EW0516

Sexual dysfunction in oncology

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Introduction Sexual dysfunction is a common consequence of cancer treatment that affects at least half of men and women treated for pelvic tumors and more than one quarter of individuals with other malignancies.
Objectives/aims Identification of the main sexual dysfunctions related to cancer treatments. Awareness to the importance of addressing sexuality to cancer patients, identifying the main reasons why healthcare providers usually do not.
Methods Literature review concerning researched articles published in Pubmed/Medline as well as related bibliography.
Results Most sexual problems are not caused by the cancer itself, but by toxicities of cancer treatment. Damage during cancer treatment to pelvic nerves, blood vessels and organ structures leads to the highest rates of sexual dysfunction. The most common sexual dysfunction in men under cancer treatment is the loss of desire for sex and erectile dysfunction. In women, the most common sexual dysfunctions are vaginal dryness, dyspareunia and loss of sexual desire, usually accompanied by difficulties in both the arousal and orgasm phases. According to literature, there are many cancer patients who would like to be informed and advised by their healthcare providers about the consequences of cancer treatment on their sexual health. Unfortunately, this rarely happens.
Conclusions This work intends to publicize current existing information on sexual dysfunction in oncology, focusing on the prevalence, etiology and clinical presentation. The authors also intend to promote communication about sexual function and possible sexual dysfunctions resulting from cancer treatments.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.130

EW0517

Structural covariance networks in anorexia nervosa (AN): A multimodal graph theoretical analysis

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Introduction The possibility of evaluating cortical morphological and structural features on the basis of their covariance patterns is becoming increasingly important in clinical neuroscience, because their organizational principles reveal an inter-regional structural dependence which derive from a complex mixture of developmental, genetic and environmental factors.
Objectives In this study, we describe cortical network organization in anorexia nervosa using a MRI morpho-structural covariance analysis based on cortical thickness, gyrification and fractal dimension.
Aim Aim of the research is to evaluate any alterations in structural network properties measured with graph theory from multi-modal imaging data in AN.
Methods Thirty-eight patients with acute AN, 38 healthy controls and 20 patients in full remission from AN underwent MRI scanning. Surface extraction was completed using FreeSurfer package. Graph analysis was performed using graph analysis toolbox.
Results In acute patients, the covariance analysis among cortical thickness values showed a more segregated pattern and a reduction of global integration indexes. In the recovered patients group, we noticed a similar global trend without statistically significant differences for any single parameter. According to gyrification indexes, the covariance network showed a trend towards high segregation both in acute and recovered patients. We did not observe any significant difference in the covariance networks in the analysis of fractal dimension.
Conclusions The presence of increased segregation properties in cortical covariance networks in AN may be determined by a retardation of neurodevelopmental trajectories or by an energy saving adaptive response. The differences between the analyzed parameters likely depend on their different morpho-functional meanings.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.131

EW0518

The link between sleep, stress and BDNF

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The protein brain derived neurotrophic factor (BDNF) is a major contributor to neuronal plasticity. There is numerous evidence that BDNF expression is decreased by experiencing psychological stress and that accordingly a lack of neurotrophic support causes depression. The use of serum BDNF concentration as a potential indicator of brain alteration is justified through extensive evidence. Recently, we reported, for the first time, a relationship between BDNF and insomnia, since we could show that reduced levels of serum BDNF are correlated with sleep impairment in control subjects, while partial sleep deprivation was able to induce a fast increase in serum BDNF levels in depressed patients. Using a bi-directional stress model as an explanation approach, we propose the hypothesis that chronic stress might induce a deregulation of the HPA system leading in the long term to sleep disturbance and decreased BDNF levels, whereas acute sleep deprivation, can be used as therapeutical intervention in some insomniac or depressed patients as compensatory process to normalize BDNF levels. Indeed, partial sleep deprivation (PSD) induced a very fast increase in BDNF serum levels within hours after PSD which is similar to effects seen after ketamine infusion, another fast-acting antidepressant intervention, while traditional antidepressants are characterized by a major delay until treatment response as well as delayed BDNF level increase. Moreover, we revealed that stress experience and subjective sleep perception interact with each other and affect serum BDNF levels. We identified sleep as a mediator of the association between stress experience and serum BDNF levels.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.132