New Candidate for Prolactin-Releasing Factor

Apart from a tonic inhibition by dopamine secreted from the tuberoinfundibular neurons, prolactin secretion from the anterior pituitary is under phasic stimulation by a yet unidentified prolactin-releasing factor [2, 6]. Although many candidates have been proposed, none fulfills all the required criteria. Yuan et al. [21] reported that a newly discovered candidate, prolactin-releasing peptide (PrRP), stimulates prolactin secretion in ovariectomized, estrogen-treated rats. The blockade of dopamine D\textsubscript{2} receptors potentiates this stimulatory action. PrRP-immunoreactive terminals are found in close contact with tyrosine-hydroxylase-immunoreactive neurons in the hypothalamic arcuate nucleus. It is concluded that PrRP may play a role in the control of prolactin secretion.

BH4 as a Potential Therapeutic Target for Postmenopausal Cardiovascular Diseases

Postmenopausal women exhibit a higher incidence of cardiovascular disease [19], which can be significantly reduced by estrogen replacement therapy [1]. To understand the underlying mechanism, Lam et al. [13] demonstrated that impairment of vascular reactivity in ovariectomized rats may be associated with a decrease in nitric oxide release and/or superoxide anion overproduction. They further pointed out that these may partly be related to a deficiency in BH4, since tetrahydrobiopterin improves vascular endothelial function in ovariectomized rats. These results provide the basis for a novel therapeutic strategy in the treatment of postmenopausal cardiovascular diseases.

Dopamine D\textsubscript{1} and D\textsubscript{2} Agonists Promote Hyperphasia

Whereas dopamine plays an important role in the regulation of feeding behavior [18], the underlying mechanism remains controversial. Kuo [12] observed that coactivation of dopamine D\textsubscript{1} and D\textsubscript{2} receptors additively reduces daily food intake, body weight and the neuropeptide Y level in the hypothalamus. Similar findings were obtained from diabetic rats. These results suggest that D\textsubscript{1}/D\textsubscript{2} agonists may promote hyperphasia during diabetes.

Bcl Upregulation by HIV-1 Tat during Infection of Monocytes/Macrophages

The ability of human monocytes/macrophages to host HIV-1 replication while resisting apoptosis is believed to be important for their biological role in serving as a viral reservoir in AIDS patients. Zhang et al. [22] reported that the expression of the antiapoptotic protein Bcl-2 is upregulated in monocytes and macrophages on infection by HIV-1. They further demonstrated that this upregulation is likely to be exerted through the HIV-1-encoded Tat protein [8].

Dermal Exposure to Methyl Parathion Poses the Greatest Health Risk

Methyl parathion is an organophosphorus insecticide which is widely used in agriculture as well as in private and business settings. The underlying mechanism for its toxicity in humans is the inhibition of acetylcholinesterase by its oxidative metabolite, methyl paraoxon [11]. Kramer et al. [10] compared that pharmacodynamics of methyl parathion based on the route of exposure. Since dermal administration induces a more gradual and prolonged inhibition of cholinesterase, they conclude that dermal exposure to methyl parathion poses the greatest health risk.

Chronic Exercise Upregulates iNOS Expression

Chronic exercise increases agonist-induced endothelium-dependent vasorelaxation and decreases agonist-evoked vasoconstriction by augmenting nitric oxide (NO) production [4]. Recent studies [5] also suggest that exercise training upregulates endothelial nitric oxide synthase (eNOS) gene expression. Whether chronic exercise also affects the expression of inducible NOS (iNOS) in blood vessels has not been examined. Yang et al. [20] found that both eNOS and iNOS are upregulated in endothelial cells after chronic exercise, although NOS gene expression in smooth muscle cells remains unaltered. They also showed that the elevated expression of iNOS may be responsible for the decrease in phenylephrine-induced vasoconstriction seen after chronic exercise.

Interaction between c-Jun and Sp1 in Phorbol Ester-Induced Activation of 12(S)-Lipoxygenase Gene Promoter

Phorbol 12-myristate 13-acetate (PMA) induces the expression of arachidonate 12(S)-lipoxygenase by activating its promoter activity in human epidermoid carcinoma A431 cells, and two Sp1 binding sites in the promoter are essential for this response [14]. Chen et al. [3] showed that PMA treatment induces ERK activation mainly through the Raf-MEK-ERK signaling pathway. The ERK activation is followed by c-Jun induction, which leads to
interaction between the c-Jun and Sp1. That the binding of the c-Jun/Sp1 complex to Sp1 is enhanced by PMA treatment suggests that the cooperative interaction between c-Jun and Sp1 plays an important role in PMA-induced activation of 12(S)-lipoxygenase gene promoter.

Hepatitis B Viral Serotypes and Genotypes in Taiwan

The serological heterogeneity of hepatitis B virus (HBV) has been established, and HBV isolates are classified into four major serotypes according to the antigenic determinants of hepatitis B surface antigen [16]. These subtypes of HBV have a specific geographic distribution and can serve as epidemiological markers. Taking advantage of the high prevalence of HBV infection and the different origin of inhabitants in Taiwan, Liu et al. [15] demonstrated that most HBV serotype adw are genotype B and all HBV serotype adr are genotype C. Patients with origins in mainland China have a higher proportion of serotype adr/genotype C infection.

Target Integration by Sp1 Zinc Finger Domain/Moloney Murine Leukemia Virus Integrase

A desirable feature of using retroviruses as vectors for gene transfer is that the virus can stably integrate a gene of interest into the host genome, thus contributing to the stability of the transduced gene. The entire integration process includes steps of 3′ processing and strand transfer is performed by a virus-encoded protein designated as integrase (IN) [7, 9]. However, since IN does not determine where to integrate the viral genome into the host cell chromosome, the problem of random integration exists. Peng et al. [17] demonstrated that IN

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