Introduction

About 10% of children older than 5 years in the community can present with a mental disorder (Meltzer et al. 2000). It is well established over the past thirty years that childhood chronic disorders, such as diabetes, asthma, rheumatic disease, cystic fibrosis, and sickle cell anemia, can significantly increase the risk of mental disorders (Breslau et al. 1985; Gortmaker et al. 1990), with the emotional adjustment affected by the severity of the condition and the degree of functional limitation. Children with diseases involving Central Nervous System present the highest psychopathological risk (Weiland et al. 1992; Howe et al. 1993). When matched with disabled children with other disorders (i.e., musculo-skeletal), children with cerebral pathologies presented a two-fold higher rate of psychiatric disorders, even when IQ, social context, and physical disability were controlled for (Seidel et al. 1975).

The notion that chronic cerebral disorders strongly increase the risk of psychiatric disorders in pediatric populations is supported by a classical, rigorous epidemiologic study, the Isle of Wight study (Rutter et al. 1970). Psychiatric disorders were found in 44% of children with structural brain lesions, compared with 12% in children with non-cerebral physical disorders and 7% in children without physical disorders. Hyperkinetic disorder was 90 times higher in children with cerebral palsy or epilepsy. Even though childhood psychiatric disorders are more common in males, the gender effect was lost in this study when a brain lesion was co-existent. However, two-thirds of the children with cerebral palsy or epilepsy were free from any psychiatric disorder.

Emotional well-being, psychological and behavioral disorders, and quality of life have been explored also in children and adolescents with cerebral palsy, and these data will be discussed in this review. Most of the studies included patients with hemiplegia, usually the mildest type of cerebral palsy, while less information is available on the rate of psychiatric disorders across all forms and severities of cerebral palsies. A major limitation in most of these studies is that the assessment is not based on specific psychiatric instruments which can allow for a specific psychiatric diagnosis. For this reason, the psychopathological risk is usually reported in terms of psychological dimensions rather than psychiatric diagnoses.
A screening questionnaire should not be considered as an equivalent of a psychiatric assessment, and emotional and behavioral symptoms are not always associated with a psychiatric disorder (Goodman and Yude, 2000), even though the existing cut-offs increase the likelihood to identify a clinical problem.

**Brain Disorders and Psychopathology**

The analysis of the association between brain lesions and behaviour disorders during infancy and childhood presents important theoretical implications, as it can elucidate the biological underpinnings of psychological development, as well as the role of early brain plasticity. Psychiatric consequences of childhood brain disorders can be affected by different mechanisms, biological as well as non-biological. Biological mechanisms include the characteristics of brain lesions (timing, size, site, side), associated cognitive impairment (mental retardation, neuropsychological deficits), comorbid disorders (epilepsy, visual or hearing deficits). Non-biological mechanisms include the effects of impaired sensori-motor and speech skills on personality development (sense of personal identity, relationship with external and social environment), psychological effects of functional impairment, stigmatizing effect of the disorder, impact on familial relations (strength of psychological familial resources), and the environmental resources affecting quality of life. Furthermore, neurological and psychosocial variables interact, although the nature of their mutual interaction in the development of psychiatric disorders is still not clear (Goodman, 2002). A brain lesion may only amplify the effect of psychosocial adversities on psychopathological risk, or, on the contrary, biological and non-biological mechanisms may operate on independent pathways in determining psychiatric disorders (Breslau, 1990). The complexity of the relation between brain lesion and psychiatric disorders is illustrated by Lewis et al’s study (1990), which reported on two identical twins with a genetic vulnerability for schizophrenia. One of them had cerebral palsy, while the other developed a schizophreniform psychosis, suggesting that brain damage does not necessarily make a genetic vulnerability more likely to be expressed.

Regarding lesion timing, early damage is less associated with clinically significant emotional and behavioral disturbances, as the brain’s potential for reorganization, based on the plasticity of the newborn CNS, can partly compensate the effects of the damage, while this capacity is less effective in later occurring lesions (Trauner et al. 2001). Regarding lesion size, findings from patients with hemiplegia and/or focal lesions report less psychiatric impairment compared to unselected samples of children with cerebral palsy (Goodman and Graham, 1996; Goodman and Yule, 1997), suggesting that bilateral and/or extensive damage may be more frequently associated with a co-occurring psychopathology. Regarding lesion site, Trauner et al. (1996) compared school-aged children with anterior and posterior perinatal focal damage (stroke) and found that posterior damage was associated with social problems (assessed with the Personality Inventory for Children), while anterior damage was associated with academic difficulties. However, differences in Externalizing, Internalizing, and Total score of the Child Behavior Checklist