[*Yakushigaku Zasshi*, **32**, 165-168 (1997)] [Lab. of Herbal Garden]

The Records of Jingoro Kodera, a Broker of Herbs in Kasuga Village (3). The Weight, Amount and Kind of Herbs Sold Written in His Sales Ledgers.

Tomomi TAKAKI and Toshihiro TANAKA*

Some names of herbs were written in twelve sales ledgers used from 1891 to 1924. These names include sweet hydrangea leaf, mixed herbs for baths, Japanese *Angelica* root and *Cnidium* rhizome. These sales ledgers were written by Jingoro Kodera, a broker of herbs who lived in Kasuga Village, at the foot of Mt. Ibuki. He sold the herbs in towns and cities on the east side of Lake Biwa, around Ichinomiya and on the west side of Ise Bay. He sold most of the herbs in the area on the east side of Lake Biwa and the least amount of herbs in the area on the west side of Ise Bay. Sales were greatest from 1896 to 1900, especially the sales of sweet hydrangea leaf. From 1891 to 1924, Jingoro failed to gain new customers and lost old customers. Many of his good customers were in the area on the east side of Lake Biwa.

[*Environ. Mutagen Res.*, **18**, 117-124 (1996)] [Lab. of Radiochemistry]

Comutagenic Effect of Bile Acids with 2-Aminoanthracene, 1, 2-Dimethylhydrazine and Pancreas-carcinogenic *N*-Nitroso Compounds.

Yukio MORI,* Midori TAKAHASHI, Kazunori IIMURA, Mitsuhiro KATADA, Akihiro KOIDE and Yoichi KONISHI

To study the role of bile acids in *N*-nitrosamine-induced pancreatic carcinogenesis in hamsters, the comutagenic activities of cholic, chenodeoxycholic, deoxycholic, lithocholic and ursodeoxycholic acids were examined in the presence of hamster liver S9-mix using nine pancreatic duct carcinogens. These included *N*-nitrosobis(2-hydroxypropyl)amine and its diacetyl, 2-oxopropyl, methyl and morpholine derivatives, together with 2-aminoanthracene (2-AA) and 1,2-dimethylhydrazine (DMH) as positive controls. In accordance with previous findings observed with rat liver S9-mix, the comutagenic effect with DMH and 2-AA in *S. typhimurium* TA100 and TA98, respectively, seems to be restricted to the secondary bile acids. In contrast, any of the five bile acids tested shows comutagenic activity on strain TA100 in the case of pancreatic carcinogens. The possible roles of metabolic activation in these comutagenic actions, and of bile acids in the initiation of pancreatic carcinogenesis, are discussed.

[*Spectrosc. Lett.*, **30**, 685-700 (1997)] [Lab. of Instrumental Center]

IR Study on Aqueous Solution Behavior of D-Cycloserine.

Ho-Hi LEE, Hisanori YAMAGUCHI, Hitoshi SENDA, Shiro MAEDA, Akio KUWAE and Kazuhiko HANAI*

IR spectra were measured for an antitubercular agent, D-cycloserine (CS), and its hydrolyzate, β-aminooxy-D-alanine (AOA), and the dimer, *cis*-3,6-bis(aminooxymethyl)-2,5-piperadinedione (CS-dimer), at various pH values in aqueous solutions. Molelcular species existing in the ionic equilibria were characterized by the pH dependence of the spectra. Band assignments were carried out by reference to the spectra of D_2O solutions and those of the related compounds. Spontaneous transformation of CS to CS-dimer occurs in the neutral aqueous solution. The spectral evidence suggests that the non-ionic form of CS plays a key role in the dimerization process.

[*Jan J. Human Genet.*, **42**, 353-356 (1997)] [Lab. of Clinical Pharmaceutics]

Polymorphism of Extracellular Superoxide Dismutase (EC-SOD) Gene: Relation to the Mutation Responsible for High EC-SOD Level in Serum.

Harutaka YAMADA, Yasukazu YAMADA, Tetsuo ADACHI,* Haruko GOTO, Nobuaki OGASAWARA, Arao FUTENMA, Mitsuru KITANO, Hironobu MIYAI, Atsushi FUKATSU, Kazuyuki HIRANO and Shinichi KAKUMU

Extracellular superoxide dismutase (EC-SOD) with amino acid substitution R213G generated by the nucleotide substitution 760C to G in the heparin binding domain is responsible for the high EC-SOD level in serum. We identified the two DNA polymorphic sites in the coding region of EC-SOD gene related to the 760C to G and determined the allele frequencies. The polymorphisms were A and G at nucleotide position (nt.) 241 and C and T at nt. 280 near the N-terminal. The haplotype frequencies in Japanese were 241A280C: 0.45, 241G280T: 0.37, and 241G280C: 0.18. The haplotype of 241A280T did not exist. The mutation 760C to G must occur on the allele having the haplotype of 241G280T.