were found dead (FD) or killed *in extremis* (KE) at their different exposure. These 56 terminated prematurely were 3 males and 5 females at the 1.5-yoe, and 26 males and 22 females at 2-yoe. The survival rate for 1.5-year study was 80% (12/15) in males and 66% (10/15) in females, and for 2-year study was 35% (14/40) in males and 55% (18/40) in females.

Pathology evaluation revealed a total of a total of 33 kinds primary tumors were observed in male and female LE rats. These tumors were 1) Schwannoma (heart), 2) hemangioma (heart), 3) hepatocellular adenoma /carcinoma, 4) lymphoma (Peyer's patches, mesenteric lymph node, multicentric), 5) islet cell adenoma/carcinoma, 6) acinar cell adenoma (pancreas), 7) malignant hibernoma (kidney), 8) renal tubule adenoma, 9) lipoma (kidney), 10) bladder papiloma, mediastinum), 11) uterine adenocarcinoma, 12) endometrial stromal polyp/sarcoma, 13) uterine leiomyosarcoma, 14) uterine squamous cell carcinoma, 15) Leydig cell adenoma, 16) phechromocytoma, 17) pheochromocytoma complex, 18) adrenal cortical adenoma, 19) thyroid follicular adenoma/ carcinoma, 20) C-cell adenoma, 21) pituitary pars distalis adenoma, 22) hemangiosarcoma (spleen), 23) large granular lymphocytic lymphoma (spleen), 24) granulocytic leukemia, 25) angiosarcoma (mesenteric lymph node), 26) thymic adenoma, 27) glioma/mixed glioma, 28) granular cell tumor (brain), 29) osteosarcoma, 30) fibroma/fibrosarcoma (skin, mammary), 31) mammary adenoma, 32) mammary fibroadenoma and 33) melanoma (ear pinna). Of these 33 kinds, 28 types were observed in males and 22 types in females of the entire LE rat population in this study. For further databases in detail of these 33 kinds, 5 kinds were observed in the 1.5 yoe rats. Of that 4 of the 5 kinds in the males and also other 4 of the 5 in the females and 28 kinds were observed in males and 22 types in females of the 2-yr exposure. One-granulocytic leukemia, of the 5 neoplasms detected in the 1.5-yoe rats, was not observed in the 2.0-yoe rats. These occurring in the male and female LE rats at 1.5-y and 2-y exposure are summarized in Table 1.

Of these 33 kinds primarily neoplastic diseases, a total of 21 male and female rats died from neoplastic diseases with 3 males and 2 females in the 1.5-yoe and 2 males and 7 females in the 2-yoe study. A summary of these are presented in Table 2.

Other information regarding neoplastic diseases occurring in these LE rats will be further presented.

[Session III] #11

Antioxidant Related Protein, Senescence Marker Protein-30 (SMP30), has an Inhibitory Effect on the Hepatic Fibrogenesis of Smad3-Mutant Mice

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The transforming growth factor-β1 (TGF-β 1) is thought to be an important cytokine in the regulation of the production, degradation and accumulation of extracellular matrix proteins, and Smad3 is a key modulator of the TGF-β1 signaling pathway. To investigate the influence of Smad3 on liver fibrogenesis, we observed histopathological findings and remarkable protein expression of Smad3-deficient mice liver after CCl_4 treatment. In Smad3-deficient mice, CCl_4-induced liver fibrosis was mild grade compared with the wild type. In a comparative proteomic analysis, different expression proteins of the CCl_4-treated Smad3-KO mice were identified as antioxidant related proteins, such as the senescence marker protein-30 (SMP30), selenium-binding proteins and glutathione S-transferase. In particular, increased the expression of SMP30 in Smad3-KO type could be reflected in significant proteomic changes. These results indicated that the Smad3 pathway was in correlation with the antioxidant defense system within liver injuries.

Our study provides initiative study of Smad3-regulated protein data profiles in Smad3-null mice liver and several specific antioxidant proteins were identified. Therefore, these data suggest that the alteration of specific antioxidant hepatic proteins, by the lack of Smad3 activation, will be bring about TGF-β1-mediated tissue damage. Furthermore, the specific control of Smad3-relavent proteins may be a critical step in the prevention of liver diseases.

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[Session III] #12

Am I Teaching Pathology Effectively?

- current debates and future designs -

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This article is to discuss current issues with regard to how to teach effectively veterinary pathology students in Korea. The old method of teaching veterinary pathology undergraduate courses in most of the 10 veterinary schools in Korea was based mainly in lecture and laboratory classes using traditional methods of teaching. The professor teaches the lecture class and it is usually a one-way learning process. The professor teaches and the students learn. Likewise, the laboratory class is taught by the professor and assisted by instructors. Laboratory class is mainly based on histopathological examination of collected cases. This method of teaching has drawn a lot of criticisms from students in that it is mainly traditional, discipline-oriented, teacher-centered curriculum and morphology-directed instruction.

With the advent of modern hi-tech electronic technologies the present methods of