Nitrous Oxide Antinociception and Opioid Peptides

It is hypothesized that stimulation of neuronal release of endogenous opioid peptide and the subsequent activation of opioid receptors underlie the antinociceptive action of the anesthetic agent nitrous oxide [9, 10]. In addition, this indirectly induced antinociception is mediated by 6-opioid receptors [9, 10]. Cahill et al [2] provided further information on the endogenous opioids involved. Based on the mouse abdominal constriction test, they reported that nitrous oxide antinociception is mediated through neuronal release of dynorphins and methionine-enkephalin but not β-endorphin in the spinal cord.

Capacitative Calcium Entry in Vascular Smooth Muscle Cells

Capacitative calcium entry occurs through store-operated channels that open in response to depletion of sarcoplasmic reticulum calcium [1]. Gardner and Benoit [6] measured Ca²⁺ currents in Atr5 vascular smooth muscle cells, in the presence or absence of several commonly used drugs that elicit a calcium response. They conclude that capacitative calcium entry can occur in aortic smooth muscle cells but this entry does not appear to contribute significantly to the response of the latter to vasopressin.

Phosphatidylethanol and Calcium-Dependent Cytosolic Phospholipase A₂ Activity

Phospholipase A is a calcium-dependent enzyme [13] that catalyzes the hydrolysis of arachidonic acid from membrane phospholipids. Various foreign and endogenous stimulants activate this enzyme and phosphatidylethanol in macrophages. Chang et al [3] observed from macrophage cell line RAW 264.7 that cytosolic phospholipase A₂ is stimulated by low, but inhibited by high, concentrations of phosphatidylethanol. The calcium-independent phospholipase A₂, on the other hand, is not affected. It is suggested that phosphatidylethanol may contribute to increasing cytosolic phospholipase A₂ in chronic ethanol treatment.

Chang et al. [4] demonstrated that the HIV-1-encoded Vpr protein exerts a repression on the G2 to M progression of the cell cycle. These authors further showed that this effect is virus-type- and cell-type-specific.

Molecular Characterization of Hepatitis E Virus from Namibia

Hepatitis E virus (HEV) infection is endemic in southeast and central Asia, the Indian subcontinent, northern and western Africa, and parts of north America (Mexico) [8]. However, it is increasingly more commonly seen in areas where HEV has previously been absent because of the increase in international travel. As HEV spreads through the fecal-oral route, it may cause huge epidemics that involve thousands of people. With the genome of this virus identified and characterized by molecular cloning in 1990 [11], further understanding of the virus is available. He et al [7] cloned an HEV isolate from stored fecal specimens collected from a patient at an outbreak in Namibia in 1983. Phylogenetic analysis showed that this HEV is closely related to other African isolates, but differs from previous Asian or American isolates. The African subgroup then joins the Asian subgroup to form a major monophyletic group. The study thus contributes to a construction of a better map of the global molecular epidemiology.

References