

中華民國比較病理學會
八十七年度會員大會暨第十一次比較病理學研
討會
大會手冊

主辦單位：佛教慈濟綜合醫院

行政院農業委員會

臺灣省政府農林廳

中華民國比較病理學會

日期：中華民國八十七年三月一日(星期日)

地點:佛教慈濟綜合醫院二期講堂

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誌謝

中華民國比較病理學會八十七年度會員大會議程

時 間	活 動	主 持 人
8:00~8:45	報 到	
8:45~9:40	開幕致詞	理事長黃文哲博 士
	專題演講暨病例92	許永祥主任
9:40~10:30	會 員 大 會	
	一、主席致詞	理事長黃文哲博 士
	二、來賓致詞	
	三、會務報告及提案審查	
	1. 八十六年度資產負債表	常務監事龐飛博士
	2. 八十六年度現金出納表	常務監事龐飛博士
	3.八十六年度資產明細表	常務監事龐飛博士
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	5.八十七年度收支預算表	常務監事龐飛博士
	6.八十七年度工作計畫	秘書長劉振軒博士
	六、 討論提案	理事長黃文哲 博士
	七、 臨時動議	理事長黃文哲

博士

八、禮成

中華民國比較病理學會八十六年度第一次理監事會議 會議記錄

時間：86年3月2日 12:00-13:30

地點：台中榮民總醫院

主席：黃文哲

出席人員：理事-黃文哲、施洽雯、周冠、陳三多、朱瑞民、洪信雄、陳安、鄭益謙
監事-林永和、李進成、龐飛、羅登源

請假人員：理事-何逸僊、祝志平、蔡信雄、方中民、陳東榮、梁善居
理事-呂福江、王金和

列席人員：劉錫光、許永祥

記錄：劉振軒

一、主席報告(略)

二、會務報告(略)

三、提案討論

- 1.審核新入會會員鄭建助等贊助會員一人、學生會員十一人、一般會員八人，共計二十人，審核通過。
- 2.討論學會功能小組分組名單(附件一)。
- 3.學會雜誌出版事宜，建議 Editorial Board Member
人選：周冠、許永祥、陳安、何逸僊、朱瑞民、龐飛、陳三多。
建議Chief Editor人選：劉錫光、侯書文、江宏、蘇益仁。
- 4.討論八十六年第九次比較病理學研討會主題、時間、地點與負責理事。
主題：肝臟病理
時間：86年7月20日
地點：新光吳火獅紀念醫院
負責理事：陳東榮
- 5.討論八十六年第十次比較病理學研討會主題、時間、地點與負責理事。
主題：泌尿系統疾病
時間：86年10月
地點：三軍總醫院
負責理事：陳安
- 6.中華民國病理學會於每年五月及十一月舉辦學術活動，其他學會活動大部分於十一月。為避免與本會活動撞期，可向國科會科資中心查詢。

四、臨時動議

五、散會

附件一：

中華民國比較病理學會功能小組

出 版 組	學術發展暨教育組	財 務 組	會 員 組
陳三多 梁善居 方中民 祝志平 何逸僊 鄭益謙 朱瑞民 施洽雯 呂福江 周 冠 陳 安	陳三多 陳東榮 方中民 祝志平 何逸僊 蔡信雄 洪信雄 朱瑞民 呂福江 陳 安	蔡信雄 洪信雄	梁善居 鄭益謙 施洽雯 周 冠

中華民國比較病理學會八十六年度第二次理監事會議 會議記錄

時間：86年7月20日

地點：新光吳火獅紀念醫院

主席：黃文哲

出席人員：理事-黃文哲、陳東榮、祝志平、陳三多、陳安、鄭益謙、呂福江
監事-林永和、李進成、羅登源

請假人員：理事-何逸僊、洪信雄、蔡信雄、方中民、朱瑞民、梁善居、施洽雯、周冠
理事-龐飛、王金和

列席人員：江宏、許永祥

記錄：劉振軒、吳佳玲

一、主席報告(略)

二、審核新加入會員資料：

計有蔡耿宇、程景章、余忠泰、周玉瑜、林柏翠、楊佳麟、黃忠志等人審核通過。

三、討論題案與決議

1. 為使本會充份發揮學術文流及服務本會會員，請秘書處將歷次繼續教育積分資料建電腦檔並加以統計。
2. 為使每次研討會之病例切片能發揮最大功用及不浪廢資源，請秘書處調查目前切片發送單位未來積極參與本會活動之意願。
3. 學生會員如畢業後更改為一般會員，於新年度起開始繳交一般會員年費，入會費則不需補交。
4. 年度會費利用年度大會或研討會時催繳，不另行發函通知。
5. 第十次比較病理學研討會預定於11月2日假三軍總醫院舉行，請陳安理事協助接洽場地，主題為泌尿系統疾病。
6. 為鼓勵外界多提供病例及提升病歷報告水準，建議編輯學會會刊申請ISSN，並由Editorial Board審核。Chief Editor：劉錫光博士，Associate Editor：陳三多教授，另一人由理事長接洽中。

四、臨時動議

1. 建議本會設榮譽會員。
2. 第十一次比較病理學研討會預定主題為乳房疾病，地點在花蓮慈濟醫院。
第十二次比較病理學研討會預定主題為神經系統。

五、散會

中華民國比較病理學會八十六年度第三次理監事會議 會議記錄

時間：86年11月2日 12:00-13:30

地點：三軍總醫院

主席：黃文哲

出席人員：理事-黃文哲、祝志平、陳三多、陳東榮、施洽雯、陳安、呂福江、鄭益謙
監事-龐飛、王金和、李進成、林永和

請假人員：理事-何逸僊、洪信雄、方中民、朱瑞民、梁善居、周冠
理事-羅登源

列席人員：劉振軒

記錄：吳佳玲

一、主席報告(略)

二、報告事項(略)

三、討論題案與決議

- 1.目前共有438位會員。審核新進會員向明育等共21名，資格符合通過。
- 2.理監事證書，比照其他學會辦理頒發。
- 3.學會雜誌，每年至少出刊2期。彩色圖片印刷則由作者自行付費，每期印刷費用預算100,000元。請秘書處搜集國科會補助優良學術刊物辦法。學會會刊工作分配如下：
 - (1)Chief Editor:劉錫光教授、黃文哲教授。
 - (2)Associate Editor:陳三多教授、李偉華教授。
 - (3)Editorial Board:由理事長、劉錫光教授、李偉華教授、陳三多教授根據過去會議記錄決定。
 - (4)執行秘書:劉振軒博士。
- 4.同意李偉華教授、陳三多教授擔任associate editor 分別聘用兩位助理，月支車馬費600元。
- 5.第十一次比較病理學會訂於87年2月中旬，以乳房病理為專題，地點：花蓮慈濟醫院，由許永祥醫師主持並由秘書處協助辦理。第十二次比較病理學會時間訂於87年7月中旬，以神經系統病理為專題，地點：苗栗縣竹南鎮台灣養豬科學研究所，由劉振軒博士策劃，請李進成醫師協助。

四、臨時動議

五、散會

中 華 民 國 比 較 病 理 學 會
第 一 屆 中 理 監 事 之 理 監 事 聯 席 會 出 勤 表

姓 名	日期/次別 職 稱	850204 1	850609 2	851110 3	860302 4	860720 5	861102 6	7
黃 文 哲	理 事 長	○	○	○	○	○	○	
何 逸 僊	常務理事	○	○	○	◎	◎	◎	
祝 志 平	常務理事	○	◎	○	◎	○	○	
陳 三 多	常務理事	○	○	○	○	○	○	
洪 信 雄	常務理事	○	○	○	○	◎	◎	
蔡 信 雄	理 事	◎	◎	○	◎	◎	◎	
方 中 民	理 事	○	◎	◎	◎	◎	◎	
朱 瑞 民	理 事	◎	○	◎	○	◎	◎	
陳 安	理 事	○	○	○	○	○	○	
陳 東 榮	理 事	○	○	◎	◎	○	○	
鄭 益 謙	理 事	◎	○	○	○	○	○	
梁 善 居	理 事	○	◎	◎	◎	◎	◎	
施 洽 雯	理 事	○	○	◎	◎	◎	◎	
周 冠	理 事	◎	○	◎	○	◎	◎	
呂 福 江	理 事	◎	○	◎	◎	○	○	
龐 飛	常務監事	○	○	○	○	◎	○	
王 金 和	監 事	○	◎	◎	◎	◎	○	
林 永 和	監 事	○	○	○	○	○	○	
李 進 成	監 事	◎	○	○	○	○	○	
羅 登 源	監 事	○	○	◎	○	○	◎	
劉 振 軒	秘 書 長	○	○	○	○	○	○	

○：出席 ※：缺席 ◎：請假 △：遞補 ☆：公差

中華民國比較病理學會八十七年度會員大會暨 第十一次比較病理學研討會議程表 (乳房專題)

時間：中華民國八十七年三月一日（星期日）08:00~16:00

地點：佛教慈濟綜合醫院（花蓮市中央路3段707號） 電話：（038）561825-2114

主辦單位：佛教慈濟綜合醫院
行政院農業委員會
台灣省政府農林廳
中華民國比較病理學會

時 間	議 程
08:00- 08:45	報到
08:45- 09:00	開幕致詞
09:00- 09:40	Section 【1】 Moderator: 許永祥 主任 專題演講 (Overview of Foot and Mouth Disease)暨 Case 92 屏東縣家畜疾病防治所 徐榮彬 獸醫師
09:40- 10:30	會員大會
10:30- 11:00	週年慶茶會
11:00- 11:15	Section 【2】 Moderator: 龐飛 教授 Case 93 國立台灣大學獸醫學系 鄭謙仁 副教授
11:15- 11:30	Case 94 羅東聖母醫院 祝志平 主任
11:30- 11:45	Case 95 國立中興大學獸醫學系 劉婉雯 獸醫師
11:45-13:30	Luncheon (中華民國比較病理學會理監事聯席會議)
13:30- 13:45	Section 【3】 Moderator: 祝志平 主任 Case 96 彰化基督教醫院 楊曉芳 醫師
13:45- 14:00	Case 97 台灣養豬科學研究所 邱慧英 獸醫師
14:00- 14:15	Case 98 省立豐原醫院 賈永芳 醫師
14:15- 14:30	Case 99 國家實驗動物繁殖及研究中心 梁鍾鼎 獸醫師
14:30- 15:00	Coffee Break
15:00- 15:15	Section 【4】 Moderator: 陳三多 教授 Case 100 中國醫藥學院 賴銘淙 醫師
15:15- 15:30	Case 101 屏東科技大學獸醫學系 張聰洲 講師
15:30- 15:45	Case 102 佛教慈濟綜合醫院 郭明勳 醫師
15:45- 16:00	討論

Comparative pathology Case 92

Contributors: Jung-Ping Hsu (徐榮彬)^{1,2}, DVM, MS; Shiuh-Tyan Huang (黃旭田)¹, DVM, MS; Hsin-Hsiung Hung (洪信雄)¹, DVM, MS; Maw-Sheng Chien (簡茂盛)², DVM, PhD.

1. Livestock Diseases Diagnostic Laboratories of Pingtung Prefecture
2. Department of Veterinary Medicine, National Chung-Hsing University

Clinical history: In April 1997, an outbreak disease was observed in a farm raising 1000 pigs in Pingtung prefecture. The infected sows showed depression, pyrexia, anorexia, and the formation of vesicles, erosion, ulceration, or crusts on the snouts, lips, coronary bands, udders, and teats. The mortality of sows was below 10%, but the mortality of nursing piglets was about 100%. The affected piglets had similar skin lesions to those seen in the sows.

Diagnosis: Foot-and-mouth disease (FMD)

Gross findings: Lameness of pigs is often the first sign. Foot lesions can be so severe as to prevent the pigs from standing. There were vesicles on the sows' snouts, lips, coronary bands, teats, and udders. Suckling and nursery pigs had similar vesicles, erosion, ulceration gross lesions on the snouts, lips, coronary bands, and tongues. Denuded areas between the claws were usually infected with bacteria and resulted in suppuration, and in some cases loss of the claws and prolonged lameness. The most striking gross lesion was found in hearts in piglets; most piglets had multiple, irregular-sized, pale-grayish stripes in the myocardium, which gave the heart a mottled appearance (tiger heart).

Histopathological findings: The development of microscopic lesions was similar in all locations. The stratum spinosum cells in the deeper layers of the epidermis were most severely affected. They were swollen and become eosinophilic with vacuolar degeneration. The intercellular bridges had broken down allowing fluid and debris to accumulate and form microvesicles. Microvesicles filled with edema fluid and cell debris were infiltrated with leucocytes and coalesced to form macrovesicles. Hyaline

degeneration of myocardial fibers and necrosis were accompanied by infiltration of large amounts of lymphocytes and some neutrophils in the myocardium of infected piglets.

Discussion: Foot-and-mouth disease is caused by picornavirus. Five genera are included in the family *Picornaviridae*, each of which contains viruses causing diseases in domestic or laboratory animals. The picornavirus virion is a nonenveloped icosahedron, 25-30 nm in diameter and is an RNA virus. The capsid is composed of 60 copies of each of four coat proteins (VP1, VP2, VP3, VP4). Seven serotypes of foot-and-mouth disease virus have been identified by cross-protection and serologic tests; they are designated O, A, C, SAT1, SAT2, SAT3, and Asia1 (Table 1).

Vesicular diseases of swine can be caused by infection with foot-and-mouth disease (FMD), swine vesicular disease (SVD), vesicular stomatitis (VS), and vesicular exanthema of swine (VES). Rapid diagnosis of foot-and-mouth disease is of paramount importance, especially in countries (Taiwan, Japan, etc.) that are usually free of infection, so that eradication can proceed as quickly as possible. FMD and three other vesicular diseases can produce clinically indistinguishable lesions in domestic animals. Confirmation by laboratory diagnosis is essential, although the history of the disease and the involvement of different species can be valuable pointers to the diagnosis (Table 2). Diagnosis of FMD can be also done by RT-PCR tests from vesicular fluid or epithelia from the clinically suspected pigs.

FMD is a highly contagious disease of cloven-hoofed animals including cattle, swine, sheep, goats, and wild ruminants. The major route of transmission for FMDV is by aerosol, but ingestion of infected food, contact with contaminated clothing, instruments, etc., can all produce infection. Particles of FMDV adhere to the upper and lower respiratory tract. Tremendous aerosols are generated by swine, which are often referred to as an "amplifier host." The main means of dissemination of virus from the initial site of entry is likely to be macrophages of either the pharyngeal lymph nodes or alveoli, following seeding of secondary targets (epithelium, mucosa, and myocardium). The virus replicates, and high levels of viremia generally persist for 3-5 days. Within 2-3 days, vesicles develop where stress and mechanical abrasions occur, which in swine includes the snout, feet, and mouth. In addition, FMDV replication is well documented in the epithelial cells of the mammary gland. High mortality rates of FMD may occur in piglets due to severe myocardial necrosis, which results in a grossly mottled appearance, or "tiger heart."

Outbreaks of FMD had been reported twice in Taiwan in 1913 and 1929; both

outbreaks were eradicated. Pingtung county is an area heavily populated with various kinds of domestic animals including pigs, cattle, goats, and deer. Except in pigs, there has been no evidence of FMDV infection in other animal species during the outbreak from the middle of March till now. It is expected that the eradication of FMD from Taiwan will be achieved in the near future.

Table 1. Geographic distribution of foot-and-mouth disease

Region	Types
South America	O, A, C
Europe	O, A, C
Africa	O, A, C, SAT1, 2, 3
Asia	O, A, C, Asia1

Table 2. Differential diagnosis of vesicular diseases based on naturally-occurring disease in different domestic animal species

Disease	Viral family	Species			
		Cattle	Sheep	Swine	Horse
Foot-and-mouth disease (FMD)	Picornaviridae	S	S	S	R
Swine vesicular disease (SVD)	Picornaviridae	R	R	S	R
Vesicular stomatitis (VS)	Rhabdoviridae	S	S	S	S
Vesicular exanthema of swine (VES)	Caliciviridae	R	R	S	R

S: susceptible by natural exposure. R: resistant by natural exposure.

Diagnostic criteria:

1. Gross signs of vesicles on the snouts, lips, coronary bands, teats and udders.
2. Piglets had multiple, irregular-sized, pale-grayish stripes in the myocardium (tiger heart) seen at necropsy.
3. Viral isolation.
4. By RT-PCR tests from vesicular fluid or epithelia of pigs.

References:

1. Chang, T. C., C. C. Chang, S. S. Tsai, G. N. Chang, M. Kuo, and W. B. Chung. An outbreak of foot-and-mouth disease in pigs in southern Taiwan. J Chin Vet Sci 23: 269-273, 1997.

2. Fenner, F. J., E. P. J. Gibbs, F. A. Murphy, R. Rott, M. J. Studdert, and D. O. White. Picornaviridae. In: Veterinary virology. Academic Press Ltd. 403-423, 1993.
3. Gibson, C. F. and A. I. Donaldson. Exposure of sheep to natural aerosols of foot-and-mouth disease virus. Res Vet Sci 41:45-49, 1986.
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Comparative Pathology Case 93

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Clinical history: A 13-year-old female mongrel dog was submitted to NTUAH due to the rapid enlargement of the Rt 4th-5th mammary glands. The results of routine hematology and blood chemistry examination were within normal limits except a marginally elevated level of BUN. Mastectomy of the Rt 3rd-5th mammary glands was performed.

Diagnosis: Mammary gland adenocarcinoma, complex type, with chondromucinous differentiation.

Gross findings: The tumor was of an ill-defined shape and occupied the 4th and 5th mammary glands with a size of 12×2×2 cm. On cutting, the mass displayed lobulated meaty structure with occasional invasions in the adjacent soft tissue.

Histopathological findings: The tumor was encapsulated but not well demarcated. The neoplastic cells were in cord-like, acinar, or irregular tubular arrangement with round to ovoid shape. The nucleus was hyperchromatic and round to ovoid in appearance with one to two nucleoli. In most parts of the tumor, the neoplastic cells were quite uniform in size; however, a moderate size variation could still be seen in some regions. The mitotic activity was low. There was abundant chondromucinous ground substance intermingled with islets of tumor cells. Necrosis and hemorrhage were frequently present in the central part of the tumor. Evidence of invasion of this tumor was revealed by the presence of tumor emboli in the lymphatics and veins and infiltration of neoplastic cells in the regional superficial mammary lymph node.

Discussion: Mammary gland tumors are very prevalent in dogs and cats, but are rare in other domestic animals. Many features of canine mammary neoplasm are similar to those of women, including age of onset, morphological appearance, frequent metastasis, the

presence of estrogen receptors on the tumor cells, and the general course of neoplastic disease. Thus, they may offer a relevant model for human breast cancer.

The classification of canine mammary gland carcinomas is far from satisfactory due to too many criteria having been proposed. In our case, the tumor was characterized by major infiltrating of neoplastic cells and varied degree of tissue pattern as well as abundant chondromucinous ground substance. The later changes may have come from the proliferation and metaplasia of myoepithelial cells and not the neoplastic cell itself. This may fit the classification of adenocarcinoma (complex type) proposed by Hampe and Misdorp (1974) although some areas resembled the change in malignant mixed tumor (carcinosarcoma).

The presence of invasion by the tumor into surrounding tissue is the single most important predictor of the behavioral malignancy in canine mammary neoplasm. In dogs, 80% of animals with invasive tumors will die due to tumor metastases within 2 years, and most of these will be within the first postoperative year. Metastases to lung (80%) and lymph nodes (65%) are frequently found at necropsy. The highly infiltrating character of the present tumor is unusual. In the latest follow-up by telephone (three months after surgery), aside from a little bit of depression and a small appetite reduction, the dog otherwise is normal.

Diagnosis criteria:

1. lobular pattern of acinar or irregular tubular neoplastic cell arrangement with evident chondromucinous change.
2. tumor emboli in the lymphatics and veins, and infiltration of neoplastic cells in the regional lymph node.

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Comparative Pathology case 94

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Clinical history: A 79-year-old male, suffering from left breast mass for more than 2 years, received OPD examination. The clinical impression was malignancy. Frozen section was arranged with a diagnosis of mucinous carcinoma. Subsequent modified radical mastectomy on Oct. 29, 1997 was performed. The specimen of breast contained a tumor mass, measuring 4.4×3.0×2.6cm, below the retracted nipple. On sectioning, the tumor was greyish, fixed, and firm with areas of hemorrhage and chalky streaks. Totally 9 lymph nodes, measuring up to 1.8×1.0×0.6cm in size, were dissected, and some of them were involved grossly in metastatic cancer.

Diagnosis:

1. Breast, left, modified radical mastectomy, showing papillary carcinoma, invasive.
2. Nipple, left, modified radical mastectomy, papillary carcinoma, invasive.
3. Lymph node, axillary, left, lymphadenectomy, papillary carcinoma, metastatic.

Histopathological findings: Sections from the breast mass showed invasive papillary carcinoma involving the nipple and underlying mammary tissues. The tumor was characterized by predominant papillary frond formation, presence of intraductal components, calcospherites, and small foci of mucin pools. Desmoplastic reaction and hemosiderin deposition were also noted. Perineural invasion could be easily found. Six out of nine axillary lymph nodes showed metastatic papillary carcinoma.

Immunohistochemistry results:

1. ER (estrogen receptor): ++
2. HER-2/neu: ++
3. PR (progesterone reception): —
4. P53: (—)
5. PSA, mucin: negative

Differential diagnosis:

1. Gynecomastia— Gynecomastia may show atypical patterns of hyperplasia and occasional mitoses. It is very rare to find male breast cancer arising out of gynecomastic ducts.
2. Intraductal papilloma (clinically and macroscopically) — Although variable, the arborescent fronds of papillary carcinoma are usually composed of a more delicate fibrovascular core; indeed, the fibrous component may be inconspicuous. The lining epithelial cells are of a single type (CEA was detected in 85% in Papotti et al's study), showing high nucleocytoplasmic ratios, increased mitotic activity, and uniform, hyperchromatic nuclei. The tumor cells may show stratification and resemble the adenomatous epithelium of a tubular adenoma. When solid areas of epithelial proliferation are present in adjacent ducts, they often show features of ductal carcinoma in situ rather than duct hyperplasia, which is associated with intraductal papilloma. The presence of invasive duct carcinoma makes benign papilloma less likely. Benign papillomas have a basal layer of actinrich myoepithelial cells (antibodies to muscle actin, HHF-35, are reliable markers), but the presence of a few myoepithelial cells alone does not exclude a malignant diagnosis in the less characteristic papillary lesions such as micropapillary duct carcinoma in situ and peripheral papillomas with cancerization.
3. Borderline lesion or in situ carcinoma— A micropapillary pattern of carcinoma in situ is relatively uncommon and the presence of sharply defined luminal borders should suggest malignancy.
4. Leukemia and malignant lymphoma— The breast is a known site for relapse of leukemia and lymphoma.
5. Metastatic carcinoma— including malignant melanoma, carcinoma of lung, kidney, and prostate. The cribriform and glandular patterns of most prostate cancers mimic a breast primarily. In addition, estrogen therapy may induce striking alterations in the histology of prostate carcinoma. Besides, there is a predilection of prostate cancer to metastasize to the breast.

Discussion: Breast carcinoma in men is a rarity, 100 times less common in men than in women (less than 1% of all breast cancers). No more than 0.2% of male tumors arise in this organ (Carlson 1980) and cause less than 0.1% male cancer death.

Although breast carcinoma has been diagnosed in males at virtually all ages, including children and young adults (e.g., 12-year-olds), it is usually seen in men over the age of

50 with a median of 66 years (5 to 10 years older than women). The incidence and age-specific death rate increase in a linear fashion with advancing age.

About 75% of patients present with a painless mass. In the remainder, the lesion is detected because of nipple ulceration, retraction, or discharge.

The majority of male breast cancers are located centrally in a retroareolar position, but eccentric lesions, particularly in the upper outer quadrant, have been reported. Synchronous, clinically evident bilateral carcinoma is exceedingly unusual (the cumulative risk for bilaterality is 3 percent or less). A surprisingly high proportion of men with breast cancer have a second primary tumor elsewhere (Yap et al. 1979).

Male breast carcinomas average 2.5cm in diameter and bear striking similarity to those in the female. All the typical subareolar male breast cancers arise from the major ducts. Most as an irregular, scirrhous, grey to tan mass. Some show focal hemorrhage and yellow streaks correlating with necrosis. Mammograms of man with breast cancer typically reveal distinct lesions with well-defined invasive margins that contrast sharply with the surrounding fatty tissue; microcalcification is found in about 3 percent. Larger tumors tend to invade the skin & pectoral muscle. The chest wall is usually involved early. Local & distant dissemination follows the usual pattern.

Despite its low prevalence in males, the histology of mammary carcinoma is strikingly similar in both sexes. The notable exception is the rarity of lobular carcinoma in the male breast.

Histologically, at least 75% of male breast cancers are infiltrating carcinoma of no special type. The combination of tumors of no special type with other histological type is common (especially true with tubular carcinoma). Papillary carcinoma, often with a prominent cystic component, are relatively more common among men (3 to 5 percent of male carcinoma) than women (1 to 2 percent). Pure mucinous carcinoma (as presenting in the lesion for frozen section) are rare.

In the literature, there is a general impression that breast cancer in males has a worse prognosis than in females. The worse prognosis is partially explained by the subareolar location of tumor (carries a greater incidence of metastasis to internal mammary nodes) and the aggressive behavior of tumor under 3cm in diameter in males (a reflection of the scant substance of the male breast, thereby allowing easier spread to the lymphatics, skin, and pectoral fascia. Histological grade and pathological stage also influence the prognosis of male breast cancer.

The preferred treatment is simple mastectomy because the breast is smaller. There is a considerable risk of local recurrence when the axillary nodes are involved. In such

patients, radiotherapy should be considered. Orchiectomy is the treatment of choice for distant metastases. (Responses have been reported in two-thirds of patients, a reflection of high incidence of hormone receptor positive in males). About 85 percent of lesions have positive levels of estradiol receptor and many have substantial levels of receptors for progesterone, dihydro-testosterone, androgen, and glucocorticoid (Pacheco 1986).

Risk factors for the development of male breast cancer include

1. Levels of estradiol, estrogenic hormones: e.g., a relative high frequency of antecedent mump orchitis among men with breast carcinoma (testicular atrophy after orchitis causing relative hyperestrogenism was reported). Long-term administration of exogenous estrogens could contribute to the development of male breast cancer.
2. Radiation exposure (atomic bomb, repeat fluoroscopy or radiotherapy to intrathoracic disease): Casagrande et al. found a trend to more frequent breast cancer in men who had the greatest thoracic radiation exposure with fluoroscopy or for therapy.
3. Klinefelter syndrome: Patients with Klinefelter syndrome (xxy chromosome, with a high estradiol-testosterone ratio or raised gonadotropin level) have close to 20 times the risk of developing breast cancer than do normal males. The reported incidence of breast carcinoma among patients with Klinefelter syndrome varies from 1 to 3 percent.
4. Gynecomastia: gynecomastia or hyperestrogenism following liver damage (cirrhosis, etc.) have been implicated. Observations linking gynecomastia to the pathogenesis of male breast carcinoma include epithelial atypia in gynecomastia, the association of gynecomastia and carcinoma with Klinefelter syndrome, and the finding of microscopic gynecomastia associated with 5 to 40 percent of carcinomas. Still, evidence suggests that gynecomastia is rarely a precancerous condition or an intermediate step in the development of carcinoma. (Histologic transitions from epithelial hyperplasia in gynecomastia to intraductal carcinoma have rarely been described).

Diagnostic criteria:

1. Papillary formations within stroma are the defining feature. Most cases present fibrovascular cores within those formations, with epithelial atypia of several types and with stroma invasion.
2. Many examples have the microscopic appearance of papillary ovarian carcinomas or mammary mucinous tumors. Papillary clusters float in mucin. The separation from mucinous carcinomas is arbitrary. Mucin was present in about two-thirds of tumors.
3. Nuclei are of intermediate or anaplastic grade; cytoplasmic characteristics are greatly

aried and include a well-developed apocrine appearance and apocrine-like apical protrusions. Many cases have adjacent in situ ductal carcinoma, usually of papillary or cribriform type.

4. Immunohistological studies exclude the diagnosis of metastatic tumor (such as PSA for prostate cancer, common leukocyte antigen for lymphoma, mucin stain to demonstrate no myoepithelium in tumor cells, etc.).

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Comparative Pathology Case 95

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Clinical history: A 3-year-old female Akita was diagnosed as suffering from transmissible venereal tumor, and had been treated with Vincristine by a local veterinarian. Although the tumor masses atrophied gradually afterward, she showed anorexia, weakness, and progressive loss of condition. Therefore, she was presented to our hospital for confirming the diagnosis and receiving further treatment.

Diagnosis: Transmissible venereal tumor.

Gross findings: On physical examination, there was a venereal tumor with several masses appearing on the body surface. The extragenital mass was hard, flat, friable, orange-red, and cauliflower-like, with a rough irregular surface. It was clearly visible when the tail lifted, measuring about 5×2×1.3cm³ swollen, with a 4 cm split. The surface was covered with a little straw-colored mucopurulent exudate. Some masses were found in the flank, neck, mammary glands, and inguinal lymph nodes. Nine firm and creamy-white masses, ranging from 0.5 to 1.5 cm in diameter were excised.

Histopathological findings: TVT cells were in compact masses or sheets, and grew in rows, cords, or loose in delicate stroma. The stroma were variable but usually minimal. The cells were uniform except for occasional bizarre cell formations, and were mainly round, ovoid, or polyhedral in outline. They had large, round, hyperchromatic nuclei, distinctly marginal chromatin, and large central nucleoli. They had a moderate amount of faintly eosinophilic cytoplasm, and the outline of the individual cell was indistinct. Many mitotic figures were found. Lymphocytes, plasma cells and macrophages were scattered irregularly throughout the tumor.

Electron microscopic findings: TVT cells contained abundant cytoplasm and a round nucleus. Many organelles, such as mitochondria, lysosomes, rough endoplasmic reticulum, ribosomes, or lipid droplets, were observed. In some degenerated cells, the rough endoplasmic reticulum was dilated or vacuolated.

Discussion: The transmission of TVT occurs by transplanting intact viable tumor cells from infected animals to tracheated mucosa during coitus, or licking. In female, the predilection site is vagina. Other genital organs and extragenital sites, including skin, lips, buccal mucosa, lymph nodes, eyes, nose, testicles, brain, muscle, thoracic and abdominal viscera could be involved too. However, very few cases have been founded in the breast. Because the cytologic appearance of TVT cells is similar to that of lymphoma, histiocytoma, or mast cell tumor, it is easy to confuse them. Hence, we tried to characterize TVT by histopathological appearance and ultrastructure. Histopathologically, TVT cells were compacted or grew in cords with delicate stroma. Ultrastructurally, they had large, round nuclei and large central nucleoli that contained the bulk of euchromatin. The nucleus/cytoplasm ratio was about 1:1, and the cytoplasm was abundant with many free ribosomes, rough endoplasmic reticulum (rER), mitochondria, Golgi apparatus, and some lysosome. The cytoplasmic membrane formed numerous microvilli interdigitating between adjacent cells.

Diagnostic criteria:

1. Cauliflower-like extragenital mass was clearly visible.
2. Tumor cell contained a large, round, hyperchromatic nucleus and various amounts of cytoplasm.
3. TVT cells contained abundant cytoplasm, and many organelles were observed.

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Comparative Pathology Case 96

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Clinical history: The 67-year-old female came to medical attention for her right breast mass which was noted by herself recently. She denied any systemic disease. Physical examination demonstrated an ill-defined hard mass measuring 2×2 cm in size in the right breast. Mammography displayed focal asymmetry of the breast tissue in the right upper outer quadrant with no definite mass or suspicious microcalcification. Mammary ultrasonography showed a hypoechoic, heterogenous, and lobulated tumor with slight posterior enhancement in the right breast. Excisional biopsy specimen was taken and sent for pathologic examination.

Diagnosis: Malignant lymphoma, large cell type, diffuse, B-cell phenotype

Gross findings: The specimen consisted of two breast tissue fragments measuring 2×1×0.8 cm and 3×2.8×1 cm in size. Grossly, they were yellowish and soft. On cut, the former showed a well-demarcated, flesh-colored and elastic nodule measuring 1.8 cm in greatest dimension. The latter revealed an ill-defined, whitish, and elastic nodule measuring 1.5 cm in greatest dimension.

Histopathological findings: Sections showed multinodular aggregations of large, cleaved lymphoid cells which had infiltrated the fibrous stroma and adipose tissue at the periphery. A reactive lymphocytic infiltrate composed of small lymphocytes was present in the surrounding tissue. A few ductal structures were observed at the periphery and lymphoepithelial lesion was present. Immunohistochemical study showed a positive reaction with leukocyte common antigen and L-26, and a negative reaction with cytokeratin and UCHL-1.

Discussion: Malignant lymphoma rarely involve the breast, manifesting either as a primary lesion or as part of a generalized process. The frequency of primary breast lymphoma is variable, ranging from 0.12 percent to 0.53 percent of all malignant breast

tumors, less than 0.5 percent of all malignant lymphomas, and about 2 percent of extranodal lymphomas.

Primary lymphoma may originate from migratory lymphocytes, parenchymal lymphocytes in the resting breast, or arise within an intramammary lymph node. It occurs in patients of all ages, ranging from 9 to 88 years, with a median of 50 years. Unilateral lymphoma affects the right breast significantly more often with a ratio of approximately 3 to 2. The bilaterality rate is about 10 percent of patients at the time of diagnosis.

The presenting symptom is a palpable, painless mass of the breast. Patients may complain of pain and suffer from night sweats, fever, and body weight loss. The tumor is often solitary, but patients with multiple lesions and diffuse infiltration have been described. Axillary lymph node involvement is seen in 30 to 50 percent of patients. Mammographically, lymphoma presents as a relatively circumscribed mass, focal or diffuse densities, or discrete nodules with irregular margins and no evidence of calcification or retraction.

Grossly, the tumors seen in primary breast lymphoma are well-defined, fleshy, tan to gray, and may have a nodular configuration, with an average size of 2 to 3 cm. Microscopically, primary lymphoma of the breast is almost always of non-Hodgkin's type and usually has a diffuse pattern of growth. In adult patients, the most common lymphoma is the large cell type, followed by small lymphocytic and follicular types; nearly all of these tumors are of B-cell nature. A high incidence of intermediate- and high-grade lymphomas are present. Bilateral Burkitt-type lymphomas have been seen in young women during pregnancy. Morphologic distinction between primary lymphoma and secondary involvement is impossible. Immunohistochemical studies show that nearly all of these cases lack evidence of marginal or mantle cell differentiation. It is believed that the majority of mammary lymphomas do not arise from mucosa-associated lymphoid tissue (MALT). On uncommon occasions, typical features of MALToma (including prominent lymphoepithelial lesion, an admixture of small lymphocytes, Isaacson's centrocyte-like cells, and lymphoplasmacytoid cells) are significant.

The prognosis of primary lymphoma of the breast depends on the stage of the disease and the histologic type of the tumor. Tumor size and bilaterality do not appear to have a significant effect on prognosis. Patients with stage I disease and those with histologically low-grade lesions have the most favorable prognosis. It has now been demonstrated that excellent focal control in the breast and regional lymph nodes can be achieved with radiation after partial mastectomy. Regardless of the type of local therapy, the majority of recurrences occur at distant sites or in the opposite breast.

Diagnostic criteria:

1. cellular discohesiveness
2. monotonous or pleomorphic population of atypical lymphoid cells
3. positive staining for leukocyte common antigen and negative staining for cytokeratin

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Comparative Pathology Case 97

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Clinical history: A 12-year-old female tiger (*Panthera tigris*) had a tumor mass in the right side of mammary glands and complete unilateral mastectomy was performed. Four months later, the tiger died from generalized metastasis.

Diagnosis: Carcinosarcoma (Malignant mixed tumors), originating from mammary gland and metastasizing to the visceral organs, tiger.

Gross findings: At necropsy, the affected mammary glands were extremely enlarged and ulcerated. The cut surface revealed that the enlargement was mostly caused by parenchymal proliferation of the mammary glands accompanied with central necrosis. White to pale yellow nodular masses of varying numbers and sizes were distributed in the lungs, pleural cavity, epicardial fat, liver, kidney, spleen, heart, adrenal gland, and lymph nodes in the thoracic cavity and inguinal region.

Histopathological findings: The tumor was composed of sheets of malignant myoepithelial cells characterized by increased N/C ratio, prominent nucleoli, and high mitotic figures with scattered foci of hyaline cartilage formation. Lobular acini filled with sheets of neoplastic cells were separated by proliferating fibrous trabeculae. Glandular structure was occasionally present in some areas of the sections. Also, massive necrosis was noted. Tumor emboli were frequently detected in the lymphatics and blood vessels. The neoplastic cells in the metastatic sites were almost replaced by massive growth of immature hyaline cartilage surrounded by undifferentiated chondroblasts.

Immunohistochemical and histochemical staining: Cytokeratin (AE1/AE3), S-100, and Vimentin were variably positive in the cytoplasm of the tumor cells. Masson trichrome stain and Alcian blue stain also confirmed the histological findings.

Laboratory examinations: Estrogen receptor (30.7 fm/ng) and progesteron receptor (40.5 fm/ng) were measured from the pieces of frozen mammary gland tissues.

Discussion: Mammary tumors are more common in women and dogs than in cats; their prevalence in other feline species was not known. In a surveillance study, the incidence of mammary gland tumors in domestic cat was 25.4/100,000 female cats. Among the malignant mammary tumors, adenocarcinomas (80-90%) are the most predominance followed by carcinomas; sarcomas are rarely observed (Table 1). The incidence increases with age, especially in cats above 8 years. Most feline malignant mammary gland tumors tend to grow rapidly and metastasize to the regional lymph nodes and lungs. Metastasis to bone is a rare event. Owing to early metastasis, the prognosis of malignant mammary gland tumors in domestic cats is generally poor. It has been reported that the single most important factor affecting the prognosis of malignant mammary tumors in cats is the tumor size at time of diagnosis. Studies have shown that cats with a tumor > 3 cm diameter had a median survival time of 6 months (Table 2).

The presence of estrogen receptors has been demonstrated in human breast tumors, rodent mammary gland tumors, and canine mammary gland tumors. In the normal cat, while growth of mammary ducts is stimulated by estrogen alone, growth of the lobuloalveolar system also requires progesterone. It is claimed that the presence, or absence, of estrogen receptors in human breast tumors is of value in directing therapy of the patient, but the findings in feline species need further study.

Table 1. Mammary tumors diagnosed in cats at 15 North American veterinary medical teaching hospitals (March 1964 to June 1978)

Cell-type	Number of cases
Malignant tumors	
Carcinoma	
Adenocarcinoma	94
Undifferentiated carcinoma	16
Squamous cell carcinoma	2
Cystadenocarcinoma	1
Sarcoma	
Fibrosarcoma	1
Myxosarcoma	1
Carcinosarcoma	
Malignant mixed tumor	4
Benign tumors	
Adenoma	9
Fibroadenoma	4
Lipoma	2
Cystadenoma	2

Table 2. Correlation of tumor size to survival time in 91 cats with mammary adenocarcinoma

No. of cats	Tumor size (cm ³)	WHO stage*	Median survival time
54	1-8 (2 cm diameter)	I - II (T ₁ to T ₂)	>3 yr
19	9-27 (2-3 cm diameter)	II - III (T ₂)	2 yr
18	≥28 (>3 cm diameter)	III (T ₃)	6 mo

*T₁ - tumor<1cm maximal diameter; T₂- tumor 1-3cm maximal diameter; T₃- tumor>3 cm maximal diameter.

Diagnostic criteria:

1. A malignant mixed tumor composed of cells resembling epithelial components and cells resembling mesenchymal elements.

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Comparative Pathology Case 98

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Clinical history: A 35-year-old woman had a breast tumor and received excisional biopsy.

Diagnosis: Mucinous carcinoma with intraductal carcinoma.

Gross findings: A 4×3×1.8 cm tumor, circumscribed, soft, solid, grayish-white, gelatinous, glistening, slimy, with fibrous capsule.

Histopathological findings: Clumps, acini, or tubules of small, uniform tumor cells, showing scant intracellular mucin, little pleomorphism and low mitotic ratios, floating in mucin. At periphery of mammary parenchyma, dilated ducts filled with monotonous, large, epithelial cells with pallor cytoplasm, high grade nuclei, and increased nuclear to cytoplasmic ratio, in a solid and cribriform growth pattern, forming regular round microlumens throughout the duct, with focal mucin production.

Histochemistry results: The acellular, pale, pink, amorphous material shows positive staining for mucosubstance (PAS, PASD, mucicarmine, alcian blue).

Discussion: As defined in the WHO classification of breast tumors, mucinous carcinoma contains "large amounts of extracellular epithelial mucus, sufficient to be visible grossly, and recognizable microscopically surrounding and within tumor cells." Other terms used to identify this tumor include gelatinous, colloid, mucous, and mucoid carcinoma. When the diagnosis is restricted to tumors consisting of pure or nearly pure mucinous carcinoma, not more than 2 percent of mammary carcinomas fall into this category. A widely accepted standard requires that at least 75 percent of the tumor have a mucinous growth pattern. The term "infiltrating duct carcinoma with focal mucinous differentiation" or "mixed mucinous carcinoma" is preferable for tumors with mixed histologic patterns. Mucinous carcinoma occur throughout the age range of breast carcinoma. The mean age of women with pure mucinous carcinoma is greater than those with nonmucinous carcinoma. The initial symptom is a mass in the majority of patients. Nipple discharge

and pain are uncommon. Fixation to the skin and chest wall occur only with large lesions. Most mucinous carcinoma occur in the upper quadrant. Only a minority of pure mucinous carcinoma have calcification. The tumor usually appears as a lobulated mass lesion on mammography. The average duration of symptoms prior to biopsy and diagnosis tends to be 3 months or less. Pure mucinous carcinoma measures from less than 1 cm to more than 20 cm in diameter. Most of them are smaller than 5 cm. Mucinous carcinoma should be distinguished from the "mucocele-like tumor." Cystic papillomas, papillary carcinoma, ductal hyperplasia of either florid or atypical type, and intraductal carcinoma, can also be accompanied by focal or sometimes abundant mucin secretion, which may accumulate in large extracellular pools. Mucinous carcinomas are variants of invasive duct carcinoma. Intraductal carcinoma is found associated with approximately 75 percent of the lesions, generally at the periphery. Pure mucinous carcinoma are virtually all diploid while only 42 percent of mixed mucinous carcinomas are diploid. About 60 percent of pure mucinous carcinoma are estrogen receptor positive. Argyrophilic granules have been detected in 25 to 50 percent of mucinous carcinoma, suggesting an endocrine differentiation. The granules can contain immunohistochemically detectable serotonin, somatostatin, and gastrin. The tumor cells may also test positive for neuron-specific enolase, synaptophysin, chromogranin, S-100 protein, and carcinoembryonic antigen. The presence of argyrophilic granules is not prognostically significant in mucinous carcinoma. Ultrastructurally, tumor cells in mucinous carcinoma contain abundant cytoplasmic filaments with a perinuclear distribution. Basal lamina and myoepithelial cells are absent. Women with pure mucinous carcinoma have a good relapse-free survival 5 and 10 years after mastectomy. The reported frequency of negative axillary lymph nodes in patient with pure mucinous carcinoma ranged from 71 to 97 percent as compared to 50 percent for mixed mucinous carcinomas. The survival for mucinous carcinoma patient after 10 years progressively declines. Late systemic recurrences have been described after mastectomy, indicating the need for long-term follow-up. Most patients with pure and mixed mucinous carcinoma have been treated by radical mastectomy. Simple mastectomy has been advocated as appropriate for women with pure mucinous carcinoma and clinically negative axillary lymph nodes. The role of radiation therapy and breast conservation for the treatment of mucinous carcinoma remains uncertain.

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Comparative Pathology Case 99

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Clinical history: A 7-month-old female specific-pathogen free (SPF) inbred BALB/cByJ (BR) mouse came from the colony originally introduced from Jackson Laboratory (Bar Harbor, Maine, USA). The mouse was retired by subcutaneous mass around right axillary and adjacent submaxillary areas.

Diagnosis: Mammary gland adenocarcinoma, type B, with pulmonary metastasis, BALB/c ByJ mouse.

Gross findings: The mouse showed subcutaneous mass located on the right ventral neck extending to the forelimb. The mass was soft, yellowish, 1.5 to 2 cm. in diameter. In addition, congested pulmonary parenchyma with small whitish spots, 0.1 to 0.2 cm, were present.

Histopathological findings: The tumor mass was located subcutaneously, encapsulated, well demarcated but invasive, adjacent to the parotid and sublingual salivary glands, with centric liquefactive necrosis.

The neoplastic cells showed variable appearances--tubular, solid cords, nest-like, cribriform, acinar, intracystic papillary projections, sheets with no signs of glandular differentiation, and delicate stroma surroundings. The neoplastic cells were basophilic, uniform, ovoid to cuboidal, with scant eosinophilic cytoplasm and large, oval, central-located nuclei. Finely-stippled chromatin and indistinct nucleoli were also noted. Pulmonary metastasis of the neoplastic cells in the alveoli and peribronchiolar and perivascular spaces were often present.

Other organs were normal without significant lesions.

Table 1. Histological classification of the mammary gland tumor and dysplasia (WHO)

A. Domestic Animals (Veterinary Medicine)	B. Human (Most Common)
Lobular hyperplasia Cyst Adenosis Duct ectasia Fibrosclerosis Epitheliosis	Fibrocystic disease Duct ectasia Sclerosing adenosis
Adenoma Fibroadenoma 1. pericanalicular 2. intracanalicular Mixed, Fibroadenomatous change (cat)	Fibroadenoma 1. pericanalicular 2. intracanalicular Phyllodes tumor
Papilloma	Intraductal Papilloma
Carcinoma A. Adenocarcinoma 1. Tubular (Simple vs Complex*) 2. Papillary (Simple vs Complex) 3. Papillary cystic (Simple vs Complex) B. Solid Carcinoma (Simple vs Complex) C. Spindle cell carcinoma (Simple vs Complex) D. Anaplastic carcinoma E. Squamous cell carcinoma F. Mucinous carcinoma	Carcinoma A. Noninvasive 1 a. Intraductal Carcinoma 1 b. Intraductal Carcinoma with Paget's disease 2. Lobular carcinoma in situ B. Invasive 1 a. Invasive ductal carcinoma-NOS 1 b. Invasive ductal carcinoma with Paget's disease 2. Invasive lobular carcinoma 3. Medullary carcinoma 4. Colloid carcinoma 5. Tubular carcinoma 6. Adenoid cystic carcinoma 7. Apocrine carcinoma 8. Invasive papillary carcinoma
Mixed Tumor (Carcinosarcoma) Malignant	
Sarcoma A. Osteosarcoma B. Fibrosarcoma C. Osteochondrosarcoma	Sarcoma A. Osteosarcoma B. Fibrosarcoma C. Osteochondrosarcoma
Gynecomastia	Gynecomastia

* Complex: with more myoepithelial components.

Table 2. Mammary tumor in high-cancer-strain inbred mice

TUMOR	STRAIN	INCIDENCE (%)	AVERAGE AGE (MOS.)	COMMENTS
Mammary tumor	A	80	12	Breeding female
		30	2	Virgin female
	BDP	Frequent		Female
	BR6	95		Breeding female having two litters or more
		46		Virgin female
	CBA/J	60-65	<12	Breeding female
	CBA/St	40		Breeding female
	C3H	100	8.8	Virgin female
		99	7.2	Breeding female
	C3H-A ^{vy}	100	6-7	All females
	DBA	76-77	12-15	Breeding female
		Lower		Virgin female
	DD	84	7.7	Breeding female
		75	10.2	Virgin female
	FM	90		Breeding female
	GR	High		Breeding female. Shows marked hormone dependence. Virus transmitted by male as well as female
	LTS/A	High		Breeding female
				Virgin female
	PBA	76	9.2	Breeding female
	PS	21		Female
	RIII	88		Breeding female

(From Squire RA, 1978)

Table 3. Histologic classification of rat and mouse mammary gland tumors

A. Rat	B. Mouse
Nonneoplastic lesions Cystic changes 1. Ductal 2. Lobular	Plaques (Pregnancy-Responsive) Hyperplastic alveolar nodules (HAN)
Epithelial neoplasma A. Benign 1. Intraductal papilloma 2. Papillary cystadenoma 3. Adenoma (a) Tubular (b) Lactating 4. Fibroadenoma	Adenoma Carcinoma, Dunn's type C
B. Malignant 1. Noninvasive carcinoma (a) Ductal papillary (b) Ductal cribriform (c) Ductal comedo 2. Invasive carcinoma (a) Papillary (b) Cribriform (c) Tubular	Carcinoma, Dunn's type A- acinar type, small tubular carcinoma Carcinoma Dunn's type B with one or more architectural patterns
3. Comedo-type Anaplastic type	Undifferentiated type Anaplastic type
Stromal neoplasms 1. Fibroma 2. Fibrosarcoma	Fibroma Fibrosarcoma Hemangioendothelioma
Adenoacanthoma Others	Adenoacanthoma 1. Molluscoid type 2. Organoid type 3. Pale cell type

Discussion: Mammary tumors rank second (behind skin tumors) as the most common neoplasms in dogs of both sexes; they are by far the most common tumors in the bitch. The comparative histological classification between domestic animals and human is shown as Table 1. In general, the pituitary gland adenoma (endocrine system) and mammary fibroadenoma are the leading tumors in rats; the systemic lymphoma and pulmonary adenoma/carcinoma are the most common tumors in mice. In a three-year spontaneous tumor survey in NLABRC, we found mammary tumors in mice were chiefly carcinoma (80%). The most common strains were C3H/HeJ and CrI: CD-1 ICR (BR).

However, spontaneous mammary tumors have a variable frequency in different mouse strains depending on (a) differences in genetic susceptibility and (b) the presence or absence of milk-transmitted mouse mammary tumor virus (MMTV). The mouse strains

most used for studies on mammary tumorigenesis are divided into three genetic categories:

(a) Resistant, MMTV-free, usually no or very low mammary tumor incidence: C57BL, O₂₀

(b) Susceptible, free of milk-transmitted MMTV, low mammary tumor incidences:

BALB/c

(c) Susceptible, carrying milk-transmitted MMTV, high mammary tumor incidence: C3H, RIII, A, DBA, GR, DD. Their incidences were 20~100% (Table 2).

There are 5 pairs of mammary glands in the mice. Their topographical nomenclature is as follows: right and left cervical; 1st thoracic; 2nd thoracic; abdominal; inguinal. The mammary tumors may be located on both sides and extend anteriorly from the neck and forelimbs to the proximal region of the hindlimbs. In the present case, the located mammary carcinoma of ventral neck must be differentiated from myoepithelioma of parotid salivary gland origin. This tumor is often diagnosed in the strain A/J, BALB/cJ, with lesser incidences in BALB/ByJ, A/HeJ and NOD/Lt.

The histologic classification of rat and mouse mammary gland tumors are similar (Table 3). However, the mammary carcinoma in mice are divided into Dunn's type A, B, C, Y (tubular, branching), L (lace-like, secretory with budding), and P (pregnancy-responsive, plaque-like).

Dunn's type A adenocarcinoma exhibited well differentiated acinar, cuboidal cell, or tubular pattern; there were the classical MMTV-related tumor, frequently found in female C3H mice. This tumor type was considered by some pathologists to be benign adenoma.

Dunn's type B adenocarcinoma shows no prevalence of a particular structure-tubular, solid, cribriform, acinar, cystic, comedo, hemorrhagic, papillary etc. Metastasis to the lungs with this type may be observed. This is the most common tumor in strains without an MMTV burden, e.g., BALB/c mice.

Type C adenocarcinoma, uncommon, occurring in very old mice, does not have the MMTV burden. This tumor resembles the fibroadenoma of humans, composed of cysts, lined with a single layer of cuboidal cells and an outer layer of myoepithelial cells.

Breast cancer prefers nulliparous women and parous female mice. The histology of human breast cancer is rather different from that of murine tumors. Principally, this is because of the much greater amount of connective tissue in human breast, at least 90% of human breast cancer is considered to be ductal in origin while mouse mammary tumors mostly arise in alveoli or ductules.

Genetic constitution of the mice, MMTVs of different origins, ovarian hormone (estrogen), cellular oncogene (int-2, C-myc, C-erb B2, ras) overexpression and/or amplification etc. play a major role in murine mammary tumor. In Taiwan, breast cancer

ranks as the sixth leading cause of cancer death in human. Genetic factor, consumption of a high fat diet, obesity, delay in first delivery, and non-breast feeding are the major contributing factors in breast cancer.

Diagnostic criteria:

1. Relationship between causative agents (i.e., MMTV, chemical carcinogens, hormones, genetic factors) and the histological categories of murine mammary tumors.
2. High-cancer-strain inbred mice (Table 2).
3. Dunn's types A, B, C, Y, L, P murine mammary adenocarcinoma et.al. (Table 3).
4. Immunohistochemistry study (vimentin, cytokeratin, K5, K6, K14, S-100, α -smooth muscle action etc.) of five cell types: basal, myoepithelial, luminal type I (ductal keratin-positive cells), luminal type II (ductal keratin-negative cells), and alveolar cells.
5. Differentiated from myoepithelioma of salivary gland.

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Comparative Pathology case 100

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Clinical history: A 70-year-old female had suffered from swelling and pain of left breast for 1 week. She had received paraffin injection over the bilateral breast thirty years ago in Tokyo. Hematologic examination revealed RBC, 378×10^3 mm³; Hb, 11.7 gm%; hematocrit, 36.3%; WBC, 8100/c.mm, segmented, 59%; lymphocyte, 40%; monocyte, 1%. CT scan of chest showed hyperdense lesion with heterogenous pattern over both sides of breast, other contrast enhancement soft tissue density at the inner region of left breast, paraffinoma comparable. Under the impression of paraffinoma with infection, a bilateral mastectomy was done. Unfortunately, the frozen section revealed malignancy of left breast. A modified radical mastectomy of left breast was then done. Pulmonary metastasis in right lobe was found one year later. Finally, she died of massive pulmonary metastasis three years later.

Diagnosis: Malignant fibrous histiocytoma and paraffinoma.

Gross findings: The left breast measured 14×10×6 cm in size and contained a large mass (9×5×5cm in size) with ill-demarcated margin. The cut surface was yellowish white with whorled or multi-nodular appearance and indurated consistency. Focal zones of calcification and necrosis were seen. The right breast measured 10×8×4 cm and contained a lump of mass (7×4×4 cm in size). On cut surface, it contained whitish and yellowish spots in the tissue.

Histological findings: The tumor consisted of large spindle-shaped or polygonal cells arranged in sheets or short intersecting fascicles. The cells were typically pleomorphic, showing a polygonal or spindle-shaped appearance with numerous mitotic figures and a whorl (storiform) pattern. Variable numbers of infiltrating lymphocytes and histiocytes were present. Foci of fat necrosis and calcification were intermingled with collections of histiocytes, foamy macrophage, and giant cells, accompanied by extensive areas of dense fibrous tissue and hyalinization, comparable with paraffinoma.

Immunohistochemistry findings: Tumor cells were stained diffusely and were strongly positive for vimentin, negative for S-100 and keratin.

Discussion:

1. Lipogranuloma (paraffinomas) almost always results from the injection of foreign substances such as paraffin, wax, silicone, or oil to enlarge the breast or penis. Microscopic examination reveals typical foreign body type granulomatous inflammation with lipid vacuoles, variable in size and embedded in dense fibrous tissue. Destructive paraffinoma of the breast and thoracic wall caused by paraffin injection for mammary increase are reported. But paraffinoma-induced breast sarcoma is rare in review of the literature. Most cases of postradiation sarcoma have been reported so far.
2. Sarcoma of the breast represents less than 1% of primary mammary malignancies, including: malignant fibrous histiocytoma (44%), liposarcoma (24%), and fibrosarcoma (16%); and clear cell sarcoma, neurogenic sarcoma, leiomyosarcoma, and alveolar soft part sarcoma (4% each).
3. Malignant fibrous histiocytoma (MFH) is composed of fibroblast and histiocyte-like cells typically showing significant pleomorphism, although several morphologic variants occur. Currently it is one of the most common types of soft tissue sarcoma. MFH cells show a fibroblastic or primitive mesenchymal cell differentiation, possibly a complex differentiation. There are four histologically distinct subtypes of MFH: storiform-pleomorphic, giant cell, myxoid, and inflammatory.
4. The differential diagnosis includes: fibromatosis (desmoid), benign fibrous histiocytoma, nodular fasciitis, fibrosarcoma, spindle cell carcinoma, monophasic synovial sarcoma, malignant schwannoma, spindle cell type of rhabdomyosarcoma, liposarcoma, and leiomyosarcoma.
5. MFH has a rather high rate of local recurrence and lung metastasis. This report emphasizes its full malignant metastatic potential. The finding suggests that failure to establish local control is associated with poor prognosis that wide local excision or simple mastectomy does not provide sufficient clearance to be used as first-line treatment. Excision of the axillary lymphatics and adjuvant radiotherapy are unlikely to be beneficial. Five of these (tumor differentiation, cellularity, mitosis count, tumor necrosis, and vascular emboli) were correlated with the advent of metastases and with survival. The overall mortality at 5 years was 64%.

Diagnosis criteria:

1. The tumor was composed of spindle cells of a storiform pattern with fibroblastic, myofibroblastic, histiocytic-like features and osteoclast-like giant cells.
2. Tumor cells stained positive for vimentin and histiocytic markers (a-1 Antitrypsin, a-1 antichymotrypsin, and KP1 (CD68); Negative for both smooth muscle, neural and epithelial markers (actin, S-100 and keratin).

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Comparative Pathology Case 101

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Clinical history: A 1-month-old young piglet was originally from a pig farm in the south of Taiwan. The affected pigs were 2 weeks old suckling piglets to 2 months old weanling pigs with skin lesions over their entire bodies. Morbidity was higher but mortality was lower. The affected pigs were sent to pathology laboratory for further post mortem examination.

Diagnosis: Swine pox

Gross findings: The affected piglets had eruptive lesions over their entire skin, particularly on the ventral abdomen, face, ears, medial sides of the legs, and the back. Skin lesions were characterized as tiny vesicles of about 1.0 to 3.0 cm in diameter; the center of the vesicular lesions was observed to be dark brown, and had shrunken scabs.

Histopathological findings: Extensive ulcerations with polymorphonuclear cells were seen in the skin extending almost to hair follicles with infiltrations of a numerous of neutrophils and a few lymphocytes and eosinophils. Epithelial hydropic degeneration and hyperplasia were seen in some areas of epidermis and hair follicles. There were granular intracytoplasmic eosinophilic inclusion bodies in the vacuolated epithelial cells. These inclusions were minute granules or the size slightly larger than the nucleus. The nuclei of affected cells revealed intranuclear vacuoles.

Discussion: Pox infection in pigs are produced by two viruses of the pox viridae family, vaccinia or cowpox and swinepox virus. Swine pox is generally a benign or mild disease in Taiwan; morbidity is high but mortality is low. The affected pigs are mostly young pigs, particularly suckling pigs of 2 weeks to 3 months of age. Older pigs are usually not fully susceptible, one affected with the lesions are less severe than those in young pigs. Sometimes both morbidity and mortality are higher. Velu (1916) reported the death of

102 young pigs in a herd of 130. Both swine pox virus and vaccinia virus can cause pox lesions in pigs. Histopathological differentiation of vaccinia and swine pox viruses is listed in the table 1.

Table 1. Histopathological differentiation of vaccinia and swine pox

	Vaccinia	Swine pox
a. Intracytoplasmic inclusion body	+	+
b. Intranuclear vacuoles	-	+
c. Multinucleated giant cells	+	-

Although the disease is transmitted by direct contact as described in most reports, congenital pox infection in swine had been described.(Borst 1990)

Swine pox can be diagnosed on the basis of typical pock lesions on the skin or by histopathological examination of the skin which reveals hydropic degeneration vacuolation. Intracytoplasmic eosinophilic inclusion body of prickle cells and by EM.

Diagnostic criteria:

1. Pock lesions on the skin.
2. Ballooning degeneration and intracytoplasmic acidophilic inclusion body of prickle cells.

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Comparative Pathology Case 102

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Clinical history: A 56-year-old woman was presented with a one-year history of a soft nodule in the lateral upper quadrant of her right breast. An excisional biopsy was performed.

Gross findings: The well-defined tumor nodule measured 3.0×2.0×1.5 cm in size. On cutting, it was fibrotic, myxoid, with focal chondroid change.

Histopathological findings: The tumor was composed of islands of epithelium and chondromyxoid stroma. The epithelium formed tubules and solid nests.

Diagnosis: Pleomorphic adenoma (benign mixed tumor)

Discussion: Pleomorphic adenoma (benign mixed tumor) is the most common tumor type of the salivary glands. The tumor is characterized by the presence of two distinctive cell types: ductal epithelial cells and myoepithelial cells. The myoepithelial cells are capable of inducing the formation of myxoid, chondroid, and osseous tissue. Because the breast is a modified sweat gland, the occurrence of pleomorphic adenoma in the breast is not unexpected.

Pleomorphic adenoma of the breast is a rare benign neoplasm that might be misinterpreted both clinically and pathologically as a malignant tumor. In canine animals, pleomorphic adenoma of the breast is a frequent occurrence. The first case of mammary pleomorphic adenoma was reported in 1968 by Nabert and associates. In reviewing the literature, 34 cases of pleomorphic adenoma of the breast have been previously described. The ages of the patients ranged from 19 to 78 years, with all but two patients being female. All patients presented with a palpable breast mass, and three had nipple discharge. The mammograms of most cases were reported as suggestive of carcinoma. Most tumors ranged in size from 0.8 to 4.5 cm in maximum diameter, with an average size of 2 cm. Most lesions (82%) were juxtaareolar. Grossly, the tumors were well-circumscribed,

lobulated, glistening, grayish-white masses with varying degrees of hard, gritty calcification. Histologically, the lesions were solitary circumscribed nodules or, less frequently, multiple closely approximated circumscribed nodules, composed of islands of epithelium and chondromyxoid stroma. The immunohistochemical profile and electromicroscopical studies of the stromal cells were characteristic of cells exhibiting myoepithelial differentiation. Positive immunostaining for S-100 protein, cytokeratin, and muscle-specific actin, as well as the ultrastructural presence of intermediate filaments with dense bodies and intercellular junctions, supported the predominant myoepithelial cell differentiation within the tumor, whereas the epithelial cell component was stained only with cytokeratin and contained formed lumina with surface microvilli.

The so-called pleomorphic adenoma of the breast is an intraductal papilloma with stromal metaplasia, resulting principally from myoepithelial cell proliferation. Evidence for deviation from intraductal papillomas is found in the association with papillomas in most of these tumors, as well as their frequent juxta-areolar location.

The most important differential diagnostic consideration is metaplastic carcinoma. The cases of metaplastic carcinoma manifest at least focal areas of cellular pleomorphism in the metaplastic stroma and usually have recognizable foci of intraductal or invasive carcinoma.

Pleomorphic adenomas are benign. It is important to draw wider attention among pathologists to the occurrence of pleomorphic adenoma in the breast. Then misdiagnosis and the resulting inappropriate treatment can be easily avoided.

Diagnostic criteria:

1. the circumscription and preferential juxta-areolar location
2. the tumor nodules were composed of islands of epithelium and chondromyxoid stroma

References:

1. Balance WA., Ro JY., El-Naggar AK., Grignon DJ., Ayala AG., Romsdahi MG. Pleomorphic adenoma (benign mixed tumor) of the breast. An immuno-histochemical, flow cytometric, and ultrastructural study and review of the literature. Am J Clin Pathol 93(6): 795-801, 1990 Jun.
2. Chen KT. Pleomorphic adenoma of the breast. Am J Clin Pathol 93 (6): 792-4, 1990 Jun.

中華民國比較病理學會第一次至第十一次比較病理學研討會病例一覽表

第一次比較病理學研討會病例（83年10月30日於台灣養豬科學研究所舉行）：

<u>動物別</u>	<u>診斷</u>	<u>提供單位</u>
1.Dog	Myxoma	美國紐約動物醫學中心
2.Ferret	Chordoma	美國紐約動物醫學中心
3.Human	Ependymoblastoma	長庚紀念醫院
4.Goat	Cryptosporidiosis	台灣養豬科學研究所
5. <i>Lemur fulvus</i>	Amoebiasis	台灣養豬科學研究所
6.Monkey	Tuberculosis	台灣大學獸醫學系
7.Human	Tuberculosis	省立新竹醫院

第二次比較病理學研討會病例（84年4月9日於台北病理中心舉行）：

8.Pigeon	Synovial sarcoma	美國紐約動物醫學中心
9.Cat	Perinephric pseudocyst	台灣大學獸醫學系
10.Human	Choledochocyst	長庚紀念醫院
11.Rat	Bile duct ligation	中興大學獸醫學系
12.Human	<i>H. pylori</i> -induced gastritis	台北病理中心
13.Human	Pseudomembranous colitis	省立新竹醫院
14.Dog	Dirofilariasis	台灣省家畜衛生試驗所
15.Human	Pulmonary dirofilariasis	台北榮民總醫院
16.Squirrel	Toxoplasmosis	台灣養豬科學研究所
17.Pig	Toxoplasmosis	屏東技術學院獸醫學系

第三次比較病理學研討會病例（84年8月27日於國立台灣大學舉行）：

18.Human	Malignant lymphoma	長庚紀念醫院
19.Wistar rat	Malignant lymphoma	國家實驗動物繁殖及研究中心
20.Human	Sparganosis	台北榮民總醫院
21.Chickens	Newcastle disease	國立台灣大學獸醫學系
22.Goldfish	Herpesvirus infection	國立台灣大學獸醫學系
23.Human	Chromomycosis	台北病理中心
24.Human	Metastatic thyroid carcinoma	省立新竹醫院
25.Human	Chordoma	新光吳火獅紀念醫院
26.Pig	Swine salmonellosis	國立中興大學獸醫學系
27.Pig	Vegetative valvular endocarditis	台灣養豬科學研究所

第四次比較病理學研討會病例（84年11月19日於新光吳火獅紀念醫院舉行）：

28. Human	Nocardiosis	台灣省立新竹醫院
29. Largemouth bass	Nocardiosis	屏東縣家畜疾病防治所

30. Dog	Demyelinating canine distemper en	台灣養豬科學研究所
31. Malayan sun bears	Adenovirus infection	國立台灣大學獸醫學系
32. Human	Actinomycosis	台灣省立豐原醫院
33. Human	Tuberculosis	苗栗頭份為恭紀念醫院
34. Dog	Interstitial cell tumor	國立中興大學獸醫學系
35. Human	Carcinoid tumor	長庚紀念醫院
36. Siamese cat	Hepatic carcinoid	美國紐約動物醫學中心
37. Human	Myositis ossificans	台北醫學院

第五次比較病理學研討會（85年 2月 4日於台北市立仁愛醫院舉行）：

中華民國比較病理學會成立大會暨專題演講

第六次比較病理學研討會（85年 6月 9日於台中榮民總醫院舉行）：

38. Ferret	Pheochromocytoma	美國紐約動物醫學中心
39. Human	Extra adrenal pheochromocytoma	新光吳火獅紀念醫院
40. Spragur-Dawley CD 1	Mammary gland fibroadenoma	國家實驗動物繁殖及研究中心
41. Human	Fibroadenoma	省立豐原醫院
42. Pointer bitch	Canine benign mixed type gland tumor	國立中興大學獸醫學系
43. Human	Phyllodes tumor	台中榮民總醫院
44. Dog	Canine oral papilloma	國立台灣大學獸醫學系
45. Human	Squamous cell papilloma	中國醫藥學院

第七次比較病理學研討會（85年11月10日於國立屏東技術學院獸醫系舉行）：

46. Cat	Feline dirofilariasis	美國紐約動物醫學中心
47. Human	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	三軍總醫院
48. wild rodents	Adiaspiromycosis	國立台灣大學獸醫學系
49. Human	Echinococcosis	台北榮民總醫院
50. Piglet	Porcine cytomegalovirus infection	台灣省家畜衛生試驗所
51. Human	Pneumocystis carinii pneumonia	台北病理中心
52. Goslings	Aspergillosis	屏東縣家畜疾病防治所
53. Human	Intracavitary aspergilloma and cavitary tuberculosis, lung.	羅東聖母醫院
54. Human	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	林口長庚紀念醫院

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| 55. Broilers | Infectious laryngo-tracheitis
(Herpesvirus infection) | 國立屏東技術學院獸醫學系 |
|--------------|--|--------------|

第八次比較病理學研討會（86年3月2日於台中榮民總醫院第一會議廳舉行）：

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| 56. Human | Gastrointestinal stromal tumor | 台中榮民總醫院 |
| 57. Chicken | Cecal coccidiosis | 國立中興大學獸醫學系 |
| 58. Human | Tuberculous enteritis with perforation | 佛教慈濟綜合醫院 |
| 59. Dog | Colonic adenocarcinoma | 美國紐約動物醫學中心 |
| 60. Human | Intestinal capillariasis | 台北馬偕醫院 |
| 61. Goose | Spirochetosis | 國立嘉義農專獸醫科 |
| 62. Human | Submucosal leiomyoma of stomach | 頭份為恭紀念醫院 |
| 63. Porcine | Proliferative enteritis (<i>Lawsonia Intracellularis</i> infection) | 屏東縣家畜疾病防治所 |
| 64. Human | 1. Adenocarcinoma of sigmoid colon
2. Old schistosomiasis of rectum | 省立新竹醫院 |
| 65. Carprine | Cryptosporidiosis | 台灣養豬科學研究所 |

第九次比較病理學研討會（86年7月20日於新光吳火獅紀念醫院B1大會議室舉行）：

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|------------------------|--|---------------|
| 66. Chapman's zebra | Echinococcosis | 國立台灣大學獸醫學系 |
| 67. Human | Hepatic ascariasis and cholelithiasis | 彰化基督教醫院 |
| 68. Human | Liver abscess (<i>Klebsillae pneumoniae</i>) | 台北醫學院 |
| 69. Pig | Pseudorabies (Herpesvirus infection) | 台灣養豬科學研究所 |
| 70. Human | Acute Q fever hepatitis | 佛教慈濟綜合醫院 |
| 71. Human | Myelolipoma | 台北耕莘醫院 |
| 72. Mouse | Reticulum cell sarcoma | 國家實驗動物繁殖及研究中心 |
| 73. Human | Hepatocellular carcinoma | 新光吳火獅紀念醫院 |
| 74. Wistar strain rats | Hepatocellular carcinoma induced by aflatoxin B1 | 台灣省農業藥物毒物試驗所 |
| 75. Rabbits | Acute yellow phosphorus intoxication | 國立中興大學獸醫學系 |

第十次比較病理學研討會（86年11月2日於三軍總醫院研究大樓一樓視聽教室舉行）：

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|-------------|---|-----------------------|
| 76. Cat | Polycystic kidney bilateral and renal failure | 美國紐約動物醫學中心 |
| 77. Human | 1.Xanthogranulomatous inflammation with nephrolithiasis, kidney, right.
2.Ureteral stone, right. | 羅東聖母醫院 |
| 78. Chicken | Marek's disease in native chicken | 屏東縣家畜疾病防治所 |
| 79. Human | Emphysematous pyelonephritis | 彰化基督教醫院 |
| 80. SHR rat | 1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate
2.Benign hypertension | 國防醫學院 & 國家實驗動物繁殖及研究中心 |

81. Human	Angiomyolipoma	羅東博愛醫院
82. Human	Inverted papilloma of prostatic urethra	省立新竹醫院
83. SD rats	Phagolysosome-overload nephropathy	國科會國家實驗動物繁殖及研究中心
84. Human	Nephrogenic adenoma	國泰醫院
85. Dog	Renal amyloidosis	台灣養豬科學研究所
86. Human	Multiple myeloma with systemic Amyloidosis	佛教慈濟綜合醫院
87. Human	Squamous cell carcinoma of renal pelvis and calyces with extension to the ureter	台北病理中心
88. Human	Fibroepithelial polyp of the ureter	台北耕莘醫院
89. Goose	1. Severe visceral gout due to kidney damaged 2. Infectious serositis	國立中興大學獸醫學系
90. Human	Clear cell sarcoma of kidney	台北醫學院
91. orange-rumped agoutis	Hypervitaminosis D	國立台灣大學獸醫學系

第十一次比較病理學研討會（87年3月1日於佛教慈濟綜合醫院舉行）：

92. Pig	Foot-and-mouth disease (FMD)	屏東縣家畜疾病防治所
93. Dog	Mammary gland adenocarcinoma, complex type, with chondromucinous differentiation	國立台灣大學獸醫學系
94. Human	1. Breast, left, modified radical mastectomy, showing papillary carcinoma, invasive 2. Nipple, left, modified radical mastectomy, papillary carcinoma, invasive 3. Lymph node, axillary, left, lymphadenectomy, carcinoma, metastatic	羅東聖母醫院
95. Dog	Transmissible venereal tumor	國立中興大學獸醫學系
96. Human	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	彰化基督教醫院
97. Tiger	Carcinosarcomas	台灣養豬科學研究所
98. Human	Mucinous carcinoma with intraductal carcinoma	省立豐原醫院
99. Mouse	Mammary gland adenocarcinoma, type pulmonary metastasis, BALB/cBYJ mouse	國家實驗動物繁殖及研究中心
100. Human	Malignant fibrous histiocytoma and paraffinoma	中國醫藥學院
101. Pig	Swine pox	國立屏東科技大學獸醫學系
102. Human	Pleomorphic adenoma (benign mixed tumor)	佛教慈濟綜合醫院

中華民國比較病理學會第一次至第十一次比較病理學研討會病例分類
一覽表

分 類	病例 編號	診 斷	動物別	提 供 單 位
腫 瘤	1	Myxoma	Dog	美國紐約動物醫學中心
	2	Chordoma	Ferret	美國紐約動物醫學中心
	3	Ependymoblastoma	Human	長庚紀念醫院
	8	Synovial sarcoma	Pigeon	美國紐約動物醫學中心
	18	Malignant lymphoma	Human	長庚紀念醫院
	19	Malignant lymphoma	Wistar rat	國家實驗動物繁殖及研究中心
	24	Metastatic thyroid carcinoma	Human	省立新竹醫院
	25	Chordoma	Human	新光吳火獅紀念醫院
	34	Interstitial cell tumor	Dog	國立中興大學獸醫學系
	35	Carcinoid tumor	Human	長庚紀念醫院
	36	Hepatic carcinoid	Siamese cat	美國紐約動物醫學中心
	38	Pheochromocytoma	Ferret	美國紐約動物醫學中心
	39	Extra adrenal pheochromocytoma	Human	新光吳火獅紀念醫院
	40	Mammary gland fibroadenoma	Spragur-Dawley CD rat	國家實驗動物繁殖及研究中心
	41	Fibroadenoma	Human	省立豐原醫院
	42	Canine benign mixed type mammary gland tumor	Pointer bitch	國立中興大學獸醫學系
	43	Phyllodes tumor	Human	台中榮民總醫院
	44	Canine oral papilloma	Dog	國立台灣大學獸醫學系
	45	Squamous cell papilloma	Human	中國醫藥學院
	47	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	Human	三軍總醫院
	56	Gastrointestinal stromal tumor	Human	台中榮民總醫院
	59	Colonic adenocarcinoma	Dog	美國紐約動物醫學中心
	62	Submucosal leiomyoma of stomach	Human	頭份為恭紀念醫院
	64	1. Adenocarcinoma of sigmoid colon 2. Old schistosomiasis of rectum	Human	省立新竹醫院

71.	Myelolipoma	Human	台北耕莘醫院
72.	Reticulum cell sarcoma	Mouse	國家實驗動物繁殖及研究中心
73.	Hepatocellular carcinoma	Human	新光吳火獅紀念醫院
74.	Hepatocellular carcinoma induced by aflatoxin B1	Wistar strain rats	台灣省農業藥物毒物試驗所
81.	Angiomyolipoma	Human	羅東博愛醫院
82.	Inverted papilloma of prostatic urethra	Human	省立新竹醫院
84.	Nephrogenic adenoma	Human	國泰醫院
86.	Multiple myeloma with systemic Amyloidosis	Human	佛教慈濟綜合醫院
87.	Squamous cell carcinoma of renal pelvis and calyces with extension to the ureter	Human	台北病理中心
88.	Fibroepithelial polyp of the ureter	Human	台北耕莘醫院
90.	Clear cell sarcoma of kidney	Human	台北醫學院
93.	Mammary gland adenocarcinoma, complex type , with chondromucinous differentiation	Dog	國立台灣大學獸醫學系
94.	1.Breast, left, modified radical mastectomy, showing papillary carcinoma, invasive 2.Nipple, left, modified radical mastectomy, papillary carcinoma, invasive 3.Lymph node, axillary, left, lymphadenectomy, papillary carcinoma, metastatic	Human	羅東聖母醫院
95.	Transmissible venereal tumor	Dog	國立中興大學獸醫學系
96.	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	Human	彰化基督教醫院
97.	Carcinosarcomas	Tiger	台灣養豬科學研究所
98.	Mucinous carcinoma with intraductal Carcinoma	Human	省立豐原醫院
99.	Mammary gland adenocarcinoma, type B, with pulmonary metastasis, BALB/cBYJ mouse	Mouse	國家實驗動物繁殖及研究中心
100.	Malignant fibrous histiocytoma and paraffinoma	Human	中國醫藥學院
102.	Pleomorphic adenoma (benign mixed tumor)	Human	佛教慈濟綜合醫院

細菌	6	Tuberculosis	Monkey	台灣大學獸醫學系
	7	Tuberculosis	Human	省立新竹醫院
	12	<i>H. pylori</i> -induced gastritis	Human	台北病理中心
	13	Pseudomembranous colitis	Human	省立新竹醫院
	26	Swine salmonellosis	Pig	國立中興大學獸醫學系
	27	Vegetative valvular endocarditis	Pig	台灣養豬科學研究所
	28	Nocardiosis	Human	台灣省立新竹醫院
	29	Nocardiosis	Largemouth bass	屏東縣家畜疾病防治所
	32	Actinomycosis	Human	台灣省立豐原醫院
	33	Tuberculosis	Human	苗栗頭份為恭紀念醫院
	53	Intracavitary aspergilloma and cavitary tuberculosis, lung.	Human	羅東聖母醫院
	54	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	Human	林口長庚紀念醫院
	58	Tuberculous enteritis with perforation	Human	佛教慈濟綜合醫院
	61	Spirochetosis	Goose	國立嘉義農專獸醫科
	63	Proliferative enteritis (<i>Lawsonia intracellularis</i> infection)	Porcine	屏東縣家畜疾病防治所
	68	Liver abscess (<i>Klebsillae pneumoniae</i>)	Human	台北醫學院
	77.	1.Xanthogranulomatous inflammation with nephrolithiasis, kidney, right. 2.Ureteral stone, right.	Human	羅東聖母醫院
	79.	Emphysematous pyelonephritis	Human	彰化基督教醫院
	89.	1. Severe visceral gout due to kidney damaged 2.Infectious serositis	Goose	國立中興大學獸醫學系
病毒	21	Newcastle disease	Chickens	國立台灣大學獸醫學系
	22	Herpesvirus infection	Goldfish	國立台灣大學獸醫學系
	30	Demyelinating canine distemper encephalitis	Dog	台灣養豬科學研究所
	31	Adenovirus infection	Malayan sun bears	國立台灣大學獸醫學系
	50	Porcine cytomegalovirus infection	Piglet	台灣省家畜衛生試驗所
	55	Infectious laryngo-tracheitis (Herpesvirus infection)	Broilers	國立屏東技術學院獸醫學系
	69	Pseudorabies (Herpesvirus infection)	Pig	台灣養豬科學研究所
	78.	Marek's disease in native chicken	Chicken	屏東縣家畜疾病防治所

	92.	Foot- and- mouth disease (FMD)	Pig	屏東縣家畜疾病防治所
	101.	Swine pox	Pig	國立屏東科技大學獸醫學系
黴菌	23	Chromomycosis	Human	台北病理中心
	47	Lung: metastatic carcinoma associated with cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma (primary)	Human	三軍總醫院
	48	Adiaspiromycosis	Wild rodents	國立台灣大學獸醫學系
	52	Aspergillosis	Goslings	屏東縣家畜疾病防治所
	53	Intracavitary aspergilloma and cavitary tuberculosis, lung.	Human	羅東聖母醫院
	54	Fibrocalcified pulmonary TB, left Apex. Mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	Human	林口長庚紀念醫院
寄生蟲	14	Dirofilariasis	Dog	台灣省家畜衛生試驗所
	15	Pulmonary dirofilariasis	Human	台北榮民總醫院
	20	Sparganosis	Human	台北榮民總醫院
	46	Feline dirofilariasis	Cat	美國紐約動物醫學中心
	49	Echinococcosis	Human	台北榮民總醫院
	60	Intestinal capillariasis	Human	台北馬偕醫院
	64	1.Adenocarcinoma of sigmoid colon 2.Old schistosomiasis of rectum	Human	省立新竹醫院
	66	Echinococcosis	Chapman's zebra	國立台灣大學獸醫學系
	67	Hepatic ascariasis and cholelithiasis	Human	彰化基督教醫院
原蟲	4	Cryptosporidiosis	Goat	台灣養豬科學研究所
	15	Amoebiasis	<i>Lemur fulvus</i>	台灣養豬科學研究所
	16	Toxoplasmosis	Squirrel	台灣養豬科學研究所
	17	Toxoplasmosis	Pig	屏東技術學院獸醫學系
	51	<i>Pneumocystis carinii</i> pneumonia	Human	台北病理中心
	57	Cecal coccidiosis	chicken	國立中興大學獸醫學系
	65	Cryptosporidiosis	Carprine	台灣養豬科學研究所
立克次體	70	Human	Acute Q fever hepatitis	佛教慈濟綜合醫院
其它	9	Perinephric pseudocyst	Cat	台灣大學獸醫學系
	10	Choledochocyst	Human	長庚紀念醫院
	11	Bile duct ligation	Rat	中興大學獸醫學系

	37.	Myositis ossificans	Human	台北醫學院
	75.	Acute yellow phosphorus intoxication	Rabbits	國立中興大學獸醫學系
	76.	Polycystic kidney bilateral and renal failure	Cat	美國紐約動物醫學中心
	80.	1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate 2.Benign hypertension	SHR rat	國防醫學院 & 國家實驗動物繁殖及研究中心
	83.	Phagolysosome-overload nephropathy	SD rats	國家實驗動物繁殖及研究中心
	85.	Renal amyloidosis	Dog	台灣養豬科學研究所
	89.	1.Severe visceral gout due to kidney damaged 2.Infectious serositis	Goose	國立中興大學獸醫學系
	91.	Hypervitaminosis D	orange-rumped agoutis	國立台灣大學獸醫學系

中華民國比較病理學會第一次至第十一次比較病理研討會
各單位提供討論病例次數統計
(八十三年十月三十日至八十七年三月一日)

單位名稱	提供討論病例數
國立台灣大學獸醫學系	10
台灣養豬科學研究所	9
國立中興大學獸醫學系	8
美國紐約動物醫學中心	8
省立新竹醫院	6
長庚紀念醫院	5
屏東縣家畜疾病防治所	5
國家實驗動物繁殖及研究中心	5
佛教慈濟綜合醫院	4
台北病理中心	4
台北榮民總醫院	3
新光吳火獅紀念醫院	3
省立豐原醫院	3
國立屏東科技大學獸醫學系	3
彰化基督教醫院	3
台北醫學院	3
羅東聖母醫院	3
頭份為恭紀念醫院	2
台中榮民總醫院	2
台灣省家畜衛生試驗所	2
中國醫藥學院	2
台北耕莘醫院	2
國泰醫院	1
三軍總醫院	1
羅東博愛醫院	1
台北馬偕醫院	1
國防醫學院	1
國立嘉義農專獸醫科	1
台灣省農業藥物毒性試驗所	1
合 計	102

會員資料更新服務

各位會員：

您好！如果您的會員資料有更新或誤刊情形，麻煩您填妥表格後寄回學會秘書處或電話連絡：

中華民國比較病理學會秘書處
苗栗縣竹南郵政信箱 23 號
病理生物系 邱慧英 小姐
Tel: (037) 672352轉505
Fax: (037) 687803

-----中華民國比較病理學會-----
會員資料更改卡

姓 名：_____ 會員類別：☐ 一般會員
☐ 學生會員
☐ 贊助會員

最高學歷：_____

服務單位：_____ 職 稱：_____

永久地址：_____

通訊地址：_____

電 話：_____ 傳 真：_____

E-Mail Address：_____

中 華 民 國 比 較 病 理 學 會

誠 摯 邀 請 您 加 入

入 會 辦 法

一、本會會員申請資格為：

- (一) 一般會員：贊同本會宗旨，年滿二十歲，具有國內外大專院校（或同等學歷）生命科學及其它相關科系畢業資格或高職畢業從事生命科學相關工作滿兩年者。
- (二) 學生會員：贊同本會宗旨，在國內、外大專院校生命科學或其他相關科系肄業者（請檢附學生身份證明）。
- (三) 贊助會員：贊助本會工作之團體或個人。
- (四) 榮譽會員：凡對比較病理學術或會務之推廣有特殊貢獻，經理事會提名並經會員大會通過者。

二、會員：

- (一) 入會費：一般會員新台幣一仟元，學生會員一百元，贊助會員伍仟元，於入會時繳納。
- (二) 常年會費：一般會員新台幣伍佰元，學生會員一百元。

三、請填妥入會申請表，並連同入會費及常年會費（一般會員合計新台幣壹仟伍佰元，學生會員合計貳佰元，贊助會員伍仟元）以郵政匯票或支票（抬頭請開：中華民國比較病理學會）寄苗栗縣竹南郵政信箱23號，中華民國比較病理學會秘書長劉振軒博士，電話：037-672352轉507，傳真037-687803。

誌 謝

下列機關團體贊助本研討會，特此致上最深忱的謝意

佛教慈濟綜合醫院

行政院農業委員會

台灣省政府農林廳

府泉實業有限公司

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