

中華民國比較病理學會章程

第一章 總 則

- 第一條 本會定名為中華民國比較病理學會，英文名稱為 Chinese Society of Comparative Pathology (CSCP) (以下簡稱本會)。
- 第二條 本會依內政部人民團體法設立，為非營利目的之社會團體，以結合人類醫學與動物醫學資源，提倡比較病理學之研究與發展，交換研究教學心得，聯絡會員友誼及促進國際間比較醫學之交流為宗旨。
- 第三條 本會以全國行政區域為組織區域，會址設於主管機關所在地區，並得報經主管機關核准設立分支機構。前項分支機構組織簡則由理事會擬訂，報請主管機關核准後行之。會址及分支機構之地址於設置及變更時應報請主管機關核備。
- 第四條 本會之任務如左：
- 一、 提倡比較病理學之研究與發展。
 - 二、 舉辦學術演講會、研討會及相關訓練課程。
 - 三、 建立國內比較醫學相關資料庫。
 - 四、 發行比較病理學相關刊物。
 - 五、 促進國內、外比較醫學之交流。
 - 六、 其他有關比較病理學術發展之事項。
- 第五條 本會之主管機關為內政部。目的事業主管機關依章程所訂之宗旨與任務，主要為行政院衛生署及農業委員會，其目的事業應受各該事業主管機關之指導與監督。

第二章 會 員

- 第六條 本會會員申請資格如下：
- 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它相關

科系肄業者 (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展有特殊貢獻，經理事會提名並經會員大會通過者。

前項一、二、三項會員申請時應填具入會申請書，經一般會員二人之推薦，經理事會通過，並繳納會費。學生會員身份改變成一般會員時，得再補繳一般會員之常年會費之差額後，即成為一般會員，榮譽會員免繳入會費與常年會費 (86 修訂)。

第七條 一般會員有表決權、選舉權、被選舉與罷免權，每一會員為一權。贊助會員、學生會員與榮譽會員無前項權利。

第八條 會員有遵守本會章程、決議及繳納會費之義務。

第九條 會員有違反法令、章程或不遵守會員大會決議時，得經理事會決議，予以警告或停權處分，其危害團體情節重大者，得經會員大會決議予以除名。

第十條 會員喪失會員資格或經會員大會決議除名者，即為出會。

第十一條 會員得以書面敘明理由向本會聲明退會。但入會費與當年所應繳納的常年會費不得申請退費。

第三章 組織及職員

第十二條 本會以會員大會為最高權力機構。

第十三條 會員大會之職權如下：

- 一、 訂定與變更章程。
- 二、 選舉及罷免理事、監事。
- 三、 議決入會費、常年會費、事業費及會員捐款之方式。
- 四、 議決年度工作計畫、報告、預算及決算。
- 五、 議決會員之除名處置。
- 六、 議決財產之處分。
- 七、 議決本會之解散。
- 八、 議決與會員權利義務有關之其他重大事項。

前項第八款重大事項之範圍由理事會訂定之。

第十四條 本會置理事十五人，監事五人，由會員選舉之，分別成立理事會、監事會。選舉前項理事、監事時，依計票情形得同時選出候補理事五人，候補監事一

人，遇理事或監事出缺時，分別依序遞補之。

本屆理事會得提出下屆理事及監事候選人參考名單。

第十五條 理事會之職權如下：

- 一、 審定會員之資格。
- 二、 選舉及罷免常務理事及理事長。
- 三、 議決理事、常務理事及理事長之辭職。
- 四、 聘免工作人員。
- 五、 擬訂年度工作計畫、報告、預算及決算。
- 六、 其他應執行事項。

第十六條 理監事置常務理事五人，由理事互選之，並由理事就常務理事中選舉一人為理事長。

理事長對內綜理監督會議，對外代表本會，並擔任會員大會、理事會主席。
理事長因事不能執行職務時，應指定常務理事一人代理之，未指定或不能指定時，由常務理事互推一人代理之。

理事長或常務理事出缺時，應於一個月內補選之。

第十七條 監事會之職權如左：

- 一、 監察理事會工作之執行。
- 二、 審核年度決算。
- 三、 選舉及罷免常務監事。
- 四、 議決監事及常務監事之辭職。
- 五、 其他應監察事項。

第十八條 監事會置常務監事一人，由監事互選之，監察日常會務，並擔任監事會主席。
常務監事因事不能執行職務時，應指定監事一人代理之，未指定或不能指定時，由監事互推一人代理之。監事會主席（常務監事）出缺時，應於一個月內補選之。

第十九條 理事、監事均為無給職，任期三年，連選得連任。理事長之連任以一次為限。

第二十條 理事、監事有下列情事之一者，應即解任：

- 一、 喪失會員資格。
- 二、 因故辭職經理事會或監事會決議通過者。
- 三、 被罷免或撤免者。
- 四、 受停權處分期間逾任期二分之一者。

- 第二十一條 本會置祕書長一人，承理事長之命處理本會事務，令置其他工作人員若干人，由理事長提名經理事會通過後聘免之，並報主管機關備查。但祕書長之解聘應先報主管機關核備。
- 前項工作人員不得由選任之職員（理監事）擔任。
- 工作人員權責及分層負責事項由理事會令另定之。
- 第二十二條 本會得設各種委員會、小組或其它內部作業組織，其組織簡則由理事會擬定，報經主機關核備後施行，變更時亦同。
- 第二十三條 本會得由理事會聘請無給顧問若干人，其聘期與理事、監事之任期同。

第四章 會議

- 第二十四條 會員大會分定期會議與臨時會議兩種，由理事長召集，召集時除緊急事故之臨時會議外應於十五日前以書面通知之。定期會議每年召開一次，臨時會議於理事會過半數認為必要，或經會員五分之一以上之請求，或監事會半數函請召集時召開之。
- 第二十五條 會員不能親自出席會員大會時，得以書面委託其他會員代理，每一會員以代理一人為限。
- 第二十六條 會員大會之決議，以出席人數過半數之同意行之。但章程之訂定與變更、會員之除名、理事及監事之罷免、財產之處置、本會之解散及其他與會權利義務有關之重大事項應有出席人數三分之二以上同意。但本會員如果辦理法人登記後，章程之變更應以出席人數四分之三以上之同意或全體會員三分之二以上書面之同意行之。
- 第二十七條 理事會及監事會至少每六個月各舉行會議一次，必要時得召開聯席會議或臨時會議。
- 前項會議召集時除臨時會議外。應於七日以前以書面通知，會議之決議各以理事、監事過半數之出席，出席人較多數之同意行之。
- 第二十八條 理事應出席理事會議，監事應出席監事會議，不得委託出席；理事、監事連續二次無故缺席理事會、監事會者，視同辭職。

第五章 經費及會計

第二十九條 本會經費來源如下：

- 一、入會費：一般會員新台幣壹仟元，學生會員壹佰元，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，學生會員壹佰元。
- 三、事業費。
- 四、會員捐款。
- 五、委託收益。
- 六、基金及其孳息。
- 七、其他收入。

第三十條 本會會計年度以國曆年為準，自每年一月一日起至十二月三十一日止。

第三十一條 本會每年於會計年度開始前二個月由理事會編造年度工作計劃、收支預算表、員工待遇表，提會員大會通過（會員大會因故未能如期召開者，先提理監事聯席會議通過），於會計年度開始前報主管機關核備，並於會計年度終了後二個月內由理事會編造年度工作報告、收支決算表、現金出納表、資產負債表、財產目錄及基金收支表，送監事會審核後，造具審核意見書送還理事會，提會員大會通過，於三月底前報主管機關核備（會員大會未能如期召開者，需先報主管機關備查）。

第三十二條 本會解散後，剩餘財產歸屬所在地之地方自治團體或主管機關指定之機關團體所有。

第六章 附 則

第三十三條 本章程未規定事項，悉依有關法令規定辦理。

第三十四條 本章程經大會通過，報經主管機關核備後施行，變更時亦同。

第三十五條 本章程經本會民國八十五年二月四日第一屆第一次會員大會通過，並報經內政部 85 年 3 月 14 日台(85)內社字第 8507009 號函准予備查。

中華民國比較病理學會第一屆理監事名單簡歷冊									
職別	姓名	性別	出生年月日	學歷	經歷	現任本職	戶籍住址	電話	傳真
理事長	黃文哲	男	25/12/12	華盛頓大學病理博士	華盛頓大學病理系教授	台北病理中心執行長兼解剖病理部主任	103 台北市重慶北路三段146號6樓	02-3257566	02-85962075
常務理事	何逸僊	男	39/10/25	國防醫學院病理學碩士	國防醫學院、三軍總醫院主治醫師	長庚醫學院、醫院主治醫師、副教授	333 桃園龜山長庚醫護社區211號2F	03-3284277	03-3280147
常務理事	祝志平	男	46/2/25	台大病理研究所碩士	台北榮民總醫院住院醫師	羅東聖母醫院	265 宜蘭縣羅東鎮中正南路160號羅東聖母醫院病理科	039-572916	039-572916
常務理事	陳三多	男	40/8/11	比利時魯汶大學博士	中興大學獸醫學系副教授	中興大學獸醫學系教授	402 台中市國光路250號	04-2853552	04-2853552
常務理事	洪信雄	男	31/11/27	中興大學獸醫研究所碩士	屏東縣家畜疾病防治所技正	屏東縣家畜疾病防治所所長	900 屏東市水源街100-1號	08-7224109	08-7224432
理事	蔡信雄	男	37/3/20	北海道大學獸醫學博士	屏東技術學院獸醫學院院長	屏東技術學院獸醫系教授	912 屏東縣內埔鄉學府路1號	08-7740297	08-7740295
理事	方中民	男	17/10/10	日本大阪醫科大學醫學博士	中國醫藥學院院長	台灣高等法院檢察署法醫中心召集人	103 台北市迪化街175巷16號	02-7370570	02-7359413
理事	朱瑞民	男	34/7/14	美國愛荷華大學博士	台灣養豬科學研究所所長	台灣大學獸醫學系教授	350 竹南鎮中華路19巷4弄6號	037-661042	
理事	陳安	男	45/10/11	國防醫學院博士	三軍總醫院主治醫師	三軍總醫院實驗病理科主任	100 台北市汀州路3段18號3樓	02-3651003	02-3672941
理事	陳東榮	男	38/12/16	台大病理學碩士	新光吳火師紀念醫院病理檢驗科主任	新光吳火師紀念醫院病理檢驗科主任	111 台北市士林區文昌路95號	02-8389307	02-8389360
理事	鄭益謙	男	45/5/14	美國佛羅里達州立大學博士	台灣養豬科學研究所副研究員	台灣養豬科學研究所副研究員兼主任	350 竹南郵政第23號信箱	037-672352-526	037-692820
理事	梁善居	男	42/11/12	美國阿拉巴馬大學比較醫系博士	國防醫學院副教授、動物中心主任	國防醫學院副教授、動物中心主任	100 台北市汀州路三段24巷五弄22號4F	02-3675843	02-3652108
理事	施洽雯	男	46/8/30	國防醫學院病理研究所	中山醫學院病理科副教授	羅東博愛醫院病理科主任	265 羅東鎮南昌街83號	039-543131-2632	039-574993
理事	周冠	男	40/8/30	國防醫學院醫學系	台中榮民總醫院病理部專科醫師	台中榮民總醫院病理部一般病理科主任	407 台中市台中港路三段160號病理部	04-3592525	04-3596532
理事	呂福江	男	37/11/21	美國漢尼門大學病理學博士	國防醫學院病理學研究所所長	耕莘醫院病理科主任	231 台北市新店市中正路362號病理科	02-2193391-5236	02-2193506
常務監事	龐飛	男	42/8/18	美國伊利諾大學獸醫病理學博士	台灣大學獸醫學系副教授	台灣大學獸醫學系教授	106 台北市舟山路142號獸醫系	02-3963932	02-23661475
監事	鄭謙仁	男	48/7/21	美國北卡羅萊納大學哲學博士	台灣大學獸醫學系副教授	台灣大學獸醫學系教授	106 台北市舟山路142號獸醫系	02-23630231-285	02-23661475
監事	林永和	男	46/2/24	台大病理研究所	台北醫學院病理科講師	台北醫學院病理科講師	110 台北市吳興街250號	02-7361661-641	02-3770054
監事	李進成	男	49/6/06	英國倫敦大學神經病理博士	長庚醫院內科醫師	新光吳火獅紀念醫院病理檢驗科醫師	112 台北市北投區行義路154巷31號7F	02-8389306	02-8389306
監事	羅登源	男	49/1/13	中興大學獸醫碩士	嘉義農專獸醫科講師	嘉義農專獸醫科講師	600 嘉義市鹿寮里紅毛埤84號	05-2766141-620	05-2784871

中華民國比較病理學會第二屆理監事名單簡歷冊									
職別	姓名	性別	出生年月日	學歷	經歷	現任本職	戶籍住址	電話	傳真
理事長	黃文哲	男	25/12/12	華盛頓大學病理博士	華盛頓大學病理系教授	台北病理中心解剖病理部主任	103 台北市重慶北路三段 146 號 6 樓	02-3257566	02-85962075
常務理事	江宏	男	32/11/7	國防醫學院	台北榮總病理部	台北榮總病理檢驗部主任	105 台北市復興北路 313 巷 17 號 6 樓	02-28757022	02-28740920
常務理事	朱瑞民	男	34/7/14	美國愛荷華大學博士	台灣養豬科學研究所所長	台灣大學獸醫學系教授	106 台北市舟山路 142 號	02-23630231-1206-7	
常務理事	陳三多	男	40/8/11	比利時魯汶大學博士	中興大學獸醫學系副教授	中興大學獸醫學院教授	402 台中市國光路 250 號	04-2853552	04-2853552
常務理事	洪信雄	男	31/11/27	中興大學獸醫研究所碩士	屏東縣家畜疾病防治所技正	屏東縣家畜疾病防治所所長	900 屏東市水源街 100-1 號	08-7224109	08-7224432
理事	鄭謙仁	男	48/7/31	美國北卡羅萊納州立大學哲學博士	台灣大學獸醫學系副教授	台灣大學獸醫學系副教授	111 台北市中山北路 622 段 419 巷 29 號 3 樓	02-23630231-285	02-23661475
理事	祝志平	男	46/2/25	台大病理研究所碩士	台北榮民總醫院住院醫師	羅東聖母醫院病理科主任	265 宜蘭縣羅東鎮中正南路 160 號羅東聖母醫院病理科	039-544106-6113	039-572916
理事	陳東榮	男	38/12/16	國立台灣大學病理學研究所	長庚紀念醫院林口醫學中心病理科系主治醫師	新光吳火獅紀念醫院病理檢驗科主任	111 台北市士林區文昌路 95 號	08-28389307	02-8389360
理事	許永祥	男	48/10/30	國立台大醫學院病理學研究所碩士	台大醫學院助教	慈濟醫院病理科主任	970 花蓮慈濟醫院病理科	038-561825 轉 2124	038-560794
理事	劉錫光	男	14/12/1	美國加州大學研究院比較病理學哲學博士	紐約 Bronx 動物園紐約野生動物保護學會	紐約動物醫學中心高級研究員	The Animal Medical Center 510 East 62 nd Street New York, New York 10021	212-838-8100	212-8329288 212-9329630
理事	賴銘淙	男	47/10/14	台大醫學院病理學研究所碩士	頭份為恭醫院	嘉義華濟醫院	403 台中市太原路一段 34 號	05-2378111-2560-1	05-2373703
理事	張聰洲	男	41/11/29	國立中興大學獸研所碩士班	國立屏東技術學院助教	國立屏東科技大學講師	710 台南縣永康市中山南路 231 巷 35 號	06-2333529	08-7740295
理事	施洽雯	男	46/8/30	國防醫學院病理研究所	中山醫學院病理科副教授	羅東博愛醫院病理科主任	265 羅東鎮南昌街 83 號	039-543131-2632	039-574993
理事	周冠	男	40/8/30	國防醫學院醫學系	台中榮民總醫院病理部專科醫師	台中榮民總醫院病理部一般病理科主任	407 台中市港路三段 160 號病理部	04-3592525-5720	04-3596532
理事	呂福江	男	37/11/21	美國漢尼門大學病理學博士	國防醫學院病理學研究所所長	耕莘醫院病理科主任	231 台北市新店市三民路 68 號 7 樓	02-2193391-5236	02-2193506
常務監事	龐飛	男	42/8/18	美國伊利諾大學獸醫病理學博士	台灣大學獸醫學系副教授	台灣大學獸醫學系教授	100 台北市南昌路二段 18 巷 7 號	02-3963932	02-23661475
監事	葉祥森	男	50/9/5	國立陽明大學醫學系	台北榮民總醫院病理部代主治醫師	行政院衛生署新竹醫院病理科主任	300 新竹市經國路一段 442 巷 25 號	035-326151-3801	035-333376
監事	林永和	男	46/02/24	台大病理研究所	台北醫學院病理科講師	台北醫學院病理科講師	110 台北市吳興街 250 號	02-7361661-641	02-3770054
監事	李進成	男	49/06/06	英國倫敦大學神經病理博士	長庚醫院內科醫師	新光吳火獅紀念醫院病理檢驗科主治醫師	112 台北市北投區行義路 154 巷 31 號 7F	02-8389306	02-8389360
監事	簡基憲	男	43/5/23	台灣大學解剖學研究所博士班	中美獸醫院院長	成功大學醫學院解剖學科講師	701 台南市東區崇善路 205 巷 24 號 4 樓	06-2905679	06-2905680

中華民國比較病理學會八十八年度會員大會

會議紀錄

一、時間：88年4月11日 12:00-13:00

二、地點：國立台灣大學農學院附設動物醫院 會議室

三、主席：黃文哲

四、出席會員：全體會員

五、記錄：詹晴評

六、主席報告：略。

七、決議與討論：

1. 第十六次比較病理研討會以最近發生的立百病毒及日本腦炎為討論專題

2. 改選第二屆理監事

理事-監票人：徐久忠

唱票人：林正忠

計票人：周國政

理事當選人：黃文哲、江宏、祝志平、許永祥、賴銘淙、陳東榮、

呂福江、周冠、洪信雄、陳三多、朱瑞民、劉錫光、施洽雯、

張聰洲、鄭謙仁。

監事-監票人：黃敬堯

唱票人：王群

計票人：邱慧英

監事當選人：李進成、葉祥森、林永和、龐飛、簡基憲。

八. 散會

中華民國比較病理學會第二屆第一次理監事聯席會議

會議紀錄

- 一、時間：88年4月11日 16:00~18:00
二、地點：國立台灣大學農學院附設動物醫院 會議室
三、主席：黃文哲
四、出席理事：黃文哲、陳三多、祝志平、鄭謙仁、劉錫光、朱瑞民、賴銘淙、
洪信雄、張聰洲、施洽雯、許永祥
出席監事：葉祥森、林永和、龐飛、簡基憲、李進成
五、列席人員：方中民、余忠泰、劉振軒
六、記錄：詹晴評
七、主席報告：略。

八、決議與討論：

1. 由朱瑞民博士、劉振軒博士及李進成醫師籌備第十六次比較病理研討會事宜。
2. 未來研討會主題：分別為 Nipah virus、Hendra virus、T.B.、禽流感、寄生蟲、黴菌性疾病及狂犬病等。
3. 第十六次比較病理研討會
主題：人畜共通傳染病（中樞神經系統之感染）
地點：新光醫院（需先行文）
日期：八十八年五月卅日（暫定）
議程：

疾 病	內 容
上午 JE	1. 流行病學（人、豬）（廖明一、金傳春） 2. 人：臨床症狀、病理變化 3. 動物：臨床症狀、病理變化
Nipah virus & Hendra virus	1. 病史 2. 人與動物（朱瑞民教授及省立台東醫院）
下午 JE	慈濟醫院
Streptococcus suis	張聰洲老師
Nipah virus	朱瑞民教授
JE	豬、馬、鼠之臨床症狀及病理變化 （吳福明、廖明一、淡水試驗所）

4. 選舉常務理事及常務監事

常務理事

當選名單：黃文哲、江宏、洪信雄、陳三多、朱瑞民

備註：洪信雄先生與祝志平醫師得票均為四票，經公開抽籤後，由

洪信雄先生當選

常務監事

當選名單：龐飛先生

5. 常務理事黃文哲先生當選第二屆理事長。

6. 理事長提議由劉振軒先生繼續擔任本會第二屆秘書長，獲全體理監事同意。

九. 散會

中華民國比較病理學會第二屆第二次理監事聯席會議會議記錄

- 一、 時間：中華民國八十八年六月六日（星期日）上午 08:00~下午 03:30
- 二、 地點：新光吳火獅紀念醫院 地址：台北市士林區文昌路 95 號
- 三、 主席：陳三多 理事
- 四、 出席理事：江宏，陳東榮，陳三多，張聰洲，祝志平，賴銘淙，許永祥。
出席監事：李進成，林永和。
- 五、請假理事：朱瑞民，施洽雯，周冠，呂福江，黃文哲，洪信雄，鄭謙仁，劉錫光。
請假監事：龐飛，葉祥森，簡基憲。
- 六、 列席人員：劉振軒
- 七、 記錄：劉振軒
- 八、 主席報告：(略)
- 九、 討論與決議：
 - 1.請討論八十八年第十七次比較病理研討會主題、時間、地點與負責理事。
決議:八十八年第十七次比較病理研討會主題為中樞神經系統感染專題 II。研討會將於八十八年十月下旬於臺北市石牌路二段 201 號台北榮民總醫院舉辦，負責理事為江宏理事。
- 十、 散會

中華民國比較病理學會第二屆第三次理監事聯席會議會議記錄

一、時間：88 年 10 月 31 日 中午 12:30~下午 2:30

二、地點：台北榮民總醫院(第一會議室)

三、主席：黃文哲理事長

四、出席理事：黃文哲，江宏，陳三多，張聰洲，洪信雄，鄭謙仁，祝志平，賴銘淙，劉錫光，許永祥。

出席監事：李進成，龐飛，葉祥森，林永和，簡基憲。

五、請假理事：朱瑞民，陳東榮，施洽雯，周冠，呂福江。

請假監事：

六、列席人員：劉振軒

七、記錄：劉振軒

八、主席報告：(略)

九、討論與決議：

1.請討論八十九年第十八次比較病理研討會主題、時間、地點與負責理事。

決議:八十九年第十八次比較病理研討會主題為骨骼與軟組織腫瘤(Bone and Soft Tissue tumors)。研討會將於八十九年四月中下旬於臺北市基隆路三段一五三號台大動物醫院(地下一樓國際會議廳)舉辦，負責理事為鄭謙仁理事。

2.請追認同意購買電腦設備一批(如附件)。

決議:同意。

3.擬請同意本會地址設於台北市舟山路 142 號台灣大學獸醫學系。

決議:請秘書處擬文函請台灣大學獸醫學系系主任同意後，發函內政部遷移會址。

4.如何增加本會財源。

決議:本會財源將以增加廣告收入為主，辦法由該次研討會負責理監事鼓勵所認識及來往之廠商贊助。

十、散會

中華民國比較病理學會

收支決算表

中華民國 88 年 1 月 1 日至 88 年 12 月 31 日

科 目				決算數	預算數	決算與預算 比較數	
款	項	目	名 稱			增加	減少
1			本會經費收入	123,814	600,000		476,186
	1		入會費	7,500	100,000		92,500
	2		常年會費	76,700	145,000		68,300
	3		贊助會費	0	65,000		65,000
	4		利息收入	16,114	40,000		23,886
	5		其他收入	23,500	250,000		226,500
	6		獎(捐)助款項				
2			本會經費支出	439,948	610,000		208,052
			獎(捐)助款項				
	1		人事費	45,400	92,000		46,600
		1	兼職人員車馬費	34,000	72,000		38,000
		2	其它人事費	11,400	20,000		8,600
	2		辦公費	280,234	350,000		69,766
		1	印刷費	224,318	240,000		15,682
		2	旅運費	13,444	30,000		16,556
		3	郵電費	36,972	50,000		13,028
		4	公共關係費	5,500	30,000		24,500
	3		業務費	53,575	90,000		36,425
		1	會議會	53,575	90,000		36,425
	4		雜費支出	22,739	30,000		7,261
	5		預備金	0	0		0
	6		提撥基金	38,000	38,000		0
3			本期餘絀	-316,134	0		

理事長：

常務監事：

秘書長：

會計：

出

納：

製表：

中華民國比較病理學會

現金出納表

中華民國 88 年 1 月 1 日至 88 年 12 月 31 日

收入之部份		支出之部份	
科目名稱	金 額	科目名稱	金 額
上期結存	544,258	本期支出	439,948
本期收入	123,814	本期結存	228,124
合 計	668,072	合 計	668,072

理事長： 常務監事： 祕書長： 會計： 出納： 製表：

□

中華民國比較病理學會

財 產 目 錄

中華民國 88 年 12 月 31 日

財產編號	財產科目	名 稱	購置日	單位	數 量	金 額
001	事務器械設備	傳真機	86.9.2	台	1	11,600
002	事務器械設備	多媒體個人電腦	88.10.1	台	1	81,550

理事長：

常務監事：

秘書長：

會計：

出納：

製表：

中華民國比較病理學會

基金收支表

中華民國 88 年 1 月 1 日至 88 年 12 月 31 日止

收	入	支	出
準備基金	145,600	準備基金	0
歷年累存	107,600		
本年度提撥	38,000		
		結 餘	145,600

理事長： 常務監事： 祕書長： 會計： 出納： 製表：

中華民國比較病理學會

收支預算表

中華民國 89 年 1 月 1 日至 89 年 12 月 31 日

科 目				預 算 表	上 年 度 預 算 數	本年度與上年度 預 算 比 較 數	
款	項	目	名 稱			增 加	減 少
1			本會經費收入	571,000	600,000		29,000
	1		入會費	100,000	100,000		
	2		常年會費	145,000	145,000		
	3		贊助會費	65,000	65,000		
	4		利息收入	40,000	40,000		
	5		其他收入	221,000	250,000		29,000
	6		獎(捐)助款項	571,000	600,000		29,000
2	1		本會經費支出	68,000	92,000		24,000
		1	人事費	48,000	72,000		24,000
		2	兼職人員車馬費	20,000	20,000		
	2		其它人事費	350,000	350,000		
		1		240,000	240,000		
		2	辦公費	30,000	30,000		
		3	印刷費	50,000	50,000		
		4	旅運費	30,000	30,000		
			郵電費				
	3		公共關係費	90,000	90,000		
		1		90,000	90,000		
	4		業務費	30,000	30,000		
	5		會議會	0	0		
	6		雜費支出	33,000	38,000		5,000
3			預備金				
			提撥基金				
			本期餘絀				

理事長：

常務監事：

秘書長：

會計：

：

中華民國比較病理學會
 資產負債表
 中華民國 88 年 12 月 31 日

資 產		負債 基金 暨 餘絀	
流動資產			
華南活存	33,361		
定期存款	300,000		
固定資產		提撥基金	145,600
		累計餘絀	597,045
		本期餘絀	-316,134
雜項設備-傳真機	11,600		
多媒體電腦	81,550		
合 計	426,511	合 計	426,511

理事長: 常務監事: 秘書長: 會計: 出納: 製表:

**中華民國比較病理學會
八十九年度會員大會暨第十八次比較病理學研討會
(骨骼及軟組織腫瘤專題) 議程表**

時間：中華民國八十九年四月三十日（星期日）上午 8:30~下午 4:10

地點：國立臺灣大學農學院附設動物醫院會議廳 地址：台北市基隆路三段 153 號

主辦單位： 中華民國比較病理學會

協辦單位： 國立臺灣大學農學院附設動物醫院

國立臺灣大學獸醫學系

時	間	議	程	主	講	者
08:30- 09:00		Registration				
09:00- 10:00		會員大會		黃文哲	理事長	
10:00- 10:30		Coffee Break				
		Section 【1】				
10:30- 11:10		專題演講:骨骼腫瘤		紐約動物醫學中心	劉錫光教授	
11:10- 11:30		Case 143		紐約動物醫學中心		
11:30- 11:50		Case 144		花蓮慈濟綜合醫院		
11:50- 12:10		Case 145		國立台灣大學獸醫學系		
12:10- 13:30		Luncheon		(中華民國比較病理學會理監事聯席會議)		
		Section 【2】				
13:30- 13:50		Case 146		行政院衛生署新竹醫院		
13:50- 14:10		Case 147		國立屏東科技大學獸醫學系		
14:10- 14:30		Case 148		台北醫學院		
14:30- 14:50		Case 149		國立中興大學獸醫學院		
		Section 【3】				
14:50- 15:10		Case 150		華濟醫院		
15:10- 15:30		Case 151		台灣養豬科學研究所&台東縣家畜疾病防治所		
15:30- 15:50		Case 152		羅東聖母醫院		
15:50- 16:10		Discussion				

註：1.臺灣病理學會會員參加本次研討會可獲 4 個教育積分。

2.報名表請洽公佈單位。有意參加者請於 89 年 4 月 25 日前將報名表寄回或傳真
中華民國比較病理學會秘書處吳憲青收。電話：02-23630231 轉 2548 再轉 1402
傳真：02-23633289 地址：台北市舟山路 142 號 中華民國比較病理學會秘書處。

3.會場供應研討會講義、茶點與午餐。中華民國比較病理學會會員免費，非會員
報名費新台幣 300 元(含餐點及講義等)請於報到時繳交。

4.歡迎加入中華民國比較病理學會會員，申請入會請洽中華民國比較病理學會秘書處吳
憲青先生收。電話：02-23630231 轉 2548 再轉 1402 傳真：02-23633289 地址：台北
市舟山路 142 號 中華民國比較病理學會秘書處。

5.開車者請出示議程表給駐衛校警，可將車停於靠近動物醫院的台大校園內。

中 華 民 國 比 較 病 理 學 會
第十八次比較病理學研討會(骨骼及軟組織腫瘤專題)
病 歷 摘 要

時 間：中華民國八十九年四月三十日（星期日）上午 8:30~下午 4:10

地 點：國立臺灣大學農學院附設動物醫院會議廳

地 址：台北市基隆路三段 153 號

主辦單位： 中華民國比較病理學會

協辦單位： 國立臺灣大學農學院附設動物醫院

國立臺灣大學獸醫學系暨獸醫學研究所

CP Case 143 紐約動物醫學中心 (S489-99)

A 9-year-old, spayed female, black rottweiler dog had a fracture of the left humerus that was repaired with cerclage wires, cross pinning, and intramedullary pinning in August 1989. The left humerus collapsed and surgically repaired with a plate in November 1989. The dog developed lameness in the left humerus. Radiographs revealed lysis and osteodensity of the left humerus and the left leg was amputated in April 1996.

CP Case 144 花蓮慈濟綜合醫院 (S1996-0375A2)

This is a 53-year-old male. Giant cell tumor of tendon sheath in right distal ulnar area was firstly noted in 1972, and he had received tumor resection. In 1974, tumor recurred in right ulnar area. Tumor resection with radiotherapy (Co 60, 2500 rads) was prescribed. In 1994, tumor recurred in right ulnar again, and he had received another tumor resection with radiotherapy (Co 60, 5000 rads). After that, he was rather well. Until 1995 Oct, tumor recurrence in right radius was noted. Wide excision with distal radial allograft was performed on Oct 30, 1995. In 1996 Jan, tumor recurred over right forearm again. And multiple nodules over bilateral lungs were revealed by CXR. Amputation and thoracotomy with resection of bilateral metastatic nodules were performed. This section is taken from the lung nodule.

CP Case 145 國立臺灣大學獸醫學系 (NTU2000-107A)

A 9-year-old intact female Rottweiler canine had a one month history of left front leg lameness and was presented to National Taiwan University Animal Hospital for evaluation. The dog was alert but appeared to be in great discomfort upon physical examination. The left shoulder joint has a firm swelling mass with no pain sensation upon touch. The owner had complained the dog has lost weight for the past month and was listless especially during the night. Radiograph revealed severe Proliferative/lytic lesion near the proximal humerus of the left front leg. The owner declined further surgical and medical intervention and the dog died two months later.

CP Case 146 行政院衛生署新竹醫院 (97-2431A4)

A 73-year old male who suffered from an enlarging mass in lateral aspect of left lower thigh noted since about half a year ago. The mass grew more rapidly in recent time. No pain was complained other than occasional itching sensation. Fine needle aspiration cytology was performed showing malignant soft tissue tumor. CT scan of the lesion revealed a 10×5×4 cm tumor located in musculus vastus lateralis of left thigh with vague bosselated outer surface and central necrosis. No direct invasion of nearby peritoneum and skin could be found. Wide excision of the tumor with skin flap reconstruction was then conducted smoothly.

CP Case 147 國立屏東科技大學獸醫學系 (WA89-574)

A 10-year-old male Leopard showed anorexia, weakness and progressive loss of condition. There were multiple tumor mass on thyroid, adrenal gland, pituitary gland and heart. The specimen of heart contained tumor masses, measuring 0.1×0.3×1 cm multiple on the epicardium.

CP Case 148 台北醫學院 (96-1634)

This is a 15-year-old male with chief complaint of a non-painful swelling over the left mandible for 5 months. Oral apical X-ray presents a radiolucent lesion on the apices of #36,37 teeth measuring 3.0×1.5 cm in dimension. A small indurative lesion with reddish discoloration of the overlying smooth mucosal surface and a mild painful sensation is noted on palpation. On operation, a cystic lesion with clear yellow fluid is found and recurrence is noted after three years post operation.

CP Case 149 國立中興大學獸醫學院 (CS980910-5)

Shih-Tzu dog, adult, 5.3 Kg, showed lameness and swelled right hind leg. Fibula fracture and osteolysis were revealed in the radiograph.

CP Case 150 華濟醫院 (8805995B)

A 31-years old male who had suffered from abdominal fullness for several days visited our ER due to bulging mass over upper abdomen and painful sensation over abdominal region for 2 days. He had gotten history of traffic accident with head injury, hemopneumothorax, multiple ribs, pubic ramus fracture and received treatment in our hospital on 88-02-23 to 88-03-23. Exploratory laparotomy was performed under impression of internal bleeding, Then, emergency operation and open biopsy was performed. The tissue section was taken from abdominal cavity.

CP Case 151 台灣養豬科學研究所台東縣家畜疾病防治所 (R00-14)

An eight-month-old goat was examined because of swollen upper and lower jaws, 1-2 months after the clinical sign was observed, the animal was unable to feed. The goat was in fair nutritional state with slightly rough hair. Laboratory hematological examination showed WBC and RBC counts were within normal range, there were elevated serum alkaline phosphatase and serum phosphorus, with lower serum calcium and serum potassium. This goat farm had 170 animals, only 6-8 months growing goats were suffered, affected goats were culled due to poor nutritional state, because they finally could not open their mouth to eat.

CP Case 152 羅東聖母醫院 (000396A1)

A 68-year-old man suffered from left hip pain after a fall (twist of femur bone). Plain film X ray examination revealed a defect area with destruction (fracture) in the subtrochanter area of femur bone. The laboratory studies revealed anemia (Hb=10.2) , WBC : 8900 , GOT : 109. He received operation and cement insertion. A few bone fragment (up to 1.6x1.4x1.2 cm) were sent for pathological examination.

Comparative Pathology Case 143

Contributor: Si-kwang Liu(劉錫光), DVM, PhD;

Pig Research Institute Taiwan, ROC. The Animal Medical Center, Cornell University, College of Medicine, New York Wildlife Conservation Society, New York, USA; (美國紐約動物醫學中心; 康乃爾大學醫學院; 野生動物保育學會; 臺灣養豬科學研究所)

Clinical history: A 9-year-old, spayed female, black rottweiler dog had a fracture of the left humerus that was repaired with cerclage wires, cross pinning, and intramedullary pinning in August 1989. The left humerus collapsed and surgically repaired with a plate in November 1989. The dog developed lameness in the left humerus. Radiographs revealed lysis and osteodensity of the left humerus and the left leg was amputated in April 1996.

Diagnosis: Osteosarcoma associated with metallic implants, of the left humerus

Gross findings: The left shoulder joint was swollen, measuring 10.5 cm in diameter. The mid-shaft of the humerus had a nonunion repaired fracture. There was extensive soft tissue swelling at the proximal portion of the fracture site. All implanted metals including plate, screws, and wires were in a good condition.

Histopathological findings: Islands of closely packed malignant cells with round or ovoid nuclei were seen. A few giant cells were present throughout the lesion. The sarcomatous cells had ill-defined cytoplasm and contained some mitotic figures. Areas of hemorrhage, necrotic soft tissue, and osteoid tissue were seen, with myriad inflammatory cells, mainly neutrophils, around the periphery blending in with sarcomatous cells. Throughout the tumor were areas of osteoid and osseous trabeculae surrounded by malignant osteoblastic sarcomatous cells. Loose, fibrous, granulation tissue mixed with plasma cells, lymphocytes, neutrophils and, occasionally, some fibroblasts were observed in the areas between the metal and adjacent sclerotic bone.

Discussion: Malignant tumors reported to arise at the site of metallic implants are exceedingly rare, with only 12 human cases recorded up to 1990 and a few more cases reported recently. Osteosarcoma, Ewing's sarcoma, hemangioendothelioma, malignant fibrous histiocytoma, lymphoma and synovial sarcoma have been associated with metallic implants.

Osteosarcoma developed in 0.12% of the total 14,400 dogs and cats with metallic implants at The Animal Medical Center during the period from 1963 to 1979. The age distribution of these dogs ranged from 2.5 to 18 years (mean 7.4 years). The sex distribution indicated a predominance of male dogs, 88%. There is a high incidence of German shepherd dogs were over represented (29%); than in the German shepherd composes 14% of the total clinical population.

The interval between insertion of the implants and development of osteosarcoma varied from 11 months to 12 years (mean 4.9 years). All appliances were made from varying compositions of stainless steel.

Grossly, the gross findings included granulation tissue, ossification, and necrosis or hemorrhage around the preinstall region of the affected bone. The fracture sites were healed except that a few dogs had pseudoarthrosis. Areas of corrosion were observed on the surface of all the metallic implants with dark purple tissue surrounding the corrosive area. All gross changes of metallic implant osteosarcoma are different from those of naturally occurring osteosarcoma.

Microscopically, in all the metallic implant associated osteosarcomas were seen areas of necrotic soft tissue and necrotic bone with myriad neutrophils around the periphery blending with sarcomatous cells and neoplastic osteoid or osseous tissues.

All of the osteosarcoma and undifferentiated sarcomas associated with metallic implant were in the midshaft of the long bone, a most typical location, and were in close proximity to the corroded metallic implants. The data presented here supports implant induction of neoplasia rather than natural development.

Diagnosis criteria:

- 1) The tumor arose midshaft of the long bone associated with a metallic implants,
- 2) Grossly were soft tissue reaction including granulation tissue, ossification, necrosis, or hemorrhage around the periphery region of the affected bone,
- 3) Microscopically, were inflammatory reaction, necrosis, sarcomatous cells, and neoplastic osteoid or osseous trabeculae.

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Comparative Pathology Case 144

Contributors : Jeng-Fang Kuo(郭正芳), MD ; Yung-Hsiang Hsu(許永祥), MD.

Department of Pathology, Buddhist Tzu-Chi General Hospital, Hwa-Lien City(花蓮慈濟綜合醫院病理科)

Clinical history: A 55-year-old male with giant cell tumor of tendon sheath on right distal ulnar area was firstly noted in 1972, and he had received tumor excision. In 1974, tumor recurred in right ulnar area, the same as previous area. Tumor resection with radiotherapy (Co60, 2500 rads) was prescribed. In 1994, tumor recurred in right ulnar area again, and he had received another tumor resection with radiotherapy (Co60, 5000 rads). After that, he was rather well. Until Oct 1995, tumor recurred in the right wrist. So he came to our hospital for help. Physical examination revealed one soft tumor mass at radial side with limited range of movement. X-ray of right wrist revealed one soft tissue density tumor lesion at right distal forearm just near the wrist area. MRI revealed one large ill-defined soft tissue density at right distal forearm and wrist adjacent muscle, and with bone invasion. Wide excision with distal radial allograft was performed on Oct 30, 1995. Unfortunately, tumor recurred over right forearm in Jan, 1996. And CXR revealed multiple nodules at bilateral lungs. Then he received right elbow disarticulation and bilateral thoracotomy with resection of metastatic nodules. Three courses of chemotherapy with MAID (Meisna + Ifosfamide + Doxorubicin + Dacarbazine) were also given in Feb 27 to Mar 2, Mar 27 to 30, and Apr 26 to 30 respectively. In July 1996, patient went to VGH Taichung for recurrence of tumor in lung. No further treatment was given. Patient expired in Dec 1996.

Diagnosis: Radiation-induced osteogenic sarcoma, metastatic, lung, bilateral

Gross findings: The specimen submitted consisted of more than 10 tissue fragments measuring up to 1.5 × 1.0 × 1.0 cm size and they were grayish and elastic firm.

Histopathological findings: The tumor was hypercellular and composed of pleomorphic spindle cell pattern with frequent multinuclear giant cell formation and prominent tumor necrosis. Malignant osteoid formation in the peripheral zone accompanied hyalinized collagen deposition diagnostic of osteosarcoma was also noted.

Discussion: Most malignant bone tumors, specifically osteogenic sarcomas, arise spontaneously, but on rare occasions they may be secondary to other benign osseous conditions and they may occur following irradiation. Approximately 5% of all osteogenic sarcomas occurring in extraosseous or osseous location arise as a direct consequence of therapeutic or incidental irradiation, not only in child and adolescents, but especially in adults, such as our case.

Age at the time of irradiation may be an important host factor. The latent period of

irradiation varied from a mean of 8.7 years in children and adolescents (16 years old or younger) to 13.5 years in adult with both osseous and extraosseous osteogenic sarcoma. There are significant differences in the mean latent periods between those 16 years or younger in contrast to those who are older than 16 years. It has been suggested that the immature tissue of the children and adolescents may be more sensitive to the effect of radiation.

In the absence of appropriate control population, it is not possible to judge whether a particular radiation plan was especially likely to induce osteogenic sarcoma. However, it is evident that sarcoma can be induced in bone by a wide variety of types of exposure to ionizing radiation and that the effect may not be apparent until after a substantial fraction of the normal life span. Recent studies have suggested that the increased use of radiotherapy with improved dosage characteristics (lower energy attenuation in bone per roentgen) may diminish the frequency of new neoplasm in bone.

Microscopically, extraskeletal osteosarcomas, like osteosarcomas of bone, range from tumors that resemble fibrosarcoma and malignant fibrous histiocytoma (fibroblastic osteosarcoma) to extremely cellular tumors having an irregular round cell or pleomorphic spindle cell pattern with considerable mitotic activity (osteoblastic osteosarcoma). Usually, the osteoid is deposited in a fine, ramifying, lacelike or coarsely trabecular pattern, sometimes showing transitions toward mature-appearing bone or hyalinized collagen. Atypical cartilage of variable cellularity may be present, but rarely becomes a dominating feature (chondroblastic osteosarcoma). There is also a varying number of benign and malignant multinucleated giant cells of osteoclastic type that are often associated with hemorrhage (osteoclastic or giant cell osteosarcoma), such as our case. Occasional examples with markedly dilated vascular spaces may resemble a vascular tumor (telangiectatic osteosarcoma).

It is not always easy to distinguish extraskeletal osteosarcomas from other benign and malignant bone and cartilage forming tumor and tumor-like lesion. Among metaplastic bone of malignant tumors is infrequently found in synovial sarcoma, epithelioid sarcoma, malignant fibrous histiocytoma, liposarcoma and other mesenchymal neoplasms. In most of these neoplasms, bone is confined to a small portion of the tumor and is usually well differentiated without the disorderly pattern and cellular pleomorphism of osteosarcoma. In fact, at times it is even difficult to draw a sharp line between the fibrillary hyalinized collagen and the more homogenous osteoid. Bhagava and Dorfman emphasized the presence of bone and cartilaginous element in the pseudocapsule and fibrous septa of malignant fibrous histiocytoma and a zoning pattern similar to that found in myositis ossificans. Drawing a sharp line between extraskeletal osteosarcoma and malignant fibrous histiocytoma with osseous metaplasia is not always possible, and diagnosis may depend on the relative amount of the newly formed malignant osteoid and bone.

Radiation-induced osteosarcomas are very aggressive tumors with a marked tendency to metastasize and a dismal prognosis. The majority of patients with this tumor succumb to metastatic growth within a period of 2 or 3 years after the initial diagnosis.

The most common sites of metastases are the lung and the regional lymph nodes.

Combination therapy, radical surgery, radiotherapy and sequential chemotherapy should be carried out in the hope of improving survival rates. Patients with radiation-related osteosarcoma and resectable lesions can be cured with surgery and intensive preoperative and postoperative chemotherapy (Tabone MD, et al, 1999).

The quoted overall death rate from osteogenic sarcoma is about 0.5 per 100,000, thus the complication of postirradiation osteosarcoma is so rare that it should not discourage the use of irradiation to treat cancer.

Diagnostic criteria:

Criteria for the diagnosis of radiation-induced bone tumor were originally established by Cahan et al. and include the following :

- (1) Roentgenographic or histopathologic evidence that bone was normal prior to irradiation;
- (2) A relatively long latent period between the radiation insult to the bone and the initial evidence of the secondary tumor;
- (3) The tumor attributed to irradiation must arise within the field of previous irradiation; and
- (4) The purported radiation-induced tumor must be histologically documented and should be consistent with a primary bone neoplasm.

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Comparative Pathology Case 145

Contributors: Jihjong J. Lee (李繼忠), DVM, MS, Lih-Seng J. Yeh (葉力森), DVM, MS, PhD, Chen-Hsuan Liu (劉振軒), DVM, MS, Ph.D.

Department of Veterinary Medicine, National Taiwan University (國立臺灣大學獸醫學系)

Clinical history: 9 years old intact female Rottweiler canine had one-month history of left front leg lameness and was presented to National Taiwan University Animal Hospital for evaluation. The dog was alert but appeared to be in great discomfort upon physical examination. The left shoulder joint has a firm swelling mass with no pain sensation upon touch. The owner complained the dog had lost weight for the past month and was listless especially during the night. Radiograph of left shoulder revealed moderate to severe osteolysis and periosteal new bone formation on the left proximal humerus greater tubercle and severe periosteal proliferative/lytic lesion near the proximal humerus of the left front leg. Multiple radiopaque areas were also noted throughout the lung. On laboratory exam, the serum alkaline phosphatase (ALKP) was greatly increased (288 U/L), so did the ALT (123 U/L). *Dirofilaria immitis* ELISA test revealed positive result. The owner declined further surgical and medical interventions and the dog was euthanized two months later. Necropsy was performed.

Diagnosis: Osteosarcoma, left proximal humerus, with wide spread metastasis, canine

Gross findings: The dog is in fair to slightly emaciated body condition with distended abdomen. Approximately 400ml yellowish clear fluid is found in the abdominal cavity.

There are two large coalescing masses between left scapular and humerus measuring 10x8x10cm and 12x12x10 cm separately. On cut surface, these masses consist of grayish white firm mass with multiple cavities filled with brownish-red clear fluid and yellow friable tissue. The proximal humerus is severely obliterated by tumor mass and become irregular and misshapen.

The right lung lobes contain multiple variable sized white firm nodules ranging from 1 to 5 cm in diameter and the left middle lung lobe also contains a firm mass measuring 0.8 cm in diameter. A 1.5x0.5x0.5 cm firm white mass is located on the right ventricle free wall. One adult heartworm is found within the right ventricular chamber between the papillary chordae. Liver has variable sized firm round white nodules throughout the lobes. The largest nodule of liver is measured 19x5x2 cm. The spleen has a 4x2.2x1.5 cm white nodule. Both kidneys are slightly roughened and the left kidney has two 2.8x2 cm yellow/white discolorations (infarcts).

An ulceration/perforation lesion is located near the pylorus measuring 1.5x1 cm. Multiple button ulcers lesions are seen in the middle portion of ileum. Among them, three lesions have been perforated.

Histopathological findings: The mass of humerus consists of pleomorphic neoplastic cells arranged in sheet to bundles separated by eosinophilic island of osteoid matrix. Majority of neoplastic cells are polygonal to irregular shaped, with large oval to angular nuclei, clump chromatin, single large prominent nucleoli, scant eosinophilic cytoplasm and indistinct cell borders. Variable amount of eosinophilic matrix is observed throughout the sections. Occasionally neoplastic cells surround by basophilic matrix (condroid) and arranged in lacunae-like structure is also noted. Within the center of these masses, multiple large areas filled with eosinophilic fluid and extravagated RBC, surrounded and intermingled by homogeneous eosinophilic neoplastic tissue are commonly seen. Mitotic figures are frequently seen but varied from area to area.

The lung has multiple well demarcated neoplastic masses consist of dense sheet of pleomorphic cells with oval to elongated nuclei, stipple to clump chromatin, prominent nucleoli, and scant cytoplasm. Within the center of these masses, densely eosinophilic osteoid islands are seen. Mitotic figures are more commonly seen within the peripheral regions.

Multiple neoplastic masses are seen in the spleen. These masses consist of interwoven to streaming spindle cells with oval to elongated nuclei, clump chromatin, prominent nucleoli, and scant eosinophilic vacuolar cytoplasm. Mitotic figures are frequently seen. Islands of dense eosinophilic osteoid are occasionally seen.

Well-demarcated neoplastic mass compressing normal hepatic tissue is seen in the liver. These masses consist of dense sheet of spindle to polygonal cells with angular to irregular nuclei, clump chromatin, single prominent nucleoli separated by dense eosinophilic bundles or osteoid islands. Mitotic figures are occasionally seen.

Discussion: Osteosarcoma is the most common neoplasm arising from the canine skeletal system accounting for approximately 80% of canine primary bone tumors (1). Osteosarcoma more commonly occurs in the appendicular than in the axial skeleton (1, 2). Osteosarcoma of the axial skeleton, non-osteogenic primary bone sarcomas, bone metastases, and fracture-associated sarcoma are traditionally grouped separately because they account for the majority of skeletal neoplasms that affect the diaphysis of long bones (in contrast to the metaphyseal location of most primary bone sarcomas) (3,4,5,6). Appendicular osteosarcoma exhibits a "close to the knee, away from the elbow" site predilection, and the distal radius is the most frequently reported primary site (2,7,8). Body size appears to be an important risk factor; large and giant breeds have up to 150 times greater risk than dogs weighing less than 10 kg (9).

Most primary bone tumors arise from within the medullary cavity or cortical bone, and are termed central or medullary bone neoplasms. However, a small subset of primary bone tumors distinct from these central tumors arises from outside the cortex, presumably from the periosteum. These are termed juxtacortical, parosteal, or periosteal bone sarcomas. Although rare, multicentric osteosarcoma does occur. Survey skeletal radiography (13) or bone scintigraphy (14,15,16) may be used to assess involvement in other skeletal locations. However, the controversy of multicentric osteosarcoma or early metastasis to other bone tissue remains

unclear.

Clinical diagnosis is readily made from radiography and physical examinations, however histopathologic evaluation is still required to confirm the diagnosis. Approximately 90% of patients have metastasis at the time of diagnosis that had greatly reduced the possibility of successful management of osteosarcoma. Local tumor control is readily achievable for appendicular tumor, but metastasis is the main source of failure.

Histologically, the presence of tumor osteoid distinguishes osteosarcoma from non-osteogenic malignant bone neoplasms, such as chondrosarcoma or fibrosarcoma. Although sub classification of osteosarcoma (including osteoblastic, osteogenic, fibroblastic, chondroblastic, and telangiectatic) helps in describing the histological composition of the tumors, nevertheless these classifications have no indication in predicting the biological behavior of the tumors. Metastases of this case within different tissue exhibit different tissue differentiation. A possible microenvironment change has influenced on bone tissue, differentiation should be considered.

Radiographic changes, such as cortical destruction and periosteal new bone formation, may support a diagnosis of skeletal neoplasia but are seldom pathognomonic for a particular histopathological type (7,8,10,11,12). Radiographs of the primary lesion are important in characterizing the extent of bone involvement. Thoracic radiographs in this case had revealed positive metastatic lesion. This diagnosis of metastatic disease however, is jeopardized by the possibility of pulmonary thromboembolism resulted from the heartworm infestation.

Older dogs are most frequently affected, although a biphasic peak incidence (2 years and 9 years) has been reported (1). In most cases, one bone lesion is detectable, although multiple bone involvement has been reported (13, 17, 18). Appendicular osteosarcoma in dogs has a high propensity to metastasize. Although thoracic radiographs show pulmonary metastases in only 5%-10% of dogs at initial presentation (12, 13), tumor metastasis has often taken place. As a result, the likelihood for cure after limb amputation is low. In two large retrospective studies, median survival after limb amputation was 18 to 19 weeks (19,20)

Limb amputation, with or without adjuvant therapy, is the standard treatment for tumors of the appendicular skeleton. This option in our case unfortunately has been overruled by the presence of advanced metastatic disease and poor prognosis from hefty body weight. No prospective, double-blind, randomized clinical trial has evaluated the efficacy of adjuvant cytotoxic chemotherapy in the treatment of appendicular osteosarcoma. However, adjuvant chemotherapy is believed to prolong survival. Limb salvage, consisting of *en bloc* tumor excision, cortical allografting, and bone plate application combined with preoperative radiation therapy, intravenous or intra-arterial cisplatin, has been used to treat dogs with appendicular osteosarcoma (20). However, there is no evidence that dogs treated with limb salvage survive longer than those treated with limb amputation and adjuvant chemotherapy.

Surgical resection has been used for the treatment of pulmonary metastases in humans with osteosarcoma (21,22,23,24), which is rarely performed in metastatic canine osteosarcoma patient.

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Comparative Pathology Case 146

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Clinical history: This 73-year-old male patient was a victim of soft tissue tumor in inner aspect of left lower thigh. The tumor was noted 6 months before the admission. It got faster growth at later time before seeking help. No pain was complained, except that of itching sensation. Sonography was conducted at OPD with fine needle aspiration examination made. Sarcoma was diagnosed and he was therefore put under surgery with wide excision.

Diagnosis: Pleomorphic rhabdomyosarcoma

Gross findings: A soft, fleshy tumor, 10 x 5 x 4 cm, with relatively defined margins, located within the skeletal muscle of vastus lateralis without invasion to its nearby periosteum and skin. Focal hemorrhage and necrosis are present revealing as heterogeneous texture.

Histopathological findings: The tumor is heterogeneous in morphology ranging from densely cellular to myxoid area with low cellularity. It is composed principally of a patternless array of large, bizarre, polygonal or spindle-shaped tumor cells, set in a variably amount of collagenous or myxoid stroma. The tumor cells are composed of loosely arranged, haphazardly oriented, large, round or pleomorphic cells with hyperchromatic nuclei and deeply eosinophilic cytoplasm. Strap or tadpole cells, cells in racquet shapes, and spider-web cells are prominent. Some tumors show predominantly storiform and approach closely the appearance of pleomorphic MFH. Cross-striations are virtually never found. Hemorrhage and necrosis is marked. The tumor involves the skeletal muscle and has infiltrative margins.

Discussion: Rhabdomyosarcoma (RMS) is the most common soft-tissue tumor in children under the age of 15. Nearly half of these cases are diagnosed in children 5 years of age or younger. In contrast, it is a rare tumor for adults. Three histotypes of RMS have been recognized: alveolar type (and its botryoid variant), embryonal type and pleomorphic type. Almost all the cases diagnosed as RMS in adults are pleomorphic type. The concept of RMS arising in adults have undergone considerable revision over the past fifty years. The initial diagnosed cases of RMS were mostly reclassified as malignant fibrous histiocytoma (MFH) after the entity was established. The advent of new means of determining rhabdomyoblastic differentiation, including immunohistochemical stainings and ultrastructural evidences, has reestablished and confirmed the entity of pleomorphic RMS in adults. As in recent series of studies, pleomorphic RMS represented about 7% of all pleomorphic sarcomas. It usually shows male predominance. The majority of cases have arisen in adults over 30 years of age. The mean age of presentation

ranges from 44 to 58 years. It almost never exists in children. Childhood cases almost always have the presence of at least small foci of embryonal or (less often) alveolar tumor in multisections. The term “anaplastic” is preferred for such tumors. Most of the cases arose within the skeletal musculature of the limbs, particularly the thigh. The other main sites affected are the musculature of chest wall, retroperitoneum, and neck. The major presenting symptom is that of a rapidly enlarging, usually painless, tumor of several months duration. A minority of cases have been present for several years. Some patients have already developed metastases at the time of primary presentation, most commonly in the lung.

The presence of sarcomeres with Z bands, actin, and myosin filaments that form hexagonal arrays on cross sections, as well as thick filaments lined by ribosomes are all diagnostic of this neoplasm. Pleomorphic RMS displays a considerable variety of morphologic features, but all cases are characterized, to a greater or lesser extent, by the presence of large, pleomorphic, eosinophilic cells related to the putative rhabdomyoblasts. The atypical cells are in different forms and are described as strap-cells, tadpole cells, racquet shape or so-called spider-web rhabdomyoblasts. Cells with cross-striations are commonly found in embryonal RMS, but are virtually never found in adult pleomorphic RMS. Muscle differentiation is demonstrable by the presence of a number of proteins, including desmin (muscle-specific intermediate filament), HHF-35 (muscular isoforms of alpha and gamma actin), hemprotein and myoglobin (sarcomeric differentiation), MyoD1 and other myogenic proteins. The expression of MyoD1 gene has been remarkably specific for skeletal muscle differentiation and has clearly been shown in juvenile RMS, including desmin-negative cases. Pleomorphic RMS has similar result. Some pleomorphic RMS may express alpha SMA, which is normally restricted to smooth muscle and myofibroblasts. Other minor skeletal-muscle specific antigens include titin, Z protein, tropomyosin, alpha actinin and skeletal muscle isoforms of creatine kinase.

The genetic changes in RMS have been found with cytogenetic techniques and other molecular methods. Both classical karyotyping and fluorescence in situ hybridization have shown that the alveolar type (ARMS) and embryonal type (ERMS) of RMS is associated with specific molecular changes: a reproducible tumor-specific chromosome translocation, t(2;13)(q35;q14), causing PAX3/PAX7-FKHR gene fusions in alveolar type and 11p15.5 allelic loss in embryonal type. The gene fusion in ARMS represents gain-of-function oncogenic mutations that generate potent transcriptional activators of target genes with PAX3/PAX7 binding sites. In contrast, the 11p15 allelic loss in ERMS acts on an imprinted region to inactivate expression of putative tumor suppressor loci. In addition to these primary mutations, genetic alterations of other oncogenes and tumor suppressor genes occur in both tumors, and indicate that these tumors arise by a multistep process as in most tumors. Both the primary and secondary events affect gene products, which function in signal transduction and gene expression regulatory pathways. These events lead to more aberrant molecular changes and alter numerous key pathways in the cells, and ultimately generate the phenotypic changes of growth autonomy, invasion and metastasis.

Common sites of metastases for ARMS and ERMS include regional lymph nodes, the lungs,

bone, and bone marrow. The small number of pleomorphic RMS precludes definitive clinical pattern. Nonetheless, it seems that it is a highly aggressive neoplasm that grows quickly and metastasizes early. Treatment includes total resection, combined with chemotherapy and radiotherapy in higher stage cases. New strategy is being developing utilizing the tumor-specific molecular targets such as the PAX3-FKHR oncogene neoprotein.

Diagnostic criteria:

1. Pleomorphic sarcoma with characteristic cells in strap, tadpole, racket or spider-web forms.
2. Immunohistochemical evidence of the presence of muscle proteins (such as MyoD, desmin, myoglobin, or muscle-specific actins)
3. Ultrastructural evidence of myofilaments.

Differential diagnosis:

1. Malignant fibrous histiocytoma
2. Pleomorphic leiomyosarcoma
3. Pleomorphic liposarcoma
4. Malignant mesenchymoma
5. Other neoplasms with presence of rhabdomyoblastic differentiation, such as malignant mixed Mullerian tumor, malignant Triton tumor and carcinosarcoma.

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Comparative Pathology Case 147

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Clinical history: A ten-year-old male Leopard showed anorexia, weakness and progressive loss of condition.

Diagnosis: Papillary mesothelioma of pericardium, Leopard.

Gross findings: At Necropsy, there were multiple tumor masses on the thyroid, adrenal gland, pituitary gland and heart. The pericardial sac was mildly distended with 100 ml of light tan fluid. Multiple light to red nodules and irregular flat plaques (0.1 cm to 1 cm in diameter) were attached to the epicardium and visceral surface of the pericardium through the heart. The masses confluent with the folded and bronched structures on some areas. All the tumor masses did not penetrate the heart.

Histopathological findings: There were multiple well vascularized connective tissue with variable arborescence attached to the epicardium, over which there was a single layer of cuboidal or flattened epithelium. There were papillary projections and free-floating bodies on the surface. In some areas, these cells had large pleomorphic nuclei and abundant cytoplasm. A locally extensive area showed necrosis and hemorrhage. Multinucleated cells were common, but mitotic figures were rare.

Discussion: Mesotheliomas, rare in domestic animals and human, may arise from any of the mesothelial coverings, such as pleura, peritoneum or pericardial sac. Mesotheliomas typically may be multiple and involve both visceral and parietal serosal surface. Either ascites or hydrothorax commonly has been reported when the peritoneum or pleura had mesothelioma. There is a strong association between pleural mesothelioma and asbestosis in man. Whereas in cattle, peritoneal mesotheliomas often are congenital and young but most other animals are adult or aged. In other species, however, spontaneous mesotheliomas have been reported in mature animals without any associated causative or epizootiological factors. In this case, we not only found mesothelioma of pericardium but also multiple endocrine neoplasia (MEN) syndromes. In domestic animals and man, pericardial mesotheliomas are considered rare. In domestic animals, most mesotheliomas originate from the mesothelial lining cells of pleura and peritoneum.

The tumor has occurred naturally in man, cattle, horse, dog, rat, cat and pig. There is no breed or sex prevalence in any species, Mesotheliomas must be differentiated from granulomatous disease of serous surface, metastatic carcinoma of the peritoneum or pleura which shares similarities with mesotheliomas in gross and microscopic features. It is necessary to use immunohistochemistry and ultrastructural examination to diagnose mesothelioma. In this case, positive cytokeratin stain and negative CEA stain were observed.

Diagnostic criteria: Uniform, papillary pattern. The papillae are branching and are covered by a single layer of uniform mesothelial cells.

1. Immunohistochemical markers : cytokeratin and Vimentin are positive,
2. E. M: Mesothelial cells with long surface microvilli.

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Comparative Pathology Case 148

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Clinical history: This is the case of a 16 year-old male patient with a chief complaint of a painless swelling on the left cheek, buccal side. Within the last two months, there was a rapid growth in the size of the mass, thus was referred by his local dentist to our dental department for further examination and management.

Dental and laboratory examinations revealed a swelling on the buccal mucosa with superficial redness, otherwise normal. Teeth #34, #35, #36, #37 near the swelling are all vital and without pain or tenderness on percussion.

Radiological examination revealed a radiolucent lesion about 1.2 x 1 cm on the roots of teeth #36, #37. With a clinical impression of radicular cyst, enucleation was done and clear fluid flowed out of the lesion, specimen number 96-1634.

Regular follow-up was uneventful until three years later, when x-rays revealed an enlargement of the lesion with seeming destruction of the cortical bone and invasion of the soft tissues, thus a biopsy was done with subsequent surgical removal of the lesion.

Diagnosis: Cystic ameloblastoma

Gross findings: There is an empty cystic lesion measuring 3.5 x 2.5 x 2.0 cm in size within the resected specimen (including ramus and body of the left mandible).

On cut, some brownish soft tissue coating on the cystic lesion is found. The bilateral cortical bones are thinned and focal destruction is noted in the lateral buccal wall. An unerupted tooth near the cystic lesion is also found. No obvious solid or polypoid tumor is found in the cystic space. The cystic lesion is located 2 cm and 0.3 cm away from the proximal and distal surgical margins respectively.

Histopathological findings: The tumor shows a picture of cystic ameloblastoma composed of an unilocular cystic lesion surrounded by a fibrous wall and lined by squamous cells or ameloblast-like cells. Focal cystic degeneration in the stellate reticulum in the small intraluminal nodule is found. The tumor's expansion is associated with bone destruction, but the neoplastic lesion is still surrounded by a fibrous wall. Chronic inflammation with cholesterol granuloma and granulation tissue formation in the nearby fibrous tissues is also seen.

Differential diagnosis³:

1. Radicular cyst. This common non-neoplastic lesion often shows quite extensive proliferation of its epithelial lining and forms a similar picture of plexiform ameloblastoma. A monocystic ameloblastoma with flattened tumor epithelium could be mistaken for non-neoplastic squamous epithelium.
2. Odontogenic keratocyst. Degenerative changes in the squamous epithelial lining due to inflammation produce a picture resembling the stellate cells of ameloblastoma.
3. Dentigerous cyst. Islets of squamous epithelium in the cyst wall with central cyst formation is similar to ameloblastoma follicle.
4. Calcifying odontogenic cysts. Frequently has a mantle of ameloblast-like cells but is distinguished by its characteristic ghost cell.

We can use the presentations of PCNA (proliferating cell nuclear antigen)⁶ and Ki-67 antigen staining to demonstrate that cystic ameloblastoma is a kind of cystic neoplasm and to understand that the invasiveness of the mural type is greater than the luminal and the intraluminal plexiform type.

We can also make use of the different results of lectin positive bindings⁷, *Ulex europaeus* agglutinin I (UEA-I), *Bandeirea simplicifolia* agglutinin I (BSA-I) and peanut agglutinin (PNA) in ameloblastoma and jaw cysts to differentiate cystic ameloblast and non-neoplastic jaw cysts.

Diagnostic criteria:

A clearly defined cyst with a fibrous wall and lined by total or partial ameloblastic cells

Discussion: Ameloblastoma is a tumor of odontogenic epithelial origin¹. It may arise from cell rests of the enamel organ, from a developing enamel organ, from the epithelial lining of an odontogenic cyst, or from the basal cells of the oral mucosa. Ameloblastomas are slow-growing, locally invasive tumors that run a benign course in most cases.

Because of different clinicoradiologic situations, different therapeutic considerations and prognosis, ameloblastomas are classified¹ as:

1. Conventional solid or multicystic (86%)
2. Unicystic (13%)
3. Peripheral (extraosseous) (1%)

	solid or multicystic	unicystic	peripheral (extraosseous)
1. age	3 rd to 7 th decade	2 nd decade	middle age
2. S/Sx	painless swelling, often asymptomatic	painless swelling, often asymptomatic	painless, non-ulcerated, sessile or pedunculated
3. location	mandible (85%) maxilla (15%) molar-ascending ramus	mandible (90%) posterior region	mandible posterior gingival and alveolar mucosa
4. x-ray	soap bubble, honey-combed	defined radiolucent, common with tooth	sometimes with alveolar bone erosion
5. histologic	1. common: follicular or plexiform 2. others: acanthomatous, granular cell, desmoplastic, basaloid	1. intraluminal ⁴ 2. plexiform unicystic 3. mural	similar to intraosseous forms
6. treatment	1. simple enucleation ⁵ 2. curettage 3. en bloc resection	1. nucleation 2. local resection	local excision
7. recurrence rate ²	17 - 23%	10 - 13%	9%

As seen in the above table, enucleation or local resection of a cystic ameloblastoma is adequate management, which was done in this patient. However, after three years, tumor recurrence was noted and on CT scan, the tumor within the body of the mandible and ramus presents with a seemingly solid pattern with outward growth and perforation through the cortical bone up to the masseter muscle and inwards up to the pterygoid space and possibly invading the surrounding soft tissues, thus making the previous enucleation seemingly insufficient. Therefore, after a second biopsy with confirmation of ameloblastoma, a bigger area of surgical resection (segmental mandibulectomy) was done. Subsequent pathological findings of the specimen was still cystic ameloblastoma and the CT finding of cortical bone perforation was actually due to the first surgical treatment of enucleation and biopsy of specimen resulting in a bone window that is still present after a period of time. In September last year, the patient underwent bone grafting and titanium mesh plate reconstruction surgery and has recovered satisfactorily.

Furthermore, ameloblastomas can also metastasize. Although there isn't any significant difference in the histologic morphology of metastatic ameloblastoma with that of ameloblastoma, the mere fact of it being metastatic is then termed as malignant ameloblastoma. Another type is ameloblastic carcinoma wherein cytologic features are that of ameloblastoma with the morphologic changes of malignant tumors and metastasis may follow.

With malignant ameloblastoma, hypercalcemia may be noted in a patient since the tumor releases a parathyroid-hormone-like peptide, possibly prostaglandin E₂, that results bone destruction and resorption. Hypercalcemia can be severe enough to cause renal calcinosis and subsequently death⁸.

However, the occurrence of either malignant ameloblastoma or ameloblastic carcinoma is very rare.

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Comparative Pathology Case 149

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Clinical history: Dog, Shih-Tzu, adult, 5.3 Kg, was discarded. She showed lameness and marked enlargement of the right hind leg. Fracture in the femur and osteolysis in the fibula were showed in the radiograph. The right hind leg was amputated and dissected for pathological examination.

Diagnosis: Giant cell tumor of bone

Gross findings: The subcutaneous tissue swelled and edematous, some tissues was replaced by fibrous connective tissue to show the white strand branch, the fibula fractured completely and the femur showed an expansile and osteolytic lesion in the lower epiphysis portion. Many tumor masses were found in the lung parenchyma.

Histological findings: The most obvious hallmark was many multinucleated giant cells scattered in the moderately fibrous stroma, in which, a lot of large, dark-staining, ovoid nuclei tumor cells were appeared. Osteoid material and bone spicules were observed in some area.

Discussion: Giant cell tumor of bone is an exceedingly rare primary bone tumor of animals, with most cases being reported in dogs and cats. It is also a fairly common in human, usually affects young adults over the age of 20 years. The tumor cells appears to arise from primitive stromal elements of the bone marrow and produce expansive osteolytic lesions primarily in the ends of long bones of the appendicular skeleton, although vertebral, costal, and metatarsal lesions are also found. In man, it is most commonly located in the epiphysis of long bones, particularly around the knee and the spine, with a typical X-ray appearance of an expansive, lytic lesion. Because the giant cells have the same enzyme histochemical behavior as osteoclasts, these tumors are sometimes called osteoclastoma. In some case, the tumor cells undergo metaplasia to form collagenous matrix and bone.

Diagnostic criteria:

1. Multinucleated giant cells
2. Osteoid material or bone spicules
3. Moderately fibrous stroma
4. Expansive, lytic lesion in the appendicular skeleton

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Comparative Pathology Case 150

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Clinical history: A 31-year old male who had suffered from abdominal fullness for several days visited our ER due to bulging mass over upper abdomen and painful sensation over abdominal region for 2 days. He had gotten history of traffic accident with head injury, hemopneumothorax, multiple ribs, pubic ramus fracture and received treatment in our hospital on 88-02-23 to 88-03-23. Exploratory laparotomy was performed under impression of internal bleeding. Then, emergency operation and open biopsy was performed. The tissue section was taken from abdominal cavity.

Diagnosis: Desmoplastic small round cell tumor (DSRCT)

Gross findings: The tumors consist of multiple nodules, presenting with a major tumor mass accompanied by nodules spread over the surrounding serosal surface. The surface of the resected tumor specimen is generally smooth and often bosselated. The tumor nodules are firm with a grayish to white cut surface.

Histopathological findings: The tumor is composed of sharply demarcated nests of small tumor cells surrounded by a large amount of abundant desmoplastic stroma. The size and shape of tumor cell islands vary from tiny clusters to large nests or strands. The shape of tumor cells is generally round, with round to oval central hyperchromatic, sometimes vesicular nuclei and inconspicuous nucleoli. The cytoplasm is sparse. Mitotic rate is up to 4-10/10 HPF. The collagen-rich stroma contains spindle cells resembling fibroblasts or myofibroblasts. These cells are arranged parallel to the border of the tumor cells nests, surrounding the individual tumor cells nests. Immunohistochemically, the tumor cells are positive for EMA, cytokeratin, NSE, and desmin while negative for S-100.

Discussion: DSRCT is first described by Gerald and Rosai in 1989¹. Many cases report about this distinct tumor had been published since then. This tumor is characterized by aggressive, wide spread growth along serosal surface, typical histological features, and a specific immunohistochemical staining pattern. Till 1996, about 100 cases had been recognized and reported⁵.

This age and gender distribution of our cases are quite accordance with other reports. This tumor has a predilection for adolescents and young adults, mean age between 18-20 years (median age in well-documented cases is 16 years) range from 3-to 48 years old. It is more often in male than female (male-female ratio is about 4/1)⁵. The symptom is chiefly presented with abdominal mass

and pain. The involved area is most in peritoneum and serosal surface of the organs in the abdominal cavity, such as intestine, stomach and liver. Pleural⁶, paratesticular⁷ and even intracranial⁸ involvement are also reported. All the tumors in presenting cases were found in the peritoneal cavity, similar to previous reported cases, visceral organs involvement were apparent.

The size of tumors vary from 0.1 cm to 40 cm and even weight 2kg. The surface of the resected tumor specimen is generally smooth and often bosselated. The tumor nodules are firm with a grayish to white cut surface. Hemorrhages and focal necrosis are often present.

Histologically, the tumor reveals a highly characteristic and almost diagnostic pattern even at a low magnification. At low magnification, the tumor is composed of sharply demarcated nests of small tumor cells surrounded by a large amount of abundant desmoplastic stroma.

Immunohistochemically, the tumor cells are reactive intensely for cytokeratin and NSE stain. Stain for desmin, is very essential for the differential diagnosis. Other positive staining of antibodies tested in the literature are S-100 (11/60), Leu-7 (14/30), chromogranin (4/64), synaptophysin (5/29), muscle specific actin (4/64), smooth muscle actin (3/18), GFAP (2/32), neurofilament (2/35), CEA (0/9) and leukocyte common antigen (0/12).¹ Although we had not carried out all the advocated antibodies, the essential positive staining of cytokeratin and desmin in combination with H&E findings, the diagnosis of DSRCT is out of question.

Histogenesis is still unclear. There are three hypotheses in the literature. 1. Neuroectodermal origin. 2. Mesothelial origin and 3. Combination of neural differentiation of Ewing's tumor and the multidirectional but predominantly epithelial differentiation of Wilm's tumor⁹. The later seems reasonable since this tumor has a specific cytogenic finding⁵. In almost all cases the chromosomal abnormality was present (11; 22) (p12; q11 or q12). Involvement of 22q 12 was also reported. Interesting, the region affected in DSRCT on chromosome 22 is very close (or identical) to the Ewing's sarcoma gene (EWS) and the break point on chromosome 11 is in the locus of the Wilm's' tumor suppressor gene (11q 13, WT1)⁵.

The differential diagnosis of DSRCT⁵ includes rhabdomyosarcoma, Ewing's tumor, rhabdoid tumor, malignant neuroendocrine tumor, monophasic synovial sarcoma, blastemic Wilms' tumor, neuroblastoma, germinoma, and non-Hodgkin's lymphoma and other small round and blue cell tumors of childhood and adolescence. To differentiate DSRCT from these tumors is not difficult when combined with the clinical information (age and site) and its characteristic histological and immunohistochemical features.

The prognosis of patient with a DSRCT is very poor. Most cases reported in the literature ran an aggressive course. In some patients, chemotherapy might improve the prognosis or prolong survival.

Diagnosis criteria:

1. Sharply demarcated nests of small tumor cells surrounded by a large amount of abundant desmoplastic stroma.
2. Positive immunohistochemical stains for desmin, NSE, cytokeratin and vimentin.

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Comparative Pathology Case 151

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Clinical history: An eight-month-old goat was examined because of swollen upper and lower jaw, 1~2 months after the clinical sign was observed, the animal was unable to feed. The goat was in fair nutritional state with slightly rough hair. Laboratory hematological examination showed WBC and RBC counts were within normal range, there were elevated serum alkaline phosphatase and serum phosphorus, with lower serum calcium and serum potassium. This goat farm had 170 animals, only 6~8 months growing goats were suffered, affected goats were culled due to poor nutritional state, because they finally could not open their mouth to eat.

Diagnosis: Osteodystrophia fibrosa, mandible, goat.

Gross findings: The carcass was emaciated but showed no abnormal nasal discharge. The horizontal rami of the mandible showed bilateral swelling starting at the level of the first premolars and extending to the base of the vertical ramus. The bone was soft, rubbery and uniform and could be cut with a knife. Post mortem radiography showed a relatively uniform radiolucency of this area. The cheek teeth were loosened and tilted and there was extensive ulceration of the gums and cheeks lateral to the teeth. The remainder of the carcass, including the parathyroids, showed moderate swelling.

Histopathological findings: The mandibular lesion in the cases consisted of an irregular loose fibrous connective tissue matrix surrounding numbers of bony trabeculae, many of which were partially or completely lacking in mineralisation. Considerable osteoclastic activity was seen associated with the mineralised portions of the trabeculae but appeared to be generally absent in unmineralised areas. The picture was suggestive of the formation of unmineralised trabeculae and the resorption of mineralised ones.

Laboratory results: Biochemical findings showed raised alkaline phosphatase and creatine phosphokinase levels with low calcium and potassium levels.

Discussion: Bone deformities caused by relative or absolute vitamin and mineral deficiencies are well recognised in domestic animals. Skeletal conditions include osteoporosis, ricket, osteomalacia and osteodystrophia fibrosa. The last is unusual in that, apart from being a

nutritional disease, it can arise following primary or secondary hyperparathyroidism (Jubb and Kennedy 1970). Hypocalcemia results in increased parathyroid activity and secondary parathyroidism in animals can thus occur where there is nutritional deficiency and also chronic renal disease (Smith and others 1974). The disease is seen in several non-ruminant species including the pig, dog, cat and in the horse and its relatives, where it is often called “bran disease” or “big head”. In ruminants it is most commonly observed in the goat and occasionally in cattle. Information concerning the condition in sheep is minimal.

In view of the increasing public interest in goat ownership the following report is presented to alert clinicians to the potential problem in this species. Additionally, one of these cases illustrates the fact that problems can occur even when the animals are reared by owners with agricultural knowledge, on an apparently adequate diet.

As indicated, the cause and pathogenesis of this startling disorder result from hyperparathyroidism, which can arise for several different reasons.

Primary hyperparathyroidism Primary hyperparathyroidism is usually the result of a functioning parathyroid adenoma, which is rare in animals. Parathyroid adenomas have been reported in horses, cattle, and dogs. In primary hyperparathyroidism, hypercalcemia occurs, as well as hypophosphatemia (renal loss) and marked elevation of serum alkaline phosphatase. Metastatic calcification of soft tissues is a consistent finding, and renal mineralization can lead to increase in serum phosphorus in animals with endstage kidneys.

Secondary hyperparathyroidism Secondary hyperparathyroidism is without question the most common cause of fibrous osteodystrophy in animals. Hypocalcemia, regardless of cause, is the stimulus for the increased activity of the parathyroid glands. In animals, secondary hyperparathyroidism occurs in nutritional deficiencies (nutritional secondary hyperparathyroidism) and in chronic renal disease. The lesions are similar to primary hyperparathyroidism, with added features of osteomalacia.

Renal secondary hyperparathyroidism Renal secondary hyperparathyroidism is most common in dogs, in which the disorder has been termed **renal rickets** or “rubber-jaw”. Inability to excrete phosphates causes these ions to accumulate in the blood and leads to lowering of the serum calcium concentration. The damaged kidney and the inhibitory effects of the active metabolite of vitamin D; the intestinal absorption of calcium subsequently decreases. Hyperphosphatemia in response to hypocalcemia causes marked resorption of bone, and hypocalcemia contributes to defective mineralization of osteoid (osteomalacia). Hyperphosphatemia is present throughout the course of the disease; serum alkaline phosphatase is also elevated. Unlike primary hyperparathyroidism—where serum calcium is elevated—serum calcium is usually low in renal secondary hyperparathyroidism. In dogs, the bones of the head undergo pronounced softening, enlargement, and radiographically detectable rarefaction; the jaws become “rubbery.” Microscopically, the lesions are those of fibrous osteodystrophy and osteomalacia; resorption of bone occurs by osteoclastic resorption and fibrous replacement; and woven-bone trabeculae that fail to mineralize properly are excessively produced. Since osteoid retards osteoclastic activity on mineralized surfaces and unmineralized surfaces, and

unmineralized bone tends to accumulate. All bones are affected to varying degrees, but lesions are most striking in the facial bones and mandible. Metastatic calcification of soft tissues is a regular feature. All of the parathyroids are grossly enlarged. Tests for renal function are indicative of severe renal insufficiency.

Nutritional secondary hyperparathyroidism Nutritional secondary hyperparathyroidism has been described in most domestic animals as well as many exotic species. The usual nutritional imbalances associated with the development of fibrous osteodystrophy are deficiency of calcium or vitamin D, or excess of phosphorus. As indicated under rickets and osteomalacia, secondary parathyroid hyperplasia may develop, causing these diseases to progress to fibrous osteodystrophy.

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Comparative Pathology Case 152

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Clinical history: A 68-year-old man, suffered from L't hip pain after he had slide down injury. Physical examination revealed marked swelling & deformity over L't hip and x-ray revealed bone defect & fracture over subtrochanter area of L't femur, so he received open reduction and internal fixation and cement insertion. The specimen was sent for pathologic examination & measured 1.6 x 1.4 x 1.2cm. Review of past history showed a DM case with poor blood sugar control & also a hepatocellular carcinoma case (status post lobectomy, chemotherapy & TAE), & followed up in NTUH. The laboratory data were: Glucose : 164~268, GOT : 109, WBC : 8900, Hb : 10.2, Alb : 3.1, HBsAg : +, Anti-HBe : +, Anti HBs : -, AFP : 9.85. Abdominal sonography showed hepatocellular carcinoma with ascites. The operation wound healed and the patient followed up in OPD.

Diagnosis: Bone, femur, L't, biopsy, hepatocellular carcinoma, metastatic.

Histopathological findings: Sections from the bone fragments showed nests of carcinoma involving the entire specimen & almost no bone tissue is found. The tumor was characterized by trabeculae and solid nests of neoplastic hepatocytes with bile canaliculi to be found. Extensive necrosis is also noted.

Immunohistochemistry results:

1. HepBSAg : (+)
2. AFP : (+)
3. Keratin(Cam 5.2) : (+)

Differential diagnosis:

1. Renal cell carcinoma : non-clear cell type RCC may show similar cytoarchitectural patterns & CK profile as HCC. Clues to correct diagnosis: prominent vascular pattern may be present & IHC includes: -p- CEA (canalicular), Hep Par-1(Hepatocyte paraffin-1) & AFP etc.
2. Prostate adenocarcinoma: can have sheet-like growth pattern, large cells with abundant cytoplasm, round nuclei with prominent nucleoli. Clues to correct diagnosis: Cytoplasm more basophilic, glands usually noted with carefully searching; cytoplasmic mucin may be present. IHC includes: +PSA, PSAP; -P-CEA, AFP, Hep Par-1 etc.
3. Angiosarcoma: Thickened cords of hepatocytes, +CD34, in endothelial cells lining sinusoids of HCC. CK can be positive. Clues to correct diagnosis: Hepatocytes are cytologically bland; endothelial cells are prominent & pleomorphic. In HCC, endothelial cells are inconspicuous.

ICH : +CD34 in pleomorphic endothelial cells.

4. Other sarcoma: Bland to pleomorphic spindle cells \pm differentiated sarcoma elements can be present in HCC(Sarcomatoid variant, carcinosarcoma). Clues to correct diagnosis: For diagnosis of HCC, need to demonstrate differentiated foci. IHC: +CK & AFP would favor HCC, otherwise not helpful.

Diagnostic criteria:

1. Main growth pattern as trabecular
2. Minimal fibrous stroma
3. Common intranuclear inclusions
4. Often with abundant eosinophilic, granular, clear cytoplasm
5. Occasionally present bile (less than one-third, 5-33%, pathognomonic of HCC), Cu & Cu-binding protein(7-41%), Hyaline globules(10-15%), Mallory bodies(2-25%).
6. The presence of bile canaliculi is diagnostic.
7. Absent mucin : (100%)
8. Often present : p-CEA (canalicular pattern)(70-80%), Hep Par-1, AFP,
9. Rarely present: m-CEA (noncanalicular)
10. IHC : CK:positive for AE3 & Cam 5.2, Negative for AE1

Criteria for fine needle aspiration (FNA):

A. Bottle : 97% sensitivity, 100% specificity for HCC

1. polygonal cells with centrally placed nuclei.
2. malignant cells separated by sinusoidal endothelial cells
3. biles

B: Cohan : 100% sensitivity & 87% specificity for HCC

1. an increased nuclear to cytoplasmic ratio
2. a trabecular pattern
3. atypical naked nuclei

Discussion: The incidence of metastatic cancer (MC) exceeds that of primary malignant tumor (MT) of the bone. The spread of MT to the bone marrow is typically hematogenous. It tends to involve older adults & is localized in the axial skeleton (70%). In the appendicular skeleton, metastasis are almost proximal to the elbows & knees. In most cases, the primary site (PS) is identifiable (PE & Lab) & a biopsy may be done only to establish the diagnosis of MC in the bone. Fine needle aspirates are ideal for confirming the diagnosis of MC. A small percentage of MC have no apparent PS, but the histologic diagnosis of MC is not difficult. Carcinomas are the most common type. Cancers of the breast, lung & prostate account for 80% of MC. Sarcomas rarely metastasize to bone. MC in the bone often induce conspicuous reactive fibrosis (esp. breast, stomach). A variety of secondary abnormalities due to MC in the bone includes: (1) increased bone destruction(osteolytic) : most tumor, esp thyroid, kidney & lung

(2) New bone formation (osteoblastic): breast, prostate, carcinoid

(3) Reactive fibrosis,

(4) Abnormalities in hemopoiesis: hyperplasia of megakaryocytes. A normocytic, normochromic anemia of chronic disease is common & the presence of teardrop-shaped RBC suggests bone metastasis (BM). An elevated serum level of alkaline phosphatase (Alk-P) is usual in any osteoblastic BM. The identification of specific bone & liver Alk-P isoenzymes is required to distinguish BM from those in the liver. Any tumor metastasis to bone, if extensive enough, may lead to hypercalcemia & elevation of serum acid phosphatase. It is imperative that presumed MC be biopsied in order to avoid treatment designed for primary malignant bone tumor. The microscopic recognition usually is simple. The sources of the MC may be suggested microscopically, particularly in cases of carcinoma of the kidney, thyroid or colon. Most cases of metastatic squamous cell carcinoma originate from the lung. Some unusual primary malignant lesions such as a hepatocellular carcinoma or a cylindroma of the salivary glands are so characteristic that one can confidently predict the PS. However, when the lesion is an undifferentiated adenocarcinoma or squamous cell carcinoma, one can only suggest possible PS. The identification of a possible PS in the patient with a MC of unknown primary tumor is of more than academic interest. Patient with tumors such as breast Ca & prostate Ca may survive a long time after BM, whereas patients with tumors such as lung cancer succumb quickly.

Differentiation of HCC & other adenocarcinoma (Ad):

Metastatic Ad & HCC usually immunostain in a diffuse cytoplasmic pattern for CEA, EMA & Leu M1. As only CEA, of these 3 antigens, has been localized to CEA, in a distinctive canalicular pattern (Ganji, 1998), this battery of stains can be useful in distinguishing HCC from other Ad.

Immunohistochemical Stains in adenocarcinoma

Organ	CEA	EMA	Leu M1
①Liver	+/	+/	—
②Kidney	+/—	++	—
③Prostate	+/	+	—
④Breast, lung, stomach	++	++	+/
⑤thyroid	—	++	+/

Bile located within neoplastic cells or tubular lumina is pathognomonic of HCC, but it is found in less than 1/3 of cases & is not evidence in poorly differentiated (PD) tumor. The presence of bile canaliculi is also diagnostic. Other than HCC, bile & bile canaliculi have been identified only in hepatoid Ad of varied PS. Using unabsorbed polyclonal anti-CEA antiserum or certain monoclonal CEA demonstrates bile canaliculi (canalicular pattern) in 70% to 80% of HCCs (24-90%). Canalicular CEA staining remains the most useful and most thoroughly investigated IHC marker in the D/D of HCC, but about 50% of PD tumors lack immunoreactivity. A false-positive HCC interpretation of a canalicular pattern may result from inclusion of

immunoreactive nonneoplastic hepatocytes within the tumor, misinterpretation of an incomplete membrane pattern as canalicular in location, or misinterpretation of periluminal immunoreactivity in Ad as staining of dilated canaliculi.

Monoclonal Ab (Mo Ab) that predominantly recognize low molecular weight & acidic cytokeratins include AE1, AE3, Cam5, 2 & 35 β H11. MoAb against the keratin subtypes may help determine the histogenesis of certain poorly differentiated tumor. For example, HCC (positive for AE3 & Cam5.2 but negative for AE1) can be separated from adenocarcinoma metastatic to the liver (positive for AE1)

Several intra-and extracellular types of hyaline glolules have been described in 10% to 15% of HCCs. They are usually PAS positive, resist predigestion with diastase, and often display immunoreactivity for AFP, A1AT or A1-antichymotrypsin. The finding of a hepatic tumor with AFP (+) is very suggestive of HCC, and its presence in PD tumors may be of particular diagnostic utility. The sensitivity varies from 15% to 70%. AFP has been found now and then in the adjacent nonneoplastic liver. Measuring serum AFP is more sensitive than finding IHC evidence of AFP in tumor tissue. Mallory's hyalin has been found in 2% to 25% of HCC, often in focal areas or in individual tumor nodules.

Hep Par-1 is a MoAb that reacts with a hepatocyte-specific epitope & the performance similar to p-CEA, with 82 % sensitivity & 90% specificity. False positives due to staining of trapped nonneoplastic hepatocytes & insensitivity of identification of PD HCC (50%) are similar to p-CEA. In summary, many investigators use a mucicarmine stain & a panel of p-CEA, m-CEA and AFP Ab when evaluating diagnostically challenging cases. Hep Par-1 may prove to complete and enhance the performance characteristics of this approach.

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<p style="text-align: center;">中華民國比較病理學會 第一次至第十八次比較病理學研討會病例一覽表</p>
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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）

動物別	診斷	提供單位
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	Pseudomembraneous 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 encephalitis	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. Rat	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 mammary 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

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|--------------|--|--------------|
| 54. Human | Fibrocalcified pulmonary TB mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM. | 林口長庚紀念醫院 |
| 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. Caprine | 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 | 國防醫學院 & 國家實驗動物繁殖及研究中心 |

	2. Benign hypertension	
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, papillary 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 B, with 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

第十二次比較病理學研討會（87年4月19日於臺灣養豬科學研究所舉行）：
心臟血管專題演講

第十三次比較病理學研討會（87年6月14日於台北市立動物園舉行）

103. Human	Atypical central neurocytoma	新光吳火獅紀念醫院
104. SD rat	Cardiac schwannoma	國家實驗動物繁殖及研究中心
105. Human	1. Mucormycosis 2. Diabetes mellitus	花蓮佛教慈濟綜合醫院
106. Dog	Parasitic meningoencephalitis, caused by <i>Toxocara canis</i> larvae migration	臺灣養豬科學研究所
107. Human	1. Primary cerebral malignant lymphoma 2. Acquired immune deficiency syndrome	台北市立仁愛醫院
108. Lamb	Listeric encephalitis	屏東縣家畜疾病防治所
109. Human	Desmoplastic infantile ganglioglioma	高雄醫學院
110. Piglet	Pseudorabies	國立屏東科技大學
111. Human	Schwannoma	三軍總醫院
112. Chicken	Avian encephalomyelitis	國立中興大學
113. Human	Tuberculous meningitis	羅東聖母醫院
114. Dog	Osteosarcoma	美國紐約動物醫學中心

第十四次比較病理學研討會（87年11月15日於國立中興大學舉行）

115. Dog	Mixed germ-cell stromal tumor, mixed Sertoli cell and seminoma-like cell tumor	美國紐約動物醫學中心
116. Human	Krukenberg's Tumor	台北病理中心
117. Human	Primary insular carcinoid tumor arising from cystic teratoma of ovary.	花蓮慈濟綜合醫院
118. Dog	Cystic endometrial hyperplasia	臺灣養豬科學研究所
119. Human	Polypoid adenomyoma	大甲李綜合醫院
120. Human	Gonadal stromal tumor	耕莘醫院
121. Dog	Cystic subsurface epithelial structure (SES)	國科會實驗動物中心
122. Human	Gestational choriocarcinoma	彰化基督教醫院
123. Horse	Ovarian granulosa cell tumor	國立中興大學

第十五次比較病理學研討會（88年4月11日於國立臺灣大學農學院附設動物醫院舉行）

124. Dog	Superficial necrolytic dermatitis	美國紐約動物醫學中心
125. Human	Solitary congenital self-healing histiocytosis	羅東博愛醫院
126. Mouse	Alopecia areata	國家實驗動物繁殖及研究中心
127. Human	Eumycotic mycetoma	花蓮佛教慈濟綜合醫院
128. Goat	Contagious pustular dermatitis	屏東縣&台東縣家畜疾病防治所
129. Human	Kaposi's sarcoma	華濟醫院

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| 130. Chicken | Fowl pox and Marek's disease | 國立中興大學獸醫學系 |
| 131. Human | Basal cell carcinoma (BCC) | 羅東聖母醫院 |
| 132. Dog | Transmissible venereal tumor | 國立臺灣大學獸醫學系 |

第十六次比較病理學研討會（88年6月6日於新光吳火獅紀念醫院舉行）

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| 133. Human | Japanese encephalitis | 花蓮佛教慈濟綜合醫院 |
| 134. Swine | Swine salmonellosis with meningitis | 國立中興大學獸醫學系 |
| 135. Swine | Meningoencephalitis, fibrinopurulent and lymphocytic, diffuse, subacute, moderate, cerebrum, cerebellum and brain stem, caused by <i>Streptococcus</i> spp. infection | 國家實驗動物繁殖及研究中心 |

第十七次比較病理學研討會（88年10月31日於台北榮民總醫院舉行）

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| 136. Lory | Viral encephalitis, polymavirus infection | 美國紐約動物醫學中心 |
| 137. Dog | Canine Glioblastoma Multiforme in Cerebellopontine Angle | 國立中興大學獸醫學院病理研究所 |
| 138. Dog | 1. <i>Aspergillus</i> spp. encephalitis and myocarditis
2. Demyelinating canine distemper encephalitis | 國立臺灣大學獸醫學系 |
| 139. Human | Disseminated strongyloidiasis | 花蓮佛教慈濟綜合醫院 |
| 140. Calf | Coliform septicemia of newborn calf | 屏東縣家畜疾病防治所 |
| 141. Human | Eosinophilic meningitis caused by <i>Angiostrongylus cantonensis</i> | 台北榮民總醫院病理檢驗部 |
| 142. Chicken | Avian encephalomalacia (Vitamin E deficiency) | 國立屏東科技大學獸醫學系 |

第十八次比較病理學研討會（89年4月30日於國立臺灣大學農學院附設動物醫院會議廳舉行）

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| 143. Dog | Osteosarcoma associated with metallic implants | 紐約動物醫學中心 |
| 144. Human | Radiation-induced osteogenic sarcoma | 花蓮慈濟綜合醫院 |
| 145. Dog | Osteosarcoma, osteogenic | 國立臺灣大學獸醫學系 |
| 146. Human | Pleomorphic rhabdomyosarcoma | 行政院衛生署新竹醫院 |
| 147. Leopard | Papillary Mesothelioma of pericardium | 國立屏東科技大學獸醫學系 |
| 148. Human | Cystic ameloblastoma | 台北醫學院 |
| 149. Canine | Giant cell tumor of bone | 國立中興大學獸醫學院 |
| 150. Human | Desmoplastic small round cell tumor (DSRCT) | 華濟醫院 |
| 151. Goat | Osteodystrophia fibrosa | 台灣養豬科學研究所&台東縣家畜疾病防治所 |
| 152. Human | Hepatocellular carcinoma | 羅東聖母醫院 |

中華民國比較病理學會
第一次至第十七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	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

分 類	病 例 編 號	診 斷	動 物 別	提 供 單 位
	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 paraffinoma	Human	中國醫藥學院
	102.	Pleomorphic adenoma (benign mixed tumor)	Human	佛教慈濟綜合醫院
	103.	Atypical central neurocytoma	Human	新光吳火獅紀念醫院
	104.	Cardiac schwannoma	SD rat	國家實驗動物繁殖及研究中心
	109.	Desmoplastic infantile ganglioglioma	Human	高雄醫學院

分 類	病 例 編 號	診 斷	動 物 別	提 供 單 位
	107.	1.Primary cerebral malignant lymphoma 2.Acquired immune deficiency syndrome	Human	台北市立仁愛醫院
	111.	Schwannoma	Human	三軍總醫院
	114.	Osteosarcoma	Dog	美國紐約動物醫學中心
	115.	Mixed germ-cell stromal tumor, mixed sertoli cell and seminoma-like cell tumor	Dog	美國紐約動物醫學中心
	116.	Krukenberg's Tumor	Human	台北病理中心
	117.	Primary insular carcinoid tumor arising from cystic teratoma of ovary.	Human	花蓮慈濟綜合醫院
	119.	Polypoid adenomyoma	Human	大甲李綜合醫院
	120.	Gonadal stromal tumor	Human	耕莘醫院
	122.	Gestational choriocarcinoma	Human	彰化基督教醫院
	123.	Ovarian granulosa cell tumor	Horse	國立中興大學
	129.	Kaposi's sarcoma	Human	華濟醫院
	131.	Basal cell carcinoma (BCC)	Human	羅東聖母醫院
	132.	Transmissible venereal tumor	Dog	國立臺灣大學獸醫學系
	137	Canine Glioblastoma Multifo Cerebellopontine Angle	Dog	國立中興大學獸醫學院病理研究所
	143	Osteosarcoma associated with metallic implants	Dog	紐約動物醫學中心
	144	Radiation-induced osteogenic sarcoma	Human	花蓮慈濟綜合醫院
	145	Osteosarcoma, osteogenic	Dog	國立臺灣大學獸醫學系
	146	Pleomorphic rhabdomyosarcoma	Human	行政院衛生署新竹醫院
	147	Papillary Mesothelioma of pericardium	Leopard	國立屏東科大學獸醫學系
	148	Cystic ameloblastoma	Human	台北醫學院
細菌	149	Giant cell tumor of bone	Canine	國立中興大學獸醫學院
	150	Desmoplastic small round cell tumor (DS	Human	華濟醫院
	152	Hepatocellular carcinoma	Human	羅東聖母醫院
	6.	Tuberculosis	Monkey	國立臺灣大學獸醫學系
	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

分 類	病 例 編 號	診 斷	動 物 別	提 供 單 位
	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
	108.	Listeric encephalitis	Lamb	屏東縣家畜疾病防治所
	113.	Tuberculous meningitis	Human	羅東聖母醫院
	134.	Swine salmonellosis with meningitis	Swine	國立中興大學獸醫學系
	135.	Meningoencephalitis, fibrinopurulent and lymphocytic, diffuse, subacute, moderate, cerebrum, cerebellum and brain stem, caused by <i>Streptococcus</i> spp. infection	Swine	國家實驗動物繁殖及研究中心
	140	Coliform septicemia of newborn calf	Calf	屏東縣家畜疾病防治所
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	屏東科技大學獸醫學系
	110.	Pseudorabies	Piglet	國立屏東科技大學
	112.	Avian encephalomyelitis	Chicken	國立中興大學
	128.	Contagious pustular dermatitis	Goat	屏東縣&台東縣家畜疾病防治所
	130.	Fowl pox and Marek's disease	Chicken	國立中興大學獸醫學系
	133.	Japanese encephalitis	Human	花蓮佛教慈濟綜合醫院

	136	Viral encephalitis, polymavirus infection	Lory	美國紐約動物醫學中心
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分 類	病 例 編 號	診 斷	動 物 別	提 供 單 位
	138	1. <i>Aspergillus</i> spp. encephalitis and myocarditis 2.Demyelinating canine distemper enceph	Dog	國立臺灣大學獸醫學系
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
	105.	Mucormycosis Diabetes mellitus	Human	花蓮佛教慈濟綜合醫院
	127.	Eumycotic mycetoma	Human	花蓮佛教慈濟綜合醫院
	138	1. <i>Aspergillus</i> spp. encephalitis and myocarditis 2.Demyelinating canine distemper enceph	Dog	國立臺灣大學獸醫學系
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
	106.	Parasitic meningoencephalitis, caused by <i>Toxocara canis</i> larvae migration	Dog	臺灣養豬科學研究所
	139	Disseminated strongyloidiasis	Human	花蓮佛教慈濟綜合醫院
	141	Eosinophilic meningitis caused by <i>Angiostrongylus cantonensis</i>	Human	台北榮民總醫院病理檢驗部
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
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分類	病例編號	診斷	動物別	提供單位
	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.	Acute Q fever hepatitis	Human	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
	151	Osteodystrophia fibrosa	Goat	台灣養豬科學研究所&台東縣家畜疾病防治所
	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	實驗動物繁殖及研究
	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
	118.	Cystic endometrical hyperplasia	Dog	臺灣養豬科學研究所
	121.	Cystic subsurface epithelial structure (SES)	Dog	國科會實驗動物中心
	124.	Superficial necrolytic dermatitis	Dog	美國紐約動物醫學中心
	125.	Solitary congenital self-healing histiocytosis	Human	羅東博愛醫院
	126.	Alopecia areata	Mouse	實驗動物繁殖及研究中心
	142	Avian encephalomalacia (Vitamin E deficiency)	Chicken	國立屏東科技大學獸醫學系

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
106 台北市大安區舟山路 142 號
國立台灣大學獸醫學系 吳憲青 先生
Tel: (02) 23630231 轉 2548 轉 1401-2(附電話答錄機)
Fax: (02) 23633289
e-mail address: wushiannching@kimo.com.tw

-----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。【註：學生會員身份變更為一般會員時，只需繳交一般會員之常年會費】

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 106 台北市大安區舟山路 142 號 國立臺灣大學獸醫學系，中華民國比較病理學會秘書處吳憲青先生收，電話：02-23630231 轉 2548 轉 1401 或 1402(附電話答錄)，傳真 02-23633289