

column. The second Sephadex G-100 solution was not shown PO activity by microgram order of LPS and B-1,3-glucan. Nanogram quantity of soluble PG was specifically quantified by using this G-100 solution in vitro. Also, I purified and characterized specific PG recognition proteins from G-100 solution by using Dextran sulfate CL-6B column and Butyl-Toyopearl FPLC.

[PC1-40] [ 04/18/2002 (Thr) 14:00 - 17:00 / Hall E ]

#### Down-regulation mechanism of Senescence Marker Protein 30 by ROS during aging

Jung KyungJin<sup>o</sup>, Ishigami Akihito, Maruyama Naoki, Chung HaeYoung

College of Pharmacy, Pusan National University, Pusan 609-735, Korea, and Dep. of Molecular Pathology, Tokyo Metropolitan Institute of Gerontology, Tokyo, Japan

The senescent changes in the expression of functional proteins affect multiple deteriorative factors for various cellular activities and homeostasis. As the cause of deterioration during aging, reactive oxygen species (ROS) are well-known factors. Senescence marker protein 30 (SMP-30) plays an important role as a calcium binding protein that is known to be identical to regucalcin. The expression of SMP-30 that is preferentially exhibited in hepatocytes and renal tubular epithelia significantly declined during aging. It has been demonstrated that SMP-30 rescues cell death by enhancing plasma membrane Ca<sup>2+</sup>-pumping activity. However, recently, there is no information on the SMP-30 modulation by the anti-aging action of calorie restriction (CR). To characterize the status of SMP-30, the study explored the effect of aging on SMP-30 modulation by CR. The kidney and liver were isolated from Fischer 344 rats at 6, 12, 18, and 24 months of age fed ad libitum (AL) and CR rats. Results showed that SMP-30 expression markedly decreased during aging, whereas this decreased expression was clearly blunted by CR, showing a comparable level of 6 month-old AL rats. To investigate an aspect that age-induced ROS are related with SMP-30 gene expression, it was examined whether LPS-induced ROS affect gene expression of SMP-30 and DNA binding activity for nuclear protein. These results suggest that down-regulation of SMP-30 is reconciled with both age-related ROS and experimentally LPS-induced ROS.

[PC1-41] [ 04/18/2002 (Thr) 14:00 - 17:00 / Hall E ]

#### Regulation of Redox-sensitive Transcription Factors in aging process

Jung KyungJin<sup>o</sup>, Kim HyonJeen, Yu ByungPal, Chang Gregory Youngnam, Chung HaeYoung

College of Pharmacy, Pusan National University, Pusan 609-735, Korea, Department of Physiology, The University of Texas Health Science Center at San Antonio, Texas 78229-3900, USA, and Dept. of Neurology, University of Southern California, LA.

Oxidative stress is considered to be the major cause of aging and many age-related diseases. Calorie restriction (CR) is known to retard the aging, and age-related deleterious processes. Recent studies documented that CR retards the aging process with its anti-oxidative ability by regulating the intracellular redox balance. Among key cellular components exquisitely sensitive to the redox status are transcriptions factors such as nuclear factor kappa B (NF- $\kappa$ B), activator protein-1 (AP-1), and hypoxia inducible factor-1 (HIF-1). Currently, there is a limited information available on the age-related and dietary modulation on these factors. In this review, major focus was placed on whether age affects the regulation of NF- $\kappa$ B, AP-1 and HIF-1, and further to delineate how the age-related changes are modulated by CR. It is concluded that the age-related increases in redox-sensitive NF- $\kappa$ B, AP-1, and HIF-1 binding activities are associated with increased ROS, and further that CR modulates their activations by suppressing oxidative stress. Data on molecular regulation provides better molecular insights into the mechanisms underlying the cellular redox maintenance, which may reveals the cross-talk between the aging and age-associated pathogenic processes.

[PC1-42] [ 04/18/2002 (Thr) 14:00 - 17:00 / Hall E ]

#### Participation of Protein Disulfide Isomerase in Molecular Fate of Thyroglobulin and its Regulation by Endogenous Oxidants and Reductants