millennium, together with full knowledge of the human genome, we should be much more capable of understanding these complex interactions and developing new approaches to the management of mental disorders in older persons. However, it is perhaps best to close with the comment of the English philosopher, Emerson Pugh: Blood-borne interleukin-1 alpha is transported across the endothelial bloodâ€"spinal cord barrier of mice. Journal of Physiology, 2 , â€" Is there a role for estrogen replacement therapy in the prevention and treatment of dementia? Journal of the American Geriatric Society, 44 7 , â€" Psychotherapy and Psychosomatics, 67 3 , â€" Carpal tunnel syndrome and gynaecomastia during growth hormone treatment of elderly men with low circulating IGF-1 concentrations. Clinical Endocrinology, 39 4 , â€" Journal of Biological Rhythms, 13 6 , â€"8. Physiology and Behavior, 58 4 , â€" Journal of the American Geriatric Society, 37 5 , â€" Learning and memory in the SAMP8 mouse. Neuroscience and Biobehavioral Review, 22 1 , 1â€" Pregnenolone sulfate enhances post-training memory processes when injected in very low doses into limbic system structures: Proceedings of the National Academy of Sciences, 92 23 , â€"

Chapter 2 : Stuart Seidman | Columbia University - blog.quintoapp.com

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References Introduction In most clinical situations Hypercalcemia is caused by primary hyperparathyroidism PHP or malignancy [1,2]. Soon it appeared that a considerable part of patients with Hypercalcemia of malignancy HHM also showed an increased urinary cAMP excretion. This was found in particular in hypercalcemic patients without bone metastases However in contrast to patients with PHP normal or low levels of 1. The HHM syndrome occured in particular in patients with squamous cell solid tumors of the bronchus, head and neck and in the renal adenocarcinoma [8]. Therefore it was important that the occurence of the HHM syndrome in breast cancer was demonstrated by Isales et al. If this is a harbinger for the development of Hypercalcemia is not known. What is well known that with resection of the tumor all biochemical parameters disappear. It is also known that the renal adenyl cyclase system in these patients shows a diminished stimulation capability [17]. In this mini -review the identification, physiological and pathophysiological role and future possible deveopments of PTHrP will be discussed. Alternative splicing generates many different mRNA species which encode three separate is forms of , or amino acids. The portions of the genes encoding the amino-terminal part are highly homologous such that the peptides share 8 of the first 13 amino acids and a similar secondary structure over the next 21 amino acids. The amino-terminal end of PTH contains 34 amino acids. PTHrP is produced in low concentrations in virtually all tissues. The physiological role of PTHrP remains incompletely understood. PTHrP has a fiunction in: Transepithelial calcium transport in the kidney and mammary gland. Smooth muscle relaxation in the uterus, bladder, gastrointestinal tract and arterial wall. Cellular differentiation and apoptosis of multiple tissues. PTHrP is an indispensible component of successful pregnancy and fetal development. Embryonic gene deletion is lethal in mammals. This PTH1 receptor belongs to the G protein-coupled receptor family. PTHrP is the master regulator and has widespread paracrine actions. The acions of recombinant PTH , as an anabolic agent in the treatment of osteoporosis mimic PTHrPs actions locally in the bone microenvironment [27]. There are only three identified circumstances in which PTHrP species are present in the circulation and act in an endocrine manner: Lactation in which PTHrP is made in the breast and reaches the circulation [30]. Fetal life, where PTHrP regulates maternal-to-fetal calcium transport [31,32]. Hence the vast majorities of PTHrP actions are paracrine in nature. Mice lacking PTHrP die at birth because all bones resulting from endochondrial bone formation develop improperly. As a result the rib cage is small and inappropriately mineralized. This phenotype was not seen in mice with PTH deletion. Recombinant PTH , teriparatide Forteo is approved for the treatment of osteoporosis but may cause bone resorption, hypercalcemia, nausea, muscle cramps and other adverse effects [38]. Although intermittent treatment with PTHrP causes an anabolic response continous treatment causes paradoxical downregulation of osteogenic genes resulting in skeletal catabolism [39,40]. This activity reflects the strong similarity of the secondary and tertiary structure despite the differences in primary amino acid sequence in this region [41]. Global knockout of PTH in the mouse resulted in hypocalcemia and hyperphosphatemia and a bone phenotype of increased trabecular and cortical bone volume the opposite of PTHrP null mice of the same age [34]. This suggests that PTH physiologically does not function to promote bone formation but rather acts as a regulator of extracellular fluid calcium postnatally and in the fetus and is essential for providing calcium for mineralization of bone. Its physiological role after development is that of a local regulator of bone remodeling, formation and resorption. Mice lacking both the midregion NLS nuclear localizing site and the carboxy terminal part after knock-in PTHrP while retaining the bioactive amino terminal region showed multiple abnormalities and early lethality at weeks age. Homozygous mice showed skeletal retardation and decreased bone mass with reduced proliferation of osteoblasts and increased apoptosis of osteoblasts. Together with fewer osteoblasts there was less trabecular and cortical bone and fewer osteoclasts. Impaired growth plate chondrocyte proliferation resulted in mice with markedly shorter long

bones. They differed quite clearly from the severe chondrodystrophy of PTHrP null mice, which resulted from premature differentiation of chondrocytes [35]. The PTHrP knock-in mice exhibited also early senescence in multiple tissues. The brain showed decreased neural proliferation and increased apoptosis with abnormal shape. PTHrP is needed in maintaining normal synapsis and plasticity. These effects are partly mediated by the PTH2 receptor [42]. In a second model, knock-in of PTHrP , which excludes a significant portion of the mid-region, resulted in a an even more severe phenotype with similar skeletal abnormalities as chondrodysplasia but also impaired hemopoiesis and mammary development, dysregulated energy metabolism and death by 5 days of age. So many actions of PTHrP are not mediated solely by the amino-terminal fragment [43]. In mice as soon as the mammary bud begins to form epithelial cells produce PTHrP which interacts with the PTH1 receptor expressed on surrounding mesenchymal cells. This interaction is necessary for proper differentiation of the dense mammary mesenchym. The formation of the human breast in human fetesus is similar to that in mice and PTHrP is indispensible for that as well [46]. During puberty PTHrP seems to regulate the growth of the mammary ducts in response to estrogen [47,48]. PTHrP is also made by breast epithelial cells during lactation and large quantities are secreted in milk [49,50]. Milk production requires a great deal of calcium, an important source of which is the maternal skeleton. During lactation elevated levels of PTHrP correlate with bone loss in human and circulating levels of PTHrP correlate directly with rates of bone resorption and inversely with bone mass in mice [51,52]. The lactating breast also expresses the CaR which signals to suppress PTHrP secretion in response to calcium delivery to the breast [52]. These interactions define a classical negative feedback loop whereby mammary cells secrete PTHrP to mobilize calcium from the bone. Calcium in turn feeds back to inhibit further PTHrP secretion from the breast. Therefore during lactation the breast and bone engage in a conversation which leads to the mobilization of skeletal calcium to ensure a steady supply of calcium for milk production. Interestingly fish PTHrP shows a similar function to mobilize calcium stored in scales to be used for egg production [53]. PTHrP and the placenta During pregnancy calcium must be actively transported across the placenta from mother to fetus. The calcium concentration in the fetus is higher than in the mother so that calcium must be transported against a gradient [54]. This suggests that fetal PTHrP is important in placental calcium transport from the mother [32]. Experiments in sheep and mice showed that the midregion PTHrP is responsible for placental calcium transport and not the amino-terminal portion [32,57]. It is in the neonatal period that intestinal calcium absorption and thereby skeletal development and mineralization become dependent upon vitamin D3 and calcitriol [58]. A few cases of overproduction of PTHrP in the placenta resulting in hypercalcemia have been described [59,60]. Stretching of the muscular cell or structure increases the expression of PTHrP. PTHrP works as a relaxant then in turn on the structure that has been stretched in an autocrine or paracrine way [22,61,64]. For organs like the bladder, stomach or uterus this may be an important feedback loop allowing gradual filling. In the vascular wall PTHrP is expressed by vasoconstrictive agents, stretch it and acts as a vasodilator to resistance vessels. In this way PTHrP may act as a local modulator of blood flow [65]. PTHrP expression is also stimulated by pathologic stimuli including atherosclerosis, restenosis after balloon angioplasty and hypertension [66]. In addition to its relaxant effect PTHrP inhibits vascular smooth muscle cell proliferation through its interaction with the PTH1R and stimulation of the cAMP-protein kinase a pathway resulting in cell cycle blockade in the G1 phase. These effects are mediated by the amino-terminal fragments of PTHrP. PTHrP may also have proliferative effects on vascular smooth muscle cells by translocating in the nucleus leading to inhibition of vascular proliferation. These effects are mediated by the mid-region fragments of PTHrP [67,68]. These results suggest that a single molecule may have opposite effects under physiological and pathological conditions [69]. PTHrP has been found in cardiomyocytes and co-localizes with atrial natriuretic peptide in granules within the atrial cells in the rat heart [70]. In PTH1R deficient mice widespread cardiomyocyte death occurred in midgestation. PTHrP has both positive inotropic and chronotropic effects and may affect coronary blood flow [71,72]. In beta-cells it is stored within secreting granules and co-released with insulin [73]. Pancreatic islet cells express the PTH1 receptor. Overexpression of

PTHrP in beta cells leads to an increased beta cell mass, hyperinsulinemia and hypoglycemia due to increased beta cell proliferation, increased insulin production and inhibition of beta cell apoptosis [74,75]. Because of the death of these mice at birth the role of PTHrP in the physiology of islets is not known. Neuroendocrine tumors secreting PTHrP resulting in hypercalcemia has been described [76,78]. PTHrP and the central and peripheral nervous system PTHrP and the PTH1 receptor are both widely expressed within the brain including the cortex, the cerebellum, the hippocampus, hypothalamus and pituitary [79,80]. PTHrP secretion by neurons is regulated by calcium influx through L-type channel activity on depolarization. PTHrP is also present in glia and astrocytes. High PTHrP levels in glial tumors correlate with a poor prognosis []. PTHrP expression increases in sites of ischemic brain injury where it may play a protective role by enhancing blood flow [83]. PTHrP inhibits the proliferation of damaged and dedifferentiated Schwann cells in peripheral nerves in this way contributing to nerve regeneration [86]. The brain contains also PTH2 receptors. PTHrP has no role in this system [87]. Teeth and PTHrP Developing teeth are surrounded by bone and must erupt through the roof of the dental crypt to emerge into the oral cavity. This requires spatial coordination of bone cell activity. Osteoclasts must resorb the bone overlying the crown of the tooth to allow it to emerge and osteoblasts must form bone at the base of the tooth to propel it upward out of the crypt. PTHrP is produced by stellate reticulum cells and it signals to dental follicle cells to promote the formation of osteoclasts above the crypt. In the absence of PTHrP these osteoclasts do not appear, eruption fails to occur and the teeth become impacted [88,90]. Various attempts have been made to develop assays that target the N-terminal or C-terminal peptide.

Chapter 3 : Peptide Therapies

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Along Roads Less Traveled: Gerbarg, and Richard P. The metabolism of these vitamins is intimately linked and supports the synthesis of S-adenosylmethionine SAMe , the major methyl group donor in methylation reactions. This article reviews the metabolic and clinical importance of folate, vitamin B12, and SAMe, as well as clinical trials in relation to depression and dementia. Freeman Over the past 2 decades, omega-3 fatty acids n-3FAs have been increasingly used and studied in the United States and worldwide for various medical and psychiatric indications. Numerous published clinical trials have examined applications of different n-3FA preparations, primarily in mood disorders but also in psychotic disorders, attention-deficit disorder, obsessive-compulsive disorder, and personality disorders. Focusing on clinical issues, this article reviews the impact of n-3FAs on these conditions and covers the relevant research, side effects, dosage guidelines, and drug interactions; clinicians should thus be able to better advise patients who are already taking n-3FAs or are interested in trying them. Brown The choice of nutrients for review is based on clinical evidence of efficacy in neuropsychiatric disorders and biochemical effects that are neuroprotective or reparative. Vitamins, minerals, amino acids, and metabolites have been shown to augment antidepressants, improve symptoms in anxiety disorders, depression, neurodegenerative diseases, brain injury, ADHD, and schizophrenia, and to reduce medication side effects. Detection and correction of vitamin and mineral deficiencies can be essential for recovery. Generally low in adverse effects when taken in therapeutic doses, nutrients can be combined for greater benefits. Further studies are warranted to validate these promising treatments. Gerbarg and Richard P. Brown Herbal medicines supported by evidence of safety and efficacy in the treatment of anxiety, insomnia, fatigue, cognitive enhancement, mental focus, and sexual function are useful as monotherapies, multiherb combinations, and as adjuncts to prescription psychotropics. Relevant mechanisms of action and clinical guidelines for herbs in common use can assist clinicians who want to enhance treatment outcomes by integrating phytomedicinals into their treatment regimens. Research is needed to strengthen the evidence base and to expand the range of disorders that can be treated with herbal extracts. Studies of herbal genomic effects may lead to more targeted and effective treatments. Panossian This article focuses on the most extensively studied adaptogens: Rhodiola rosea, Eleutherococcus senticosus, and Schisandra chinensis. Clinical studies, evidence for stress-protective and simulative effects, and molecular mechanisms of action on metabolic and other processes regulated by the neuroendocrine system are discussed. Mechanisms of action, including emerging pharmacogenetic data, safety, and clinical considerations are also detailed. Indications, Mechanisms, and Safety 73 Bruce J. Diamond and Mary R. Bailey Ginkgo biloba special extract EGb is used in most randomized control trials. Indications include cognition and memory in Alzheimer disease, age-associated dementia, cerebral insufficiency, intermittent claudication, schizophrenia, and multi-infarct dementia. Mechanisms of action include increasing cerebral blood flow, antioxidant and antiinflammatory effects, with antiplatelet effects attributed to flavone and terpene lactones. Possible interactions with monoamine oxidase inhibitors, alprazolam, haloperidol, warfarin, and nifedipine have been reported. Saffron, Passionflower, Valerian and Sage for Mental Health 85 Amirhossein Modabbernia and Shahin Akhondzadeh Many cultures have developed folk herbal remedies to treat symptoms of mental illness. An evidence-based view is now being developed for some of these so-called alternative herbal treatments. This article discusses clinically relevant scientific information on medicinal extracts of 4 herbs: Ancient Yoga texts and Modern Studies 93 Shirley Telles and Nilkamal Singh The practice of yoga is gaining in popularity with a wide range of practices. Recent research and descriptions from the ancient texts are often concurrent with regard to the effects of the practice, taking into account expected differences between modern scientific terms and those used in the original texts. Voluntarily regulated yoga breathing practices form a bridge between physical and

mental changes. The voluntarily regulated yoga breathing has distinct effects on metabolism, the autonomic nervous system, higher brain functions, and mental state. The effects of meditation on the nervous system and mental state are even clearer. Evidence suggests that these practices may be effective at treating a range of physical health conditions, and at improving health-related quality of life. There is growing interest in the use of Tai Chi and Qigong to treat mental disorders, because they are noninvasive, exercise-based therapies, and because patients with mental disorders frequently use complementary and alternative medicine. Evidence is promising that these treatments may be effective in reducing depressive symptoms, stress, anxiety, and mood disturbances. Gerbarg, and Fred Muench Neurophysiological studies may explain how breathing techniques normalize stress response, emotion regulation, and autonomic and neuroendocrine system function. Breath practices have been shown to reduce symptoms of stress, anxiety, insomnia, post-traumatic stress disorder, mass disasters, depression, and attention deficit disorder. Technology assisted breathing interventions facilitate therapeutic breathing by using either static cues such as a breath pacer or real-time feedback based on physiological parameters such as heart rate variability. The empirical literature indicates that technology-assisted breathing can be beneficial in mental health treatment, though it may not be appropriate for all individuals. Initial in-person training and evaluation can improve results. Marchand Mindfulness meditation-based therapies are being increasingly used as interventions for psychiatric disorders. MBSR is beneficial for general psychological health and pain management. MBCT is recommended as an adjunctive treatment for unipolar depression. Informed clinicians can do much to support their patients who are receiving mindfulness training. This review provides information needed by clinicians to help patients maximize the benefits of mindfulness training and develop an enduring meditation practice. Fehmi and Susan B. Shor This article describes the role of attention training and brainwave synchrony training in the resolution of stress- and pain-related symptoms. It describes the origin of Open Focus attention training as it was distilled from observations of space-generated brain wave activity. It provides a map of the various attentional styles and associated EEG activity. It has special application where patients have adverse reaction to psychopharmacologic treatments and psychotherapy, cognitive behavioral therapy, and dialectical behavior therapy have proved ineffective. Neurofeedback is strong in clinical confirmations of efficacy case studies and has thus far limited controlled studies in the peer-reviewed journals. Kirsch and Francine Nichols Cranial electrotherapy stimulation is a prescriptive medical device that delivers a mild form of electrical stimulation to the brain for the treatment of anxiety, depression, and insomnia. It is supported by more than 40 years of research demonstrating its effectiveness in several mechanistic studies and greater than clinical studies. Often used as a stand-alone therapy, because results are usually seen from the first treatment, cranial electrotherapy stimulation may also be used as an adjunctive therapy. Seidman and Mark Weiser Age-associated hypothalamic-pituitary-gonadal HPG axis hypofunction, or partial androgen deficiency of the aging male, is thought to be responsible for various age-associated conditions such as reduced muscle and bone mass, mobility limitations, frailty, obesity, sleep apnea, cognitive impairment, sexual dysfunction, and depression. It has been difficult to establish consistent correlations between these symptoms and plasma testosterone levels in middle-aged men, but testosterone replacement does lead to improved muscle strength, bone density, and sexual function. This article focuses on the relationship between testosterone and mood in older men, and the treatment of age-related depression with exogenous testosterone. Beyond Psychopharmacology Kelly Brogan A discussion of pharmacologic and nonpharmacologic management of mental disorders in the pregnant woman is presented, with the focus on alternative health approaches and nutrition awareness. The article explores some considerations of modifiable risk factors thought to play a role in epigenetic manifestations of infant and child illness. Several case examples show the potential for integrative medicine in patients of reproductive age. Complementary, Alternative, and Integrative Treatments Psychotropic medications have revolutionized the treatment of serious mental disorders, yet in a significant number of cases, they are partially effective or ineffective. Psychotropics are necessary for many patients but they can contribute to the burden of side effects, and the cost of psychotropics contributes to the cost of health care and disposal of these

medications may cause environmental pollution. Phytomedicines, nutrients, and mind-body practices tend to be less costly and to have fewer side effects. Although psychotropics and psychotherapies will continue to be mainstays of psychiatric practice, specific combinations of herbs and nutrients can enhance the effectiveness of prescription drugs or reduce the necessary doses. Moreover, nutritional and phytomedicinal compounds can prevent or counteract various acute and long-term side effects of medications such as fatigue, Parkinsonian symptoms, akathisia, and elevated hepatic enzymes. Integrative psychiatrists are finding that mind-body practices can facilitate progress in psychotherapy. Identifying safe and effective nutrients, phytomedicines, and mind-body practices is therefore vital to better mental health care. Integrative treatments provide the clinician with additional therapeutic tools and empower the patient to participate actively in recovery. We have invited authors to focus on treatments supported by an evidence base of significant benefits, associated with few and modest side effects. From the wide array of complementary, alternative, and integrative medicines CAIM , we chose to include diverse points of view from experts who are well known, as well as from those whose work is not widely read by mainstream psychiatrists but who are highly regarded in their fields. The authors have been tasked with discussing the evidence base, neurophysiology, risks, benefits, and clinical applications for each treatment. Due to space limitations, commonly accepted and widely published treatments such as hypnosis and acupuncture are not included. To accommodate as much content as possible, several authors have opted to allow the publisher to post most of their reference lists online, retaining only key references with their articles. Modern research is rediscovering and improving on the benefits of nutrients, herbs, and mind-body practices. Every culture has used local medicinal plants whose active constituents can now being analyzed. The neurophysiologic changes that underlie psychiatric disorders involve multiple mechanisms, metabolites, anatomic structures, and neuro-endocrine networks. Nutrients and herbal extracts contain bioactive substances that can scavenge free radicals, protect cellular structures, enhance mitochondrial energy transport, increase production of neurotransmitters, upregulate or downregulate genes, and replenish vital metabolites. The rationale for integrating treatments is that targeting multiple etiologic factors often results in better outcomes than targeting only one, such as a particular neurotransmitter. The scientific measurement of psycho-neuro-immuno-hormonal and genomic changes induced by mind-body practices opens a vast domain for treatments derived from spiritual, meditative, fitness, and brain stimulation techniques. Studies are finding that mind-body interventions can activate or mute neural networks involved in emotion regulation. Such interventions act to balance stress response systems, including the autonomic nervous system and the hypothalamic-pituitary-adrenal axis. Among thousands of mind-body practices, one can discern certain common healing elements, for example, breathing practices. Initially developed prior to BC in India as well as in Asia, Africa, Polynesia, and the Americas, these techniques reappeared in medieval monasteries and martial arts. Today such practices are used in yoga classes and in Special Forces training. It is not surprising that such time-tested treatments show significant clinical benefits in randomized controlled trials. Developing specific mind-body programs for various psychiatric conditions and treatment settings is an appealing future direction. Political, economic, and environmental forces are driving large-scale natural and man-made disasters. Relying solely on expensive pharmaceuticals or one-on-one therapies will not address the global epidemic of depression and posttraumatic stress disorder. Affected populations need inexpensive, accessible, safe, sustainable treatments. The large-scale cultivation of medicinal herbs is increasing available supplies. Local teachers, care providers, clergy, and community leaders can be trained to provide and to train others in self-healing mind-body practices. Resiliency training could help at-risk communities prevent or recover from the psychological sequelae of traumatizing events. Mind-body programs could also enable members of the military to endure combat stress better and recover from service-related posttraumatic stress disorder. Integrative psychiatry seeks to enrich mainstream mental health care with valuable treatments from global healing traditions as well as from modern laboratories in related fields, such as neurofeedback, breath pacing, and genomics. Interest, support, and research are growing, but much more is needed to strengthen the evidence base and to refine treatments for specific conditions. Educating ourselves, our peers, and our patients

is essential for the safe and optimal use of CAIM approaches.

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[et al.] -- Selective serotonin reuptake inhibitors and newer antidepressants / Richard C. Shelton and Natalie Lester -- Lithium and mood stabilizers / Paul E. Keck Jr. and Susan L. McElroy -- Antipsychotic medications / Stephen M. Strakowski and Richard C. Shelton -- Targeting peptide and hormonal systems / Stuart N. Seidman.

Advanced Search To the editor: We write to highlight a relevant underreporting in an article reported in JCEM. Specifically, in the report by Steidle et al. Allow us to place this in context. There is a clinical consensus among endocrinologists and andrologists that testosterone replacement enhances mood in hypogonadal men 2 , 3. We have been especially interested in looking at systematically collected data that can establish such effects empirically. Counterintuitively, in a randomized clinical trial conducted by one of us S. We reviewed published data from clinical trials of testosterone replacement for frankly hypogonadal men in which mood was systematically assessed. In studies with at least 10 subjects, we found only one placebo-controlled trial of testosterone replacement that reported mood 5. In this study, the lack of a mood effect was reported in just one sentence, and the data were not shown. Most authors who reference improved mood with testosterone replacement use the influential study by Wang et al. In this and a similarly designed trial by McNicholas et al. Both studies demonstrated large improvements in positive moods and reduction in negative moods 6 â€” 8. Notably, Wang et al. Yet, importantly, no placebo controls or placebo substitutions were included in these studies, leaving the question open as to whether such enhanced mood might have been equally detectable in a placebo group of hypogonadal men who thought they might be receiving testosterone. Mood was considered a primary outcome, and patients rated positive moods alert, full of energy, friendly, well, or good and negative moods angry, irritable, sad or blue, tired, nervous on a 0â€”7 Likert scale. Yet, in the nine-page report, only one sentence describes the mood results: The data collected for every other a priori outcome of interest was reported in meticulous detail using text, figures, and tables; for mood, it was limited to just this one sentence. Admirably, this was the largest placebo-controlled testosterone replacement study ever done. Because mood is the least studied of the presumed hypogonadal symptoms, missing this opportunity to better detail the mood effects of testosterone replacement is a loss for science and the public health 9 , These public health issues have proponents who advocate testosterone replacement for mood. Data bearing on these issues helps clinicians make well-informed clinical decisions, rather than expose men to unwarranted treatment. To avoid even the appearance of conflict of interest, it would be good editorial practice to ensure that studies supported by a pharmaceutical company gave proper attention to unwelcome findings. Acknowledgments A response to this letter was invited, but the authors of the original article chose not to provide one.

Chapter 5 : The American Psychiatric Publishing textbook of mood disorders (Book,) [blog.quintoapp.com]

Seidman SN Targeting peptide and hormonal systems. In: Stein DJ, Schatzberg AF, Kupfer DJ, eds. Textbook of mood disorders. In: Stein DJ, Schatzberg AF, Kupfer DJ, eds. Textbook of mood disorders.

We write to highlight a relevant underreporting in an article reported in JCEM. Specifically, in the report by Steidle et al. Allow us to place this in context. There is a clinical consensus among endocrinologists and andrologists that testosterone replacement enhances mood in hypogonadal men 2 , 3. We have been especially interested in looking at systematically collected data that can establish such effects empirically. Counterintuitively, in a randomized clinical trial conducted by one of us S. We reviewed published data from clinical trials of testosterone replacement for frankly hypogonadal men in which mood was systematically assessed. In studies with at least 10 subjects, we found only one placebo-controlled trial of testosterone replacement that reported mood 5. In this study, the lack of a mood effect was reported in just one sentence, and the data were not shown. Most authors who reference improved mood with testosterone replacement use the influential study by Wang et al. In this and a similarly designed trial by McNicholas et al. Both studies demonstrated large improvements in positive moods and reduction in negative moods 6 , 7 , 8. Notably, Wang et al. Yet, importantly, no placebo controls or placebo substitutions were included in these studies, leaving the question open as to whether such enhanced mood might have been equally detectable in a placebo group of hypogonadal men who thought they might be receiving testosterone. Mood was considered a primary outcome, and patients rated positive moods alert, full of energy, friendly, well, or good and negative moods angry, irritable, sad or blue, tired, nervous on a 0â€”7 Likert scale. Yet, in the nine-page report, only one sentence describes the mood results: The data collected for every other a priori outcome of interest was reported in meticulous detail using text, figures, and tables; for mood, it was limited to just this one sentence. Admirably, this was the largest placebo-controlled testosterone replacement study ever done. Because mood is the least studied of the presumed hypogonadal symptoms, missing this opportunity to better detail the mood effects of testosterone replacement is a loss for science and the public health 9 , These public health issues have proponents who advocate testosterone replacement for mood. Data bearing on these issues helps clinicians make well-informed clinical decisions, rather than expose men to unwarranted treatment. To avoid even the appearance of conflict of interest, it would be good editorial practice to ensure that studies supported by a pharmaceutical company gave proper attention to unwelcome findings. A response to this letter was invited, but the authors of the original article chose not to provide one. J Clin Endocrinol Metab Vermeulen A Diagnosis of partial androgen deficiency in the aging male. Ann Endocrinol Paris Morley JE Testosterone replacement in older men and women. J Gend Specif Med 4: J Clin Psychiatry Testosterone Gel Study Group. Seidman SN Targeting peptide and hormonal systems. Textbook of mood disorders. American Psychiatric Press, in press.

Chapter 6 : Complementary and Integrative Therapies for Psychiatric Disorders - Breath Body Mind

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The outcomes discussed below are not representative for all patients. Peptides are molecules that consist of between two to fifty amino acid sequence. The first commercial peptide therapy used was insulin What are peptide therapies? Fundamentally, peptide therapies give cells the ability to change cell behavior and to handle stress better. Peptide therapies are natural modulators of cell signaling. You can get started and just use a growth hormone releasing peptide GHRP , eg, Ipamorelin by itself without spending a lot of money to see if your sleep improves and bone density improves. Use a GHRP together with bio-identical hormones and get great results. There are 7, natural peptides in the body. There are currently 60 FDA-approved peptides in clinical use. There are now clinical trials happening at this moment in time October and there are some clinical studies being set up right now. As a forward-thinking medical practice, we now embrace peptide therapies as an additional mainstay treatment for acute and chronic illness. Our armamentarium of tools has increased. Peptide therapies have a variety of applications such as immune enhancement; accelerates injury repair to muscles, reverses sarcopenia loss of muscle , tendons, bones, nerves; progressive fat loss and improved bone mineral density, increase in Testosterone by increasing pulse size and frequency, decrease inflammation in the brain and the rest of the body, natural release of growth hormone, improves sleep. Hormone Optimization and Growth Hormone Secretagogues: Sexual Enhancement and Function: Melanotan II and PT Peptides, as signaling substances, help reclaim efficiency of hormone receptor sites. Peptides and certain other nutrients can help improve the functionality of the receptor so that the hormones we give work as intended. Importantly, peptide therapy works best in the background of great nutrition, cortisol balance, and the removal of environmental toxins. Melanotan II increases melanogenesis for photoprotection and increased tanning, plays a role in improving autoimmune disease, eg, Lyme disease. Plays a role in sexual dysfunction and can improve libido increases sexual arousal and erectile dysfunction in men and sexual desire in females through the CNS, and not through the Cardiovascular System which is the way medicines like Cialis, and Viagra work. Has significant anti-inflammatory disease. Can affect appetite and cause weight loss. Works well with intermittent fasting. Activation of the vagus nerve leads to cholinergic signaling and inhibits tumor necrosis factor TNF and other pro-inflammatory cytokines overproduction Cholinergic anti-inflammatory pathway. This pathway is activated by Melanocortin 3R and 4R receptors. This pathway is critical and is significant in anti-inflammation in the brain. This plays a role in neuroprotection and is also cardioprotective, too. The melanocortin system peptides can be used with Thymosin alpha-1 to balance Th1 and Th2 issues. This can also be used with DSIP with Glycine where you can upregulate glutathione peroxidase and superoxide dismutase. This gives us new approaches to autoimmune diseases. Immune cells have melanocortin receptors 1,3,5. Melanocortin receptor 3,4 is in the brain. Melanocortin receptor 4 plays a role in appetite control. The adrenal cortex is melanocortin 2. Alpha melanin-stimulating hormone reacts with all 5 receptors. Melanotan receptor 1 is on the endothelial cell. Melanotan 1 does not cross the blood-brain barrier. Melanotan 2 crosses the blood-brain barrier. Melanotan 1 will not give you sexual desire effects but will give you tanning and is responsible for melanogenesis. Melanotan 2 gives you both tanning sunless tanning and sexual desire. In women, they can increase pigmentation and more freckles than with men. The face and hands may get darker than the rest of the body. The use of Melanotan 1 is an art, follow the skin pigmentation to determine dosing adjustments. It has the cosmetic effect of tightening the collagen in the face. Psoriasis may also be treated by Melanotan 2, but when treatment is stopped, the disease will come back. Melanotan 2 works through the vagus nerve through the cholinergic anti-inflammatory pathway. Can be useful for Seasonal Allergy because of the anticholinergic effects. Can be used on a daily, or regular basis vs. PT bremelanotide which should only be used twice weekly to prevent desensitization. Melanotan 2 is neuroprotective, as well as plays a role in

decreasing opioid addiction and ethanol consumption. Melanotan 2 improves libido and erectile dysfunction in men. Melanotan 2 plays a role in appetite and metabolism. Swiss Analytic Labs claims to have tested most if not all peptides advertised on the internet. Be particularly skeptical of marketing for very inexpensive peptide therapies. It simply is impossible for reputable companies to manufacture these products and make money at very low prices. Growth Hormone causes weight loss of both visceral deep fat and retroperitoneal fat, whereas Testosterone and exercise are responsible for the superficial fat loss. Affects lipid metabolism women greater than men. Best for weight loss of pounds. Promotes chondrocyte production of collagen and proteoglycan. The success rate for weight loss improved with work-outs or fasting. AOD with HA hyaluronic acid useful for cartilage regeneration in knee, hip, shoulder, ankle, and tendinopathies. AOD with HA- also good for trigger point injections. MK is fraught with side effects such as involution of receptor sites in the brain and irreversible neurologic damage. It also elevates cortisol and increases depression and anxiety. Also has muscle repair function and anti-inflammatory effects. It has no side effects, no toxicity, and no drug interactions. It helps the body to achieve homeostasis. It is a powerful cell signaling messenger that decreases inflammation and accelerate healing muscle, brain, bone, tendon, nerve, ligament, cornea, intestines, as examples. It increases fat loss, improved immune function, well being, and bone mineral density. Some patients claim that their chronic pain has been substantially reduced. BPC can help you heal faster from injury to bone, muscle, ligament, tendon, nerve, brain, teeth, intestine, cornea via cell signaling. This peptide responds specifically to injury. It also has an influence on neurotransmitters related to stress, anxiety, mood, and behavior via its effects on the serotonergic, dopaminergic, GABAergic and opioid systems. May help with depression. It has effects on the GI tract via its anti-ulcer, cytoprotective effects. It improves GI mucosal integrity anecdotal info for healing leaky gut mcg, orally, twice daily. It improves nitric oxide NO. Can be used after initial use of steroids for acute anti-inflammatory purposes thus prevents the long-term adverse effects of steroid use. Ameliorates alcohol and opioid withdrawal symptoms may be combined with DSIP , and opposes alcohol intoxication. Helps with homeostasis of dopaminergic and serotoninergic systems. Improves nerve axonal and myelin sheath regeneration. It rapidly and permanently counteracts QTc prolongation of the heart caused by neuroleptic medicines Haldol, etc and prokinetics. Intranasal use for Lyme and Mold brain fog. Cerebrolysin is a neuro-regenerative and neuroprotective peptide. It is a low molecular weight peptide that can cross the blood-brain barrier. It can be used to increase memory and learning and can improve synaptic function and synaptic density. Protects nerves from free radicals and oxidative stress damage and improves the metabolic activity of neurons, and protects neurons from neurotoxic effects of glutamate. Enhances cognitive function, memory, learning, creativity, and motivation. It can cross the blood-brain barrier and decreases beta-amyloid formation and deposition as well as Tau protein phosphorylation. It has a positive effect on behavior and neurotrophic stimulation. In dementia, improves neuronal cytoarchitecture which results in improved cognitive and behavioral performance. This can help mitigate the continued cognitive impairment. Significant improvement was seen in those with mild to moderate dementia. Deep or Delta Sleep Inducing Peptide DSIP This peptide not only helps regulate sleep by way of improving the circadian function of sleep mostly stage 4, slow wave sleep, when growth hormone is released and decreasing wake ups throughout the night. It is not a sedative drug. There are no significant side effects from treatment. Oxidative stress in the brain causes an age-associated decrease in Testosterone.

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Stuart N. Seidman and Donald F. Klein Seidman SN Targeting peptide and hormonal systems. In: Stein DJ, system, with no adverse effects on the prostate as measured by symp-.

Chapter 9 : Volume 89 Issue 12 | The Journal of Clinical Endocrinology & Metabolism | Oxford Academic

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