Poly (ADP Ribose) Synthetase Inhibitor Attenuates Ischemia-Reperfusion Liver Injury

Excessive activation of poly (ADP-ribose) synthetase (PARS) contributes to injury of hepatocytes caused by reactive oxygen species in vitro, and inhibition of PARS activity reduces the degree of reperfusion injury in the heart, skeletal muscle and brain in vivo [1, 21]. Chen et al. [4] further demonstrated that niacinamide, a PARS inhibitor, significantly attenuates ischemia-reperfusion-induced liver injury, reverses the decrease in blood ATP concentration and reduces the release of hydroxyl radical, TNF-α and nitric oxide. These results indicate that niacinamide exerts potent anti-inflammatory effects during ischemia-reperfusion-induced liver injury.

α1-Adrenoceptor Stimulation Enhances Dysfunction in Ca2+ Homeostasis in Failing Ventricular Tissues

Whereas α1-adrenergic responsiveness in isolated myocytes of 8-month-old BIO 14.6 hamsters is enhanced [18], Chen and Su [6] observed that stimulation of α1-adrenoceptors prolongs action potential duration and comparable positive inotropicism in normal or failing ventricular tissues. Further examination of the effect of phenylephrine on the force-frequency relationship and mechanical restitution showed a difference in the initial positive force-frequency relationship and a faster decay time constant for the postrest potentiation in failing ventricular tissues. This reveals a defect in intracellular Ca2+ handling in the failing heart that is not reversible by an α1-adrenergic mechanism. Furthermore, α1-adrenoceptor stimulation enhances a gradual loss of Ca2+ from intracellular storage.

Dopamine D1 and D2 Receptor Subtypes Are Involved in Anorexia Induced by Repeated Amphetamine Treatment

Repeated treatment with amphetamine induces anorexia on the first day, followed subsequently by tolerance [16]. Amphetamine also reinstates polydipsia induced by chronic activation of dopaminergic D2 receptors [8]. By repeated systemic administration of amphetamine in conjunction with dopamine agonists or antagonists in rats, Kuo and Cheng [12] found that both D1 and D2 receptor subtypes are involved in anorexia induced by repeated amphetamine treatment, as well as the tolerant feeding response to it.

Dermal Exposure to Methyl Parathion Changes Behavior and Blood Cholinesterase Activity

Pope et al. [17] suggested that changes in brain cholinesterase (ChE) activity after methyl parathion treatment could be estimated by alterations in blood ChE activity. Based on this suggestion, Zhu et al. [23] demonstrated that a single dermal exposure to subtoxic doses of methyl parathion causes reversible inhibition of blood ChE and changes in motor function. In contrast, repeated low-dose dermal exposure results in sustained inhibition of ChE activity and impairment of both motor function and memory retention.

Organophosphate Intoxication Involves Brain-Inducible Nitric Oxide Synthase

The organophosphate mevinphos (Mev) induces death by overstimulation of neurons in the rostral ventrolateral medulla (RVLM), the medullary origin of sympathethetic neurogenic vasomotor tone, via extensive accumulation of acetylcholine [22]. A likely mediator is the M2 subtype of muscarinic receptors (M2R), which are responsible for cholinergic regulation of systemic arterial pressure by the RVLM [9]. Based on laser scanning confocal microscopic analysis, Chang et al. [3] showed that M2R and inducible nitric oxide synthase (iNOS) are colocalized in RVLM neurons. In addition, coinjection of Mev with iNOS inhibitors into the RVLM significantly reverses the cardiorespiratory suppression that accompanies Mev intoxication. It is concluded that, as a cholinesterase inhibitor, Mev may induce toxicity via nitric oxide produced by iNOS on activation of the M2R by the accumulated acetylcholine in the RVLM.

Combined Treatment with Ribavirin and Interferon-α Elevates Interferon-γ Level

Combining ribavirin, a guanosine analogue with broad-spectrum antiviral activities, with interferon-α has been shown to be more efficacious than interferon-γ monotherapy [2, 13]. The underlying mechanism, however, is unknown. Fang et al. [7] reported that the level of interferon-γ produced by hepatitis C core antigen-stimulated peripheral blood mononuclear cells is elevated in patients receiving the combination therapy. The core-specific cytotoxic T cell activity is also increased. They proposed that these events may account for the increased efficacy of combining ribavirin with interferon-α in the treatment of chronic hepatitis C.

Hepatitis B Viral Poi Fusion Proteins Are Biologically Active

Hepadnaviruses [e.g. hepatitis B virus (HBV)] and retroviruses (e.g. HIV) are evolutionarily related families because they both require the process of reverse transcription for genome replication [10], al-
 though the respective source of polymerase is different [11, 15]. Chen et al. [5] found that secreted particles obtained from a human hepatoma cell line, HuH-7, transfected with a core-pol fusion protein show positive signals of HBV DNA. The fusion protein is also detected in the cytoplasm of the transfected cells. Immunofluorescence staining indicated that the HBV core-pol fusion protein is colocalized with the hepatitis C virus (HCV) core protein. These results suggest that the HBV core-pol fusion protein is actively involved in DNA polymerization and can form a complex with the HCV core protein.

**Sequencing and Expression of LUZP in Neural Lineage Cells**

LUZP is a novel leucine zipper motif-containing protein that is predominantly expressed in the brain [19] and putatively functions as a transcription factor [20]. After showing that LUZP is only expressed in the nucleus of neurons, Lee et al. [14] established that the Luzp gene has a composite sequence of 11,559 nucleotides. The Luzp knock-out/lacZ knock-in clone retains its potential to develop into tissue types derived from all three embryonic germ layers when injected into nude mice. However, LUZP expression is found exclusively in differentiated neural cells.

**References**


