Summary

Depression and other mental disorders are the leading cause of disability worldwide and their burden has increased considerably over past decades. However, advances in psychopharmacology of psychiatric disorders are not in measure with this negativ trend. As a result, a large body of research in psychiatry and neurosciences tries to further our understanding of pathophysiological mechanisms underlying mood disorders and other mental illnesses in order to improve the efficacy of current treatments and to identify new therapeutic agents. According to current evidence, stress-related pathways and inflammation processes are directly involved in the development of depressive disorder and several other psychiatric conditions. The study of the effects and consequences of stress exposure requires an interdisciplinary approach, taking into account specific aspects of the "inputs", such as chronic stress and traumatic experiences, and related psychological processes, with the crucial role of dissociation.

Following these theoretical findings, the empirical research performed in two cohorts of inpatients with depressive disorder focused on immune and endocrine responses to stress and their relationship to psychopathological symptoms, specifically trauma-related symptoms, psychic and somatoform dissociation and depressive symptoms.

The main findings of the empirical research shows that TNF-α, that has been intensively studied as one of the major candidates in the cytokine model of depression, is related to both psychic and somatoform dissociative symptoms and trama-related symptoms. This finding suggests that TNF-α regulation and dissociation could be interrelated in the complex response to stress.

Other main results of this study indicate that prolactin is associated with psychic dissociative symptoms, supporting the findings of previous research, pointing to the relationship between prolactin regulation and passive coping behaviour. Finally, cortisol has been found to be related to somatoform dissociation, suggesting that low level of cortisol could be associated with active defensive or antiarousal intrapsychic processes, such as avoidance, withdrawal and denial.

Although no clinical conclusions can be taken at this point, the findings encourage further efforts to describe more in details the interplay between TNF-α, prolactin and cortisol regulations, chronic stress and dissociative processes in the development and clinical manifestation of depression.