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Rassegna - Review

Neurochimica della anoressia neoplastica

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Neurochemical basis of cancer anorexia

ABSTRACT: Under physiological conditions, energy intake is tightly controlled in the hypothalamus by: i) transducing metabolic/sensorial inputs arising from the periphery into neuronal response; ii) integrating the information originating from different tissues; and, iii) triggering the appropriate feeding responses. Data suggest that cancer anorexia is secondary to a disruption of the physiological mechanisms controlling energy intake. It is therefore conceivable that its pathogenesis may lie in an abnormal input of information to the hypothalamus, or in its defective transduction and integration, or in the induction of exaggerated and inappropriate feeding responses. There is general consensus about the multifactoriality of the pathogenesis of cancer anorexia, which involves most of the neuronal signalling pathways modulating energy intake. Thus, a number of factors has been proposed as putative mediators of cancer anorexia, including hormones (e.g., leptin), neuropeptides (e.g., NPY), cytokines (e.g., IL-1, IL-6, TNF) and neurotransmitters (e.g., serotonin and dopamine), and close interrelationships appear to exist among them. In line with this reasoning, consistent experimental and human data suggest that hypothalamic monoaminergic neurotransmission, and particularly serotonergic activity, may represent a major target on which different anorexia-related factors converge. Thus, interfering pharmacologically with hypothalamic serotonin activity has been recently tested as a therapeutic strategy in anorexic cancer patients with encouraging results. Furthermore, it could be speculated that modulation of hypothalamic activity may also result in reduced catabolic signals to skeletal muscles, thus improving cancer cachexia as well. (RINPE 2002; 20: 21-8)

KEY WORDS: Cancer, Anorexia, Neurotransmission, Hypothalamus, Serotonin

PAROLE CHIAVE: Tumore, Anoressia, Neurotrasmissione, Ipotalamo, Serotonina