inactivation of PON. Based on these results, it is proposed that the PON inactivation during LDL oxidation may be ascribed mainly to the Cu^{2+}-catalyzed oxidation. This work was financially supported by research grant (1-209-003-2) from the Korea Science and Engineering Foundation, Korea.

[PC1-29] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Protein disulfide isomerase-catalyzed multimerization of partially reduced bovine thyroglobulin

Liu XiWen°, Sok DaiEun

College of Pharmacy, Chungnam National University

Multimerization of thyroglobulin (Tg) is known to be a way to store Tg at high concentrations in the lumen of the thyroid follicles. Multimerization of bovine thyroglobulin has been intermolecular reactions through the oxidative processes, such as disulfide and dityrosine formation, as well as isopeptide formation. It is known that the disulfide formation is more responsible for Tg multimerization than the other reactions. Here, we investigated the protein disulfide isomerase (PDI) or peroxidase-induced multimerization of normal bovine Tg or partially denatured thyroglobulins, which were prepared from the treatment with thiol, urea or thiol/urea, on the basis of SDS-PAGE analyses. In addition, the enzymatic multimerization was compared with non-enzymatic multimerization, thiol-mediated or thermal. The thiol-induced multimerization of normal Tg was dependent on GSH concentration, in the presence of low GSH concentrations, the dimeric form, 660 kD, was predominant, while multimeric forms were the major in incubations with higher GSH concentration. In addition, the multimerization of Tg under thermal denaturation at 60 ℃ also required glutathione. Meanwhile, the multimerization of partially reduced Tg was accelerated by PDI in a time-dependent manner up to 30 min at 38 ℃. Similarly, the multimerization of partially reduced Tg was also mediated by peroxidase. However, PDI showed no remarkable effect on the peroxidase-mediated or nonenzymatic multimerization. Accordingly, the PDI-mediated multimerization of thyroglobulin through the disulfide linkage may require a specific molecular form of thryglobulin, optimally reduced and denatured. This work was supported by Korean Research Foundation Grant (KRF-2000-F00302).

[PC1-30] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

BNip3 expression in hypoxia-induced neuronal apoptosis

Lee HyeJa°, Park JaeHoon, Rho YoungSoo

Department of Medicinal Chemistry and Pathology, College of Pharmacy and Medicine, KyungHee University, Seoul 130-701, Korea

Hyoxia/ischemia is one of the most common causes of neuronal injury and implicated in the pathogenesis of degenerative diseases, including dementia. It has been reported that hypoxia/ischemia induces acute neuronal necrosis or delayed cell death, depending on severity and duration of hypoxic injury. Hypoxia-induced delayed neuronal death (apoptosis) plays critical roles in the process of ischemia progression. Even though putative molecules mediating hypoxic injury, including p53 and Bax, were suspected, the precise molecular mechanisms are still unknown. Recently, it was shown that NBip3, a memember of pro-apoptotic Bcl-2 family proteins, was transcriptionally activated by hypoxic injury, suggesting certain roles in neuronal apoptosis. The object of this study is to clarify the functions of BNip3 in the apoptotic [rocess induced by hypoxia and we show evidences that BNip3 is one of the strong candidate molecules mediating hypoxic injury.

[PC1-31] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

The release of GPI-anchored proteins from the renal proximal tubules by Bacillus