

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八十七年度理監事座談會

時 間：87年 3月 1 日 12：30-13：00

地 點：佛教慈濟綜合醫院

主 席：理事- 黃文哲、祝志平、陳三多、施洽雯、陳安

監事- 龐 飛、李進成

請假人員：理事- 何逸遷、洪信雄、蔡信雄、方中民、朱瑞民、陳東榮、鄭益謙、

梁善居、周 冠、呂福江

監事- 王金和、林永和、羅登源

列席人員：許永祥、賴銘淙、羅振軒

記 錄：陳姿妤

一、主席報告（略）

二、報告事項（略）

三、討論議案與決議

1. 將每次會議之Moderator列名於會議議程表上，並將Speaker列名於單位之後。
2. 建請中華民國病理學會協助及鼓勵其會員參加本會活動，例如中華民國比較病理學會與病理學會合辦，二會會員可以互相討論、觀摩、不須加入會員。
3. 王金和監事因時間不夠，謹辭監事一職。候補監事由鄭謙仁副教授遞補。
4. 部份會員希望學會能發給會員證書，酌收工本費200元。
5. 第十二次比較病理研討會以心臟為專題，時間訂於四月份，劉錫光博士以演講方式，不寄發切片。地點：台灣養豬科學研究所。
6. 第十三次比較病理研討會定於六月份，以神經系統為專題，由劉振軒博士及李進成博士策畫，分為腫瘤及傳染病二部份，病例的排列順序以漸進性教學方式，並安排C.J.D.專題演講。
7. 學會會員若有興趣present，但不一定有case，可與其他有病例單位共同提供。
8. 鼓勵理、監事提高出席率。可否出席未過半數，將被迫改成座談會。選舉理監事，須經當事人同意，而選票上也應列出有意參選的會員名單。

**中華民國比較病理學會八十七年度理監事聯席會議
會議紀錄**

一、時間：87年4月19日 下午12:00～13:30

二、地點：台灣養豬科學研究所

三、主席：黃文哲

四、出席理事：黃文哲、周 冠、陳三多、施洽雯、何逸遷、呂福江

出席監事：李進成、林永和、龐 飛、鄭謙仁

五、請假理事：洪信雄、蔡信雄、朱瑞民、陳東榮、鄭益謙、梁善居、方中民、
祝志平、陳 安

請假監事：羅登源

六、列席人員：劉錫光、劉振軒

七、記錄：陳姿妤

八、主席報告：(略)

九、決議與討論：

(一)參觀台灣養豬科學研究所病理生物系。

(二)討論八十七年第十三次比較病理研討會主題、時間、地點與負責理事。

主題：神經病理專題

時間：八十七年六月十四日

地點：台北市立動物園教育中心演講廳

由李進成醫師及劉振軒博士共同籌劃

十、散會

**中華民國比較病理學會八十七年度理監事聯席會議
會議紀錄**

一、時間：87年6月14日 下午12:00～13:30

二、地點：台北市立動物園教育中心演講廳

三、主席：黃文哲

四、出席理事：黃文哲、方中民、陳三多、施洽雯、何逸遷、祝志平、陳 安

出席監事：李進成、林永和、龐 飛、鄭謙仁

五、請假理事：洪信雄、蔡信雄、朱瑞民、陳東榮、鄭益謙、梁善居、周 冠、呂福江

請假監事：羅登源

六、列席人員：許永祥、江宏、余忠泰、劉振軒

七、記錄：陳姿妤

八、主席報告：(略)

九、決議與討論：

討論八十七年第十四次比較病理研討會主題、時間、地點與負責理事。

主 題：生殖系統 (reproductive system)

時 間：八十七年十一月十日

地 點：國立中興大學

負責理事：陳三多

十、散會

中華民國比較病理學會八十七年度理監事聯席會議 會議記錄

- 一、時 間：87年11月15日12:30-14:00
- 二、地 點：中興大學-國際會議廳
- 三、主 席：黃文哲
- 四、記 錄：鄭三福
- 五、出席理監事：黃文哲、祝志平、陳三多、周 冠、龐 飛、施洽雯、林永和、
呂福江、鄭謙仁
- 六、列 席 人員：劉錫光、陳憲全、余忠泰、賴銘淙、劉振軒
- 七、請假理監事：何逸僊、洪信雄、蔡信雄、方中民、朱瑞民、陳東榮、鄭益謙、
羅登源、梁善居、李進成、陳安
- 八、主席報告：(略)
- 九、會務報告：(略)
- 十、討論

(一) 希望利用 E-mail 傳送資料和 handout 利用此管道，使學術界和醫學界有互相交流訊息的管道，請大家(會員) 提供 E-mail address。

決議：秘書處已在本次大會手冊中列出 E-mail Address，將來也會發函會員請提供 E-mail address。

(二) 理事長為榮譽職，不得連任，每次只選秘書長，秘書長任滿後即任理事長。

決議：理事長之產生應按本會章程規定，除非修改大會章程。至於下屆理監事的選舉應考量出席率及參與熱誠度。此外，秘書處應先發函各會員徵詢是否有意願參與理監事選。

(三) 建議下屆理監事選舉採通訊方式投票。

決議：下一屆理監事選舉方式原則上仍以親自出席及委任投票實施。

(四) 請討論下次開會時間、地點、負責理事與專題。

決議：主題以皮膚疾病為專題，時間在1999年春季，確定日期將由負責理事龐飛教授與秘書處討論，地點在國立台灣大學動物醫院舉行。

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	46/02/25	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	40/08/11	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	37/03/20	北海道大學 獸醫學博士	屏東技術學院 動物醫院院長	屏東技術學院 獸醫系教授	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 查署法醫中心召集人	103 台北市迪化街175巷 16號	02-7370570	02-7359413
理事	朱瑞民	男	34/07/14	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	45/05/14	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	46/08/30	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	40/08/30	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	42/08/18	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	46/02/24	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 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	49/01/13	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
八十八年度會員大會暨第十五次比較病理學研討會（皮膚系統專
題）

議 程 表

時 間：中華民國八十八年四月十一日（星期日）上午08:30~下午3:50

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：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
中華民國比較病理學會

時 間	議 程
08:30- 09:20	報 到
09:20- 09:30	開幕致詞
	Section 【1】
09:30- 09:50	Case 124 美國紐約動物醫學中心
09:50- 10:10	Case 125 羅東博愛醫院
10:10- 10:30	Case 126 國科會國家實驗動物繁殖及研究中心
10:30- 11:00	Coffee Break
11:00- 13:30	會員大會 第二屆理監事改選 午餐
	Section 【2】
13:30- 13:50	Case 127 花蓮慈濟綜合醫院
13:50- 14:10	Case 128 屏東縣家畜疾病防治所&台東縣家畜疾病防治所
14:10- 14:30	Case 129 華濟醫院
	Section 【3】
14:30- 14:50	Case 130 國立中興大學獸醫學系
14:50- 15:10	Case 131 羅東聖母醫院
15:10- 15:30	Case 132 國立臺灣大學獸醫學系
15:30- 15:50	綜合討論
16:00- 17:30	新舊任理監事交接及第二屆理監事第一次聯席會議

中 華 民 國 比 較 病 理 學 會
第十五次比較病理學研討會（皮膚系統專題）
病 歷 摘 要

時 間：中華民國八十八年四月十一日（星期日）上午8:30~下午3:50
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：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
中華民國比較病理學會

CP Case 124美國紐約動物醫學中心 (A41168)

This is a 5-year-old, Belgian Shepherd male dog had a history of seizures, which had been treated with barbiturates for more than a year. Recently, the dog also had a history of anorexia, vomiting, and weight loss. Dog developed ulcerative skin of dorsal terminal digits and hyperkeratosis lesions on all four feet. Blood chemistry tests revealed alkaline phosphates were higher, 1823 (10-150) IU/L; SGPT (ALT) was higher, 101 (5-60) IU/L; CPK was higher, 656 (10-200); BUN was lower, 6 (7-27) mg/L; and total bilirubin was higher, 0.5 (0.0-0.4) gm/DL; Urinalysis was bilirubin 3+, blood 3+, and protein 3+.

CP Case 125 羅東博愛醫院 (94-3516)

A male newborn was born on October 31, 1994. At the time of birth, the newborn was found to have a solitary ulcerated skin lesion measuring 1.0 cm in diameter at left perinipple area. The patient was initially treated with antibiotics (Augmentin) under the clinical impression of infectious granuloma. Since the size of the skin lesion increased gradually and the ulcer was still present, a skin biopsy was performed.

CP Case 126 國科會國家實驗動物繁殖及研究中心 (C3H/J)

A one-year-old, C3H/HeJ female mice, showed diffuse ventral and patchy dorsal alopecia. No significant ulcerative lesions, ectoparasite, or fungi were found.

CP Case 127 花蓮慈濟綜合醫院 (95-3788)

This 48 year-old female patient was suffered from right thigh tenderness and swelling for more than 8 years. Physical examinations revealed multiple discharging sinuses with milky thick secretion mixed with some blackish granules in the right thigh. Surgical debridement was done.

CP Case 128 屏東縣家畜疾病防治所&台東縣家畜疾病防治所

A dairy goat farm reared about 120 kids. Some affected kids, 20 to 45 days old, showed

signs of depression and reluctant to suck or graze. Pustules or crusty lesions typically formed on the lips, muzzle, face, and around the eyes of the kids. One year ago with the same signs, the morbidity in young kids approached 100%, while mortality from starvation and secondary infection reached about 90%. After proper disinfection of artificial suckling instruments, antibiotic treatment, and segregation of the kids, the mortality reduced to 3% at this time. As requested by the owner, the tissues from the crusty lesions were taken for diagnosis. Lesions regressed in 4 weeks.

CP Case 129 華濟醫院 (8802407D)

This 89 years old female patient was admitted via OPD on 87-06-19. She had the history of both eye blind. According to the statement of patient's family, she was suffered from a huge mass about 6×6 cm. and multiple small mass in left leg. A few day the mass grew larger gradually was noted. So she came to our hospital for help and admitted to surgical unit for further treatment. After discharged in this 3rd september, she developed alternation of consciousness and coma was noted. She was taken to our ER at 87-09-07. She was in shock status when arriving our hospital. The Lab data revealed leukocytosis WBC 27100, She was then admitted to our ICU for critical care, unfortunately, she expired at 87-09-08 due to sepsis.

CP Case 130 國立中興大學獸醫學系 (CO99-7574)

The farm raised about 200 native chickens, aged about 2 months old. They showed typical Marek's disease and some of the affected birds were sent to our teaching hospital for pathological diagnosis. One of the chickens showed several greyish-brown nodules scabs on the unfeathered parts of the head, including comb, wattles, eyelids and the corners of the beak.

CP Case 131 羅東聖母醫院 (990621 A5)

A 81-year-old male, suffered from back skin itching for years with increased severity. Recently, he came to our hospital and was admitted under the impression of skin cancer. Wide excision and local skin flap reconstruction then were performed. A $8.5 \times 4.6 \times 1.2$ cm skin was submitted with a central large elevated lesion, up to 5.6×4.4 cm to be noted. Serial sectioning to mapping the distribution of skin lesion was performed and one of the representative blocks was demonstrated in the slide.

CP Case 132 國立臺灣大學獸醫學系 (NTU 960410A)

This 8-month-old male, Husky dog was presented to our hospital with more than 20 hard masses (5-8 cm in diameter) found subcutaneous tissue over the whole body. These masses were observed by the owner 2-3 weeks ago. He had normal appetite, and treated with Amoxicillin, Prednisolone and eventually Oncovin. Masses got smaller after the treatment and some of them disappeared, but 6 months later, one new mass was observed on his right side of abdomen.

Comparative Pathology Case 124

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

Caspary Research Institute, The Animal Medical Center, Cornell University Medical College, Wildlife Conservation Society, New York, New York USA; Pig Research Institute Taiwan, ROC (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: This is a 5-year-old Belgian Shepherd male dog had a history of seizures, and had been treated with barbiturates for more than a year. Recently, the dog also had a history of vomiting, and weight loss. Dog developed ulcerative skin lesions on all four feet. Blood chemistry tests revealed alkaline phosphates were Higher, 1823 IU/L (normal 10-150 IU/L); SGPT(ATL) was higher, 101 IU/L (normal 5-60 IU/L); CPK was higher, 656 IU/L (normal 10-200 IU/L); BUN was lower, 6 mg/L, (normal 7-27 mg/DL); Total bilirubin was higher, 0.5 mg/DL (normal 0-0.4mg/DL). Urinalysis revealed bilirubin 3+, blood 3+, and protein 3+. Hepatocutaneous syndrome was diagnosed by skin biopsy. The dog was euthanatized for necropsy by owner requested.

Diagnosis: Superficial necrolytic dermatitis (Hepatocutaneous syndrome)

Gross findings: Bilateral erythematous, erosive, and crusted lesions were observed in the muzzle, lips, periocular, shoulder and elbow areas. Remarkable, ulcerative hyperkeratosis was observed in the footpads of all four legs, and ulcerative, crusted skin of dorsal terminal digits. The liver was smaller and nodular, most of the nodules were a few mm to 17 mm in diameter. The capsule between the nodules was thickening and opaque, but over the nodules was ordinary thickness.

Histopathological findings: In the footpads, extensive hyperkeratosis and parakeratosis were observed in the stratum corneum. There were inter-and intracellular edema in the keratinocytes in the acanthotic subcorneal layer. Occasional neutrophil accumulation and formation of pustulus were seen in the subcorneal region. In the eroded skin marked neutrophilic exocytosis and inflammatory crust covered the surface. In the liver extensive proliferation of fibrous connective tissue and infiltration of lymphoplasmacytes and a few neutrophils were observed in the portal region. Hepatic cord cells were distorted and revealed degeneration and necrosis. Hepatocellular hyperplasia and lipidosis were seen in the adjacent areas.

Discussion: The clinical signs of hepatic failure, gross erythematous, ulcerative, parakeratotic, crusted dermatitis in the footpads, and histologic features including a thick layer of hyperkeratosis, acanthosis and parakeratosis in the stratum corneum, a zone of intra- and

intercellular edema in the corneal epidermal junction are characteristic of canine superficial necrolytic dermatitis is closely resembling human necrolytic migratory erythema. The disease is commonly associated with glucagon-secreting tumors of the pancreatic tumors, diabetes mellitus and hepatic failure in human patients and dogs, and in a dog associated with the ingestion of mycotoxins. Blood chemistry tests revealed marked increased volum of alkaline phosphates, SGPT, CPK and totaal bilirubin indicated hepatic failure, hepatic cirrhosis was comfirmed at necropsy examination. Hepatic cirrhosis may be results of chronic barbiturates toxicity since the dog has been treated seizures for more than 13 months.

The pathogenesis of superficial necrolytic dermatitis is unknown; however, hepatic dysfunction and derangements of glucose and amino acid metobolism are clearly involved. Hypoaminoacidemia resulted in sustanined gluconeogenesis is documented in both canine and human patients, which may be due to deplete epidermal protein and induce epidermal necrosis.

The gross and histologic features of superficial necrolytic dermatitis have many similarities to those of parakeratosis in swine with zinc deficiency and dogs with zinc-responsive dermatoses. The requirementt of swine for zinc is remakable high 30 – 50 parts per million of the diet and a deficiency of the mineral leads to severe parakeratosis. The lesions are characterized by severe acanthosis and parakeratosis of the skin, tongue and esophagus, and without hepatic lesions.

Herbivorous animals ate certain plans which are photosensitizers are able to obtain energy for chemical activation by the absorption of light of the appropriate wavelengths. Heavy pigmented areas which restrict the penetration of light escape. Heavy fleece and haircoats are also protective. The white skin ears, eyelids, face and muzzle are usually affected, and the black, heavy fleece or hair area not at all. The affected areas of skin and undergo necrosis, and dry gangrene, and eventually slough with or without hepatic diesase. These lesions are different from those of dogs with supeerficial ulcerolytic dermatitis and with hepatic failure.

Diagnostic criteria:

1. Extensive hyperkeratosis, parakeratosis and acanthosis in the stratum corneum
2. A zone of intra- and intercellular edema in keratinocytes of the subcorneal areas.
3. Neutrophil accumulation and formation of subcorneal pustulus.
4. Hepatic failure

References:

1. Walton DK, Center SA, Scott DW, Collins K: Ulcerative dermatosis associated with diabetes mellitus in the dog: A report of four cases. J Am Anim Hosp Assoc 22:79-88, 1986.
2. Miller WH, Scott DW, et al. Necrolytic erythema in dogs: A hepatocutaneous syndrome. J Am Anim Hosp Assoc 26:573-581, 1990.
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6. Franchimont C, Pierard GE, Luyckx AS, Gerald J, Lapire CM. Angioplasic necrolytic migratory erythema. Am J Dermatopathol 4:485-495. 1982.
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Comparative Pathology Case 125

Contributors: Chia-Wen Shih (施洽雯)¹, MD, MS; Wei-Hwa Lee (李偉華)², MD, PhD; Yung-Ching Chang (張永青)³, MD; Wei-Wu (吳維)⁴, MD.

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(羅東博愛醫院)

Department of Pathology², Tri-Service General Hospital, National Defense Medical Center, Taipei. (三軍總醫院病理部)

Clinical history: A male newborn was born on October 31, 1994 to a 26-year-old gravida 1, Para 1 healthy woman after an uneventful prenatal course. The newborn weighed 2900 gm and was 48 cm long.

At the time of birth, the newborn was found to have a solitary ulcerated skin lesion measuring 1.0 cm in diameter at left perinipple area. Otherwise, the newborn was in excellent health. Neither fever nor hepatosplenomegaly nor lymph node enlargement were noted. Laboratory studies disclosed the following values: RBCs, 3,560,000/cumm; hemoglobin, 10.9 g/dl; hematocrit, 31.6%; Platelets, 615,000/cumm; WBCs, 12,910/cumm with 54.9% lymphocytes, 38.0% neutrophils, 5.0% eosinophils, 2.0% monocytes, and 0.1% basophils.

The patient was initially treated with antibiotics (Augmentin) under the clinical impression of infectious granuloma. Since the size of the skin lesion increased gradually and the ulcer was still present, a partial skin biopsy was performed on December 13, 1994. Since the infant was clinically well, no medical treatment was given. The newborn was followed up regularly by the clinicians. The skin lesion resolved spontaneously over the next one month following diagnosis, leaving a small, atrophic, white scar. The infant was asymptomatic with normal growth and development at the age of 2 years. No evidence of recurrence or systemic involvement was noted.

Diagnosis: Solitary congenital self-healing histiocytosis.

Gross findings: The specimen submitted consisted of a small tissue fragment measuring 0.2×0.2×0.2 cm in size, fixed in formalin. Grossly, it is grayish-brown in color and soft in consistency.

Histopathological findings: Hematoxylin and eosin-stained sections of the biopsy showed that the upper to lower dermis was infiltrated by proliferation of histiocytic cells. Similar-appearing cells were also noted diffusely extending into the subcutaneous tissue. The histiocytic cells contained abundant acidophilic pale-staining cytoplasm. Some had foamy cytoplasm. The

nuclei were round or oval with evenly dispersed chromatin and inconspicuous nucleoli. The cellular infiltrations were also intermixed with lymphocytes, eosinophils, and neutrophils.

Immunohistochemical findings: Immunohistochemical study for four markers was performed by peroxidase-antiperoxidase method. It revealed the histiocytic cells to be strongly positive for S-100 protein (DAKO, Japan) but negative for lysozyme (DAKO, Japan), alpha-l-antichymotrypsin (DAKO, Japan), and leukocyte common antigen (DAKO, Japan). The lysozyme and alpha-l-antichymotrypsin showed positive in median-sized monocytes which were not stained by S-100. The leukocyte common antigen stain showed positive only in the small lymphocytes.

Discussion: Congenital self-healing histiocytosis (CSHH), also known as congenital self-healing reticulohistiocytosis, is a rare entity first described by Hashimoto and Pritzker in 1973 (1). To date, only forty-one cases have been reported (1-9). CSHH frequently presents a diagnostic dilemma in the newborn. In general, these cases were characterized by 1) congenital or perinatal occurrence of multiple cutaneous nodules, 2) absence of extracutaneous involvement, 3) skin biopsy showed dermal infiltration of histiocytic cells, 4) spontaneous involution of the lesions within two to three months, 5) no recurrence. However, Berger et al. reported the first four cases of solitary CSHH in 1986 (10). Since then, only seven other cases of solitary CSHH have been reported in the world literature (4,6-8,11-13) (Table 1). Because the histological and immunohistochemical pictures of CSHH are similar to the better known-aggressive forms of histiocytosis X, CSHH frequently presents a diagnostic dilemma in the newborn. The ability to distinguish CSHH from classical histiocytosis X is very important (Table 2), for the former will regress spontaneously but the latter requires aggressive treatment and always has a poor prognosis.

The pathogenesis of CSHH is still an enigma. Some investigators consider it to be a reactive process with a proliferation of Langerhans' cells secondary to an underlying immunologic defect (13), whereas, others view it as a congenital migration defect of immature Langerhans' cell (2). Recently, Willman et al. reported that Histiocytosis X is a clonal neoplastic disorder with highly variable biologic behavior (14). Although some authors consider CSHH to be a benign variant of classical histiocytosis X (15), there is no report of CSHH about the clonality, and CSHH is now still known as a distinct clinical entity (1,6). The surrounding thick inflammatory infiltrate including eosinophils may play an important role in the rapid spontaneous regression of the lesion (13).

Clinically, differentiating CSHH from histiocytosis X is sometimes easy because the former is characterized by multiple cutaneous eruptions of firm, reddish-violaceous or brown nodules scatter over the scalp and face, and also the trunk and extremities. Importantly the mucous membranes are always spared and the systemic involvement is always absent in CSHH. In contrast, the mucous membrane and systemic involvement is common in histiocytosis X. The constant and characteristic features of CSHH are its congenital occurrence and spontaneous regression within two to three months. In contrast, patients with apparently localized histiocytosis X may subsequently develop additional lesions, usually within 6-12 months (16).

Microscopically, the differentiation between CSHH and the cutaneous form of histiocytosis X on routine hematoxylin-eosin stain is indeed very difficult. However, the followings are helpful and raise the possibility of CSHH (15): 1) The tumor cells are often large and may up to 45um in diameter. 2) The tumor cells contain predominant acidophilic ground-glass cytoplasm and coarse nuclear chromatin. 3) The proliferative tumor cells often coexist with lymphocytes, neutrophils, and eosinophils. 4) Not only the dermis but also the epidermis and skin appendages are infiltrated. 5) The cellular infiltrate may deeply reach the subcutaneous fat tissue.

Immunohistochemically, in contrast to the infiltrate in most non-X histiocytoses, CSHH is very similar to classical histiocytosis X (6) with the infiltrating cells generally showing the phenotype of Langerhans' cells and strongly positive for S100 protein (2,11). Similarly, they also show positive staining for CD1a (2,10,11) and HLA-DR (human leukocyte antigen-DR) (2). However, they are negatively stained by lysozyme (17), alpha-lantichymotrypsin (2), and leukocyte common antigen (2).

Ultrastructural study is helpful but not necessary in making the diagnosis of CSHH. The most characteristic is its laminated dense bodies (9,15). The Birbeck's granules are present in most (over 50%) of the histiocytosis X, but can be found in only 10-20% of CSHH (1,2). It has been hypothesized that CSHH is a benign Langerhans' cell disease in which Birbeck granules are transformed to laminated dense bodies and possibly degraded by lysosomal enzyme (2).

In histiocytosis X, the mortality is as high as 80% when the disease presents in the neonatal period (18). However, therapy is not encouraged for CSHH, and the prognosis is always favorable. Therefore, the major problem seems to be the risk of overdiagnosis and dangerous unnecessary treatment. Some of the previously reported similar cases, which had only skin lesions of histiocytosis, were diagnosed as histiocytosis X and were treated with radiotherapy (19,20), chemotherapy (21), corticosteroid (22,23), or even the three combined. However, it is not well documented if such therapy indeed influenced the course of disease, for they may be cases of CSHH and not the classical histiocytosis X by the current definition. It is our opinion that patients with histiocytosis confined to the skin should be closely followed up to rule out the possibility of progressive disease. Aggressive therapy is not necessary and time must be allowed for spontaneous resolution.

In summary, solitary cutaneous nodules apparent at birth present a diagnostic challenge. Differential diagnosis is very important. We present the case with the hope that both clinicians and pathologists to be more acquainted with this disease to avoid making an over-diagnosis resulting in unnecessary treatment.

Table 1. Cases of solitary CSHH Published in the Literature

Authors	Year	Sex	Location	Age of onset	Time of ulceration	Time of involution	Immunohisto-chemistry
Berger et al(1)	1986	F	Temple, R't	Birth	1 week	18 weeks	S-100 + Lysozyme
		M	Foot, R't	Birth	Birth	9 weeks	OKT6 + OKT4
		M	Hand, R't	Birth	Birth	Excised	ND
		M	Groin, L't	Birth	2 days	10 weeks	S-100 +
Taieb et al(19)	1986	F	Gluteal, L't	3 weeks	8 weeks	Excised	S-100 + OKT6 + OKT4 + OKM1 +
Jordaan et al(11)	1986	M	Iliac, R't	Birth	Absent	4 weeks	S-100 -
Ofuji et al(16)	1987	M	Shoulder, L't	Birth	2 days	36 days	ND
Boullie et al(3)	1988	F	Scapula, L't	Birth	Birth	Few days	ND
Divaris et al(6)	1991	M	Scalp	Birth	Birth	11 days	S-100 + alpha-1-anti- trypsin +
Levisohn et al(14)	1993	M	Sacrum	Birth	Birth	24 weeks	S-100 +
Bernstein et al(2)	1993	M	Thigh, L't	Birth	Birth	12 weeks	S-100 + LCA -
Presetit case	1995	M	Chest, L't	Birth	Birth	10 weeks	S-100 + LCA - Lysozyme - alpha-1 -anti - chymotrypsin

CSHH: congenital self-healing histiocytosis; ND: not done; OKT: orthoclone T-lymphocyte antigen;

LCA: leukocyte common antigen.

Table 2. The usual differential diagnosis of CSHH and Histiocytosis X

	CSHH	Histiocytosis X
Age of onset	At birth	Infancy or young children
Skin lesion	Exclusively nodular lesion without admixture with papule or crusting	Nodular lesion admixture with papule or crusting
Mucous membrane involvement	No	Yes
Systemic involvement	No	Yes
Clinical course	Spontaneous regression	Progressive
Histopathology	Infiltration of Langerhan's cells in the upper and middle dermis, tend to involve deep dermis and subcutaneous tissue sometimes sparing the papillary dermis and epidermis.	Infiltration of Langerhan's cells chiefly in the papillary dermis and epidermis.
Immunohistochemistry		
S-100	Positive	Positive
Leukocyte common antigen	Negative	Negative
Lysozyme	Negative	Negative
Alpha-1-antichymotrypsin	Negative	Negative
Electron microscopy		
Birbeck' granule	Positive in 10-20 %	Positive in over 50 %
Birbeck' granule coexist with laminated dense body	Usually present	Does not occur
Treatment	Unnecessary	Corticosteroid Chemotherapy Radiotherapy
Recurrence	No	Yes
Prognosis	Favorable	High mortality

Diagnostic criteria:

1. Age of onset: At birth.
2. Exclusively nodular lesion without admixture with papule or crusting.
3. No mucous membrane involvement, No systemic involvement.
4. Spontaneous regression, no recurrence.
5. Histopathology: Infiltration of Langerhans cells in the upper and middle dermis, tend to involve deep dermis and subcutaneous tissue.
6. Immunohistochemistry: S-100 (+), Lysozyme
Leukocyte common antigen
Alpha-1-antichymotrypsin

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Comparative Pathology Case 126

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Clinical history: A 12-month-old, female specific pathogen free (SPF) inbred mouse (C3H/HeJ) was from the colony originally introduced from Jackson Laboratory (Bar Harbor, Maine, USA). The mouse showed pathy to diffuse alopecia of the dorsal and abdominal skin without visible ulcerative changes.

Diagnosis: Alopecia areata, diffuse, C3H/HeJ mouse

Gross findings: Pathy to diffuse alopecia of the dorsal skin and abdomen without visible gross lesions are noted. Other organs are grossly normal.

Histopathological findings: In general, the epidermis is normal but thin. Hair follicles are small, short, and sometimes distorted in contour and have overall telogen configuration. Lymphocytes macrophages, and occasional netrophils encircle and are generally oriented around the lower two thirds of the anagen hair follicles; less commonly, lesions that more closely resemble those of humans are characterized by a tight" swarm of bees" accumulation of lymphocytes around the hair bulbs only.

In the affected anagen follicles, dystrophic hair formation is observed with apoptosis and dyskeratosis, amorphous material in the pilary canal, and adjacent pigmentary incontinence.

Dicussion: In the United States about 1% of the population have experienced alopecia areata (AA), however about 90% of these people have only had peridic episodes. Heredity plays a role in 20% alopecia areata cases. The exact cause of alopecia areata is unknown, however researchers believe it is an auto-immune condition. There is no cure for alopecia areata, although there are ways of treating the condition, with varing degrees of effectiveness.

Alopecia areata often occurs in families whose members have had asthma, hay fever, atopic eczema, or other autoimmune diseases such as thyroid disease, early onset diabetes, rheumatoid arthritis, lupus erythematosus, vitiligo, pernicious anemia, or Addison's diseases.

The most common treatment is the injection of cortisone into the bare skin patches. 2-5%

topical minoxidil solution, anthralin cream or ointment, cortisone pills and topical immunotherapy are the other methods treating alopecia areata.

Alopecia areata, seen very rarely in the dog and cats. C3H/HeJ inbred mice develop a diffuse ventral and patchy dorsal alopecia that closely mimics human AA. The hair loss increases in frequency as animal age and with selective breeding within the colony. This type of alopecia is found between 4 and 5 months and increases in frequency with age in both male and female. In large production colonies, approximately 0.25% of the females and 0.035% of the males are affected by age 6 months.

A number of lesions were concurrent with AA in these C3H/HeJ mice. These lesions include retinal dystrophy, cardiac calcinosis, renal tubular mineralization, skeletal muscle and lingual mineralization, blepharitis, Meckel's diverticulum, otitis media, coccygeal hemivertebrae, and hepatic telangiectasia.

Differential diagnosis for AA include those atrophic follicular diseases e.g. canine familial dermatomyositis and post-rabies vaccination alopecia in dog, chronic ulcerative dermatitis in black mice.

Diagnostic criteria:

1. C3H/HeJ mice species-dependant
2. Patchy to diffuse ventral and dorsal alopecia
3. No pathogenic organism is consistently identified by culture, serological testing.
4. In general, no ulcerative gross visible lesion of the skin.
5. Perifollicular mononuclear cells infiltration(CD8 cells > CD4 cells) are present around and within the follicular epithelium.
6. Need differentiate from chronic ulcerative dermatitis in black mice, C57BL/6J and C57BL/10J.

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Comparative Pathology Case 127

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Clinical history: This 48 year-old female patient was suffered from right thigh tenderness and swelling for more than 8 years. Physical examinations revealed multiple discharging sinuses with milky thick secretion mixed with some blackish granules in the right thigh. Surgical debridement was done.

Diagnosis: Eumycotic mycetoma

Histopathological findings: The dermis and subcutaneous tissue contain localized abscesses, each with one or more granule in its center. Between abscesses, there is extensive granulation tissue and fibrotic tissue. G.M.S stain demonstrates broad, septate, fungal hyphae, in the granules. So eumycotic mycetoma is diagnosed.

Discussion: Mycetoma are tumorous lesions of the subcutaneous tissues and bone caused by a wide variety of geophilic actinomycetes and eumycotic fungi that form granules within tissue. It is commoner in males, especially those in their third or fourth decade who work on the land. Patients are infected when these exogenous organisms are introduced into some part of the body, usually the lower extremities or the trunk, as the result of the trauma. The clinical triad of subcutaneous nodules, sinuses and discharge usually lead to diagnosis; the disease is commonly painless. They are two distinct types of mycetomas: actinomycotic and eumycotic. Accurate histologic differentiation between the granules formed by actinomycetes and fungi is crucial in determining the form of treatment and the prognosis of mycetoma. Special stains for bacteria and fungi may be needed. Granules of actinomycotic mycetomas contain delicate, gram-positive, branched, bacterial filaments, about 1 μ m in width, whereas those of eumycotic mycetomas contain broad septate, fungal hyphae, 2 to 6 μ m or more in width. Because many granules have a characteristic architecture, presumptive etiologic identification can often be made histologically. However, culture is needed for definitive identification. Medical therapy is the treatment of choice for actinomycetoma. Surgery is seldom used for actinomycetoma. A combination of medical treatment and surgery is advised for eumycetoma. However, recurrence is common, rates ranging from 20 to 90 percent.

Comparative Pathology Case 128

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Clinical history: A dairy goat farm reared about 120 kids. Some affected kids, 20 to 45 days old, showed signs of depression and reluctant to suck or graze. Pustules or crusty lesions typically formed on the lips, muzzle, face, and around the eyes of the kids. One year ago with the same signs, The morbidity in young kids approached 100%, while mortality from starvation and secondary infection reached about 90%. After proper disinfection of artificial suckling instruments, antibiotic treatment, and segregation of the kids, The mortality reduced to 3% at this time. As requested by the owner, The tissues were taken from the crusty lesions for diagnosis. Lesions regressed in 4 weeks.

Diagnosis: Contagious pustular dermatitis

Gross findings: The gross lesions of affected kids were multiple thick brown-gray crust formed on the lips, muzzle, face, gum, and around the eyes. The lesions may be elevated 2-4 mm above the skin surface with 0.5-2 cm in diameter and showed some yellowish pus or congestion when the crust was peeled off.

Histopathological findings: The microscopic lesions were characterized by vacuolation and swelling of the keratinocytes in the stratum spinosum, ballooning degeneration with intracytoplasmic eosinophilic inclusion bodies, reticular degeneration, marked epidermal proliferation and accumulation of scale-crust, composed of ortho- and parakeratotic hyperkeratosis, proteinaceous fluid, degenerating neutrophils, cellular debris, and bacterial colonies. The dermal lesions included superficial edema, marked capillary dilation, and lots of mononuclear cells, neutrophils and fibroblasts infiltration.

Electron microscopic findings: Elongated ovoid viruses of parapox were found in scab suspensions by electron microscope.

Discussion: Contagious pustular dermatitis is a poxvirus disease of sheep and goats, with incidental infections occurring in humans, cows, wild ruminants and very rarely, dogs. The disease is caused by a parapoxvirus closely related to pseudocowpox and bovine papular stomatitis. Synonyms for contagious pustular dermatitis include contagious ecthyma, infectious

labial dermatitis, soremouth, scabby mouth, and orf. Orf is used to describe human infections.

Contagious pustular dermatitis affects sheep and goats of all breeds . It is predominantly a disease of lambs and kids. Infection is established through cutaneous abrasions, particularly those associated with dry and prickly pasture or forage. Clinically affected lambs may transmit the virus to the udder of the ewe. The virus is hardy and probably persists in the environment indefinitely in crust material shed from affected animals. The morbidity in young kids often approaches 100%, while mortality from starvation and secondary infections may be as high as 20%. In an outbreak in Brazil, 93% mortality in kids was reported. The high mortality in this case which may be due to the starvation and secondary infection is similar to the one in Brazil

The gross lesions usually commence at the commissures of the lips and spread around the lip margins to the muzzle. Primary lesions sometimes occur on the face about the eyes. In severe cases, lesions may develop on the gingiva, dental pad, palate, and tongue. Lesions mainly confined to the tongue must be differentiated from those of foot-and-mouth disease. Very rarely, lesions extend to the esophagus, rumen, and omasum and recorded in lungs and heart, and in the lower alimentary canal, causing ulcerative gastroenteritis. Lesions on the limbs are less common than on the lips and tend to involve the coronet, interdigital cleft, and bulb of the heels. They may extend, in severe cases, to the knee or hock on the posterior aspect of the leg. Lesions of the mammary gland affect the teats and adjacent skin of the udder. The lesions develop through the typical pox phases but are much more proliferative. The vesicular stage is transient, and pustules are flat rather than umbilicated. The most significant gross lesion is the elevated thick brown-gray crust above the skin surface. Regression is normally complete in 4 weeks as this case but the lesions are limited to the mouth, muzzle and face of the kids in this case. The microscopic lesions are characterized by ballooning degeneration with intracytoplasmic eosinophilic inclusion bodies. The differential diagnosis of the lesions includes Orthopoxvirus, Capripox, and Parapoxvirus infections.

Human orf virus infection occurs mainly in relatively well-defined 'at risk' populations, such as veterinary surgeons, shepherds and abattoir workers in whom it is an occupational hazard. Human infection appear most common during the shearing and slaughtering season for sheep and goats and is often related to meat handling. The hands are the most common sites of infection, with other sites such as face only occasionally being involved. The incubation period is usually 3 to 6 days, with clinical presentation 3 to 4 weeks after infection. Macules developing into papules are the initial features. Between 1 and 2 weeks, the lesions develop a red center surrounded by a white ring and red halo, and then progress from a nodular stage with central umbilication to a papillomatous stage 3 to 4 weeks after infection. Healing occurred after 4 to 24 weeks. Regional adenitis is present and Erythema multiforme bullosum is noted.

Diagnostic criteria:

1. Crusty, proliferative lesions typically formed on the lips, muzzle, face, and ears.
2. Ballooning degeneration with intracytoplasmic eosinophilic inclusion bodies in the microscopic lesions.
3. Electron microscopy or immunological techniques to demonstrate antigen in scabs or serology.

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Comparative Pathology Case 129

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Clinical history: This 89 years old female patient was admitted via OPD on 87-06-19. She had the history of both eye blind. According to the statement of patient's family, she was suffered from a huge mass about 6×6 cm. and multiple small mass in left leg. A few day the mass grew larger gradually was noted. So she came to our hospital for help and admitted to surgical unit for further treatment. After discharged in the 3rd september, she developed alternation of consciousness and coma was noted. She was taken to our ER at 87-09-07. She was in shock status when arriving our hospital. The Lab data revealed leukocytosis WBC 27100, she was then admitted to our ICU for critical care, unfortunately, she expired at 87-09-08 due to sepsis.

Diagnosis: Kaposi's sarcoma

Gross findings: Several papular or nodular or coalesce to form enlarging patches and plaques in the left low leg are seen. A huge fungating mass (6×6 cm.) with central erosion or ulceration in noted.

Histopathological findings: The tumor is the presence of spindle cells forming slits containing red blood cells. Admixed in this lesion are lymphocytes, hemosiderin-laden macrophages and other inflammatory cells a network of spindle cells and large vascular spaces will be seen with characteristic thin-walled “back-to-back” capillaries. The interweaving bands of spindle cells embedded in a network of reticulin, eventually replacing the collagen, and a maze of vascular spaces are invariable features of the tumor. The vascular comonent is formed partly by delicate capillaries and partly by cleft-like spaces between the spindle cells. Extravasated erythrocytes may be seen. The blood vessels around the tumor may show proliferative endarteritis. Foci of necrosis occur in some of the masses. A characteristic feature is the presence of both intracellular and extracellular PAS-positive hyaline globules. Free red cells are also seen.

Immunohistochemistry findings: Strong immunoreactivity for Factor VIII-related antigen and vimentin indicate that is dervived from vasoformative mesenchyme with multipotential capabilitis. It has been suggested that the spindle cells of this lesion are endothelial, of either blood vessel or lymph vessel origin.

Focally positive for S-100.

Discussion: First reported in 1872 by Moricz Kaposi, this disease is multicentric neopastic tumor characterized by endothelium-lined channels and vascular spaces admixed with variably

sized aggregates of spindle shaped cells. Kaposi's sarcoma [KS] primarily affects the skin, but numerous other tissue may be similarly affected.

KS is associated with four clinical settings [Sieglar et al, 1984]: (1) Classic KS, (2) endemic KS, (3) KS associated with AIDS, and (4) KS associated with lymphoproliferative disease or organ transplant.

1. The classic KS [sporadic cases] are similar to those described originally by Kaposi. They are found mainly in elderly males, particularly Jews of eastern European origin. The lesions begin slowly and insidiously around the ankle and slowly spread up the leg. They are very rarely responsible for the death of the patient.

2. Endemic cases are found in Azire, Uganda and Rwanda. In adults, males predominate but this form is also seen in children. Crops of vascular lesions develop on the skin, and may be associated with gross edema. Both cutaneous and visceral lesions may occur, and the prognosis is poor if there is extracutaneous involvement. The condition responds to chemotherapy.

3. Kaposi's sarcoma associated with infection with HIV

This variant was first recognized in 1979 when an epidemic of Kaposi's sarcoma was identified in the homosexual community in New York. Since that time it has become firmly associated with the later stages of infection with HIV. However, it is much commoner in homosexuals than in others at risk, such as drug abusers or hemophiliacs. Kaposi's sarcoma usually develops in the later stages of the disease.

4. Kaposi's sarcoma associated with non-HIV induced immunosuppression

This is seen in transplant patients and after cytotoxic chemotherapy for lymphomas. In transplant patients it is 150 times commoner than expected, and affected patients may be younger than in other types of Kaposi's. Both systemic and cutaneous involvement may occur, and the progress of the disease may be aggressive, causing the death of the patient. If it is possible to remove the immunosuppression, the lesions will regress.

The lesions of Kaposi's sarcoma can generally be divided into patch, plaque and nodular stages on the skin.

The pathological differential diagnosis in the early stages with include a simple angiomatous malformation, and on the lower legs a venous dermatitis. Arteriovenous malformations sometimes known as pseudo-Kaposi's sarcoma. Another condition to consider in the differential diagnosis is the bacillary angiomatosis of AIDS.

The early lesion is most likely to be confused with a benign vascular proliferation. It must also be distinguished from histiocytoma or from other types of sarcoma. Its evolution from a macular lesion and its characteristic colour, slow development and multifocal distribution make the diagnosis likely in most instances.

Recent studies indicate that an infectious agent closely related to gamma-herpesvirus is strongly associated with KS. Chang et al first identified DNA sequences of a new herpes virus in AIDS-associated KS tumor tissue. Subsequently the DNA sequence of this novel virus, named human herpes virus-8 (HHV-8) was also found in non-AIDS KS, including classic KS, endemic African KS and iatrogenic KS in immunosuppressed organ transplant recipients.

Diagnostic criteria:

1. Elongated spindle cells showing minimal atypia are separated by slits containing red blood cells and extravasated erythrocytes.
2. Numerous eosinophilic hyaline bodies is very helpful for the confirmation of the diagnosis, but it is not pathognomonic.
3. Strong immunoreactivity for Factor VIII-related antigen, CD34 or ulex lectin.

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Comparative Pathology Case 130

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Clinical history: The farm raised about 200 native chickens, aged about 2 months old. They were typical Marek's disease and sent to our teaching hospital for pathological diagnosis. One of the chickens showed several greyish-brown nodules scabs on the unfeathered parts of the head e.g. comb, wattles, eyelids and the corners of the beak.

Diagnosis: 1. Fowl pox
2. Marek's disease

Gross findings: Except the typical Marek's disease lesions (liver, spleen, heart and skin of wings with several gray nodules) we focused on the gray-white lesions on the unfeathered part of the head. Only one of the chickens the comb, wattles, eyelids and the corners of the beak with several greyish-brown nodules scabs. If the scab is removed early in its development, there is a moist, seropurulent exudate underneath covering a hemorrhagic granulating surface. When the scab drops off naturally, a smooth scar may be present; in mild cases there may be no noticeable scar.

Histopathological findings: Except the typical Marek's disease dermal and hair follicles lesions, the cutaneous lesion is hyperplasia of the epithelium and enlargement of cells, with associated inflammatory changes. Characteristic eosinophilic A-type cytoplasmic inclusion bodies (Bollinger bodies) are observable by light microscopy. The inclusion body may occupy almost the entire cytoplasm, with resulting cell necrosis.

Isolation and identification: Chicken embryo inoculation- A suspension of specimen emulsion from a dermal lesion was inoculated 9- to 12-day old chicken embryos on the chorioallantoic membrane (CAM) via the artificial air cell. The CAM revealed typical pox lesions 5-7 days after inoculation.

Electron microscopy observe- In negative staining, typical pox virus particles were observed in suspension of skin lesions emulsion.

Diagnosis criteria: Eosinophilic intracytoplasmic inclusion bodies in the ballooning change and hyperplastic epithelia.

Discussion: Pox is a common viral disease of domestic birds (chickens, turkeys, pigeons, and canaries) and has been reported in more than 60 species of wild birds representing 20 families. It is a slow-spreading disease characterized by the development of discrete, nodular, proliferative skin lesions on the unfeathered parts of the body (cutaneous form) or fibrino-necrotic and proliferative lesions in the mucous membrane of the upper respiratory tract, mouth, and esophagus (diphtheritic form).

In the mild cutaneous form of the disease, flock mortality is usually low but it may be high with generalized infection, when in diphtheritic form, or when the disease is complicated by other infections or poor environmental conditions.

Avian pox is not of public health significance. It does not generally affect mammals, however, a poxvirus isolated from a rhinoceros was characterized as fowl poxvirus.

Cutaneous lesions typical of avian pox must be confirmed by either histopathology (presence of cytoplasmic inclusions) or virus isolation.

Differential diagnosis:

1. Pantothenic acid or biotin deficiency in young chicks
2. T-2 toxin
3. *Trichomonas gallinae* in doves and pigeons may be mistaken for diphtheritic pox lesions
4. Infectious laryngotracheitis (ILT) in chickens may be mistaken for diphtheritic pox lesions

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Comparative Pathology Case 131

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Clinical history: A 81-year-old male, native of Taiwan and lived in 宜蘭鹿埔, suffered from itching over back skin for years with increased severity, recently. Past history revealed a previous operation due to intestinal necrosis, 10 years ago and another admission due to abnormal liver function and abdominal pain, 4 years ago. No history of exposure to radiation was noted but drank underground water instead of tap water for years was confirmed. There was no family history of skin cancer, xeroderma pigmentosum or basal cell nevus syndrome. On physical examination, two pigmented dome-shaped skin lesions over back were noted. Besides, generalized hyperkeratosis of skin was noted. Clinical work up including chest radiology, abdominal sonography & laboratory examinations showed no abnormal data, nor evidence of immunodeficiency. Under the impression of skin cancer, he received wide excision of the skin lesion with local skin flap reconstruction.

Diagnosis: Basal cell carcinoma (BCC), skin.

Gross findings: An oval piece of skin ($8.5 \times 4.6 \times 1.2$ cm) was submitted. There was a large, geographic, elevated lesion (5.6×4.4 cm in dimension) in the central portion with spots of brownish nodules to be noted. On serious sectioning, there were multicentric, nodular lesions in the superficial areas of skin.

Histopathological findings: Typical pictures of BCC were noted, including the multicentric superficial type BCC and nodular type BCC. The cancer islands showed peripheral basal cell palisading & mitotic figures were noted. Solar elastosis & solar lentigo were noted.

Immunohistochemistry stain: a.P53: positive nuclear staining (80%). b.bcl 2: strong,diffuse cytoplasmic labeling. c.CD44. d.CK. e.Her-2/neu. f.mucin stain.

Differential diagnosis:

1. squamous cell carcinoma: use bcl2 & Ber-EP4.
2. trichoepithelioma (TE): bcl2 & CD34 are reliable markers in D/D both TE & BCC
(TE shows negative bcl2 stain but positive TGF-beta result).
3. malignant melanoma: use epiluminescence microscopy to D/D with pigmented BCC.

4. Solar keratosis with SBCC: when small-punch or superficial shave biopsies (use bcl2).

Diagnostic criteria:

1. Infiltrative, morphea form BCC (IBCC):

- a. small islands of basaloid cells with angulated & "spiky" configuration.
- b. poor peripheral palisading & minimal stromal retraction.
- c. fibrotic & variably mucinous stroma.
- d. deep infiltration & poor circumscription.

2. Nodular BCC (NBCC):

- a. small to large aggregates of basaloid cells emanating from the epidermis or follicular structures.
- b. peripheral palisading, stromal retraction & mitoses are present.
- c. necrosis, apoptosis, calcification & mucin production are variable.

3. Superficial BCC (SBCC):

- a. small, discrete islands of basaloid cells present intermittently along the basal layer.
- b. prominent peripheral palisading & stromal retraction.
- c. peritumor mucin production.

Unique cytomorphologic features of metastatic BCC (FNA):

- 1. variable-sized & irregular-shaped cohesive epithelial clusters (tight clusters).
- 2. round to oval, small, monomorphic, hyperchromatic nuclei (crowded & overlapping nuclei).
- 3. bland chromatin pattern (finely granular chromatin).
- 4. sparse basophilic cytoplasm with indistinct border (high N/C).
- 5. inconspicuous nucleoli.
- 6. peripheral palisading of nuclei (on Diff Quik stain).

Discussion: BCC is the most common (skin) cancer in Caucasians & was originally described by Jacob in 1927. The incidence of BCC increased (Between 1971 and 1977, 18%). Each year there are about 750,000 to 930,000 new cases diagnosed in the US. BCC typically begins after age 30 & peak at age 70. Childhood case is rare in immunocompetent patients. BCC is made of transformed basal cells of human epidermis & spreads along the epidermis-dermis junction & often forms tumor cell mass that protrudes toward the dermal connective tissue with many branches. Antikeratin Ab have shown that BCC possess a keratin component not normally found in the basal layer & stain similarly to the lower part of the hair follicle epithelium. The location of BCC includes sun-exposed skin (85% on head & neck) & sun-protected skin (10-15%).

Anatomic areas with increased sebaceous gland are more likely to be involved.

Etiologic factors include UV & ionizing radiation, chemical carcinogens, possible, HPV infection & some BCC contain genetic mutation. Risk factors for BCC include exposure to arsenic, X-ray irradiation, coal-tar derivatives & UV light. Immunocompromised hosts (organ transplantation cases) are at increased risk for BCC because of impaired CMI & and increased

susceptibility to oncogenic viruses. Certain genodermatoses (albinism, XP, Rasmussen syndrome, Darier's disease. etc) have an increased incidence of BCC. Besides the environmental arsenic exposure, Chinese medications were also the source of arsenic poisoning & caused internal malignancies & BCC. There have been nearly 70 different histologic subtypes of BCC described, but only few subtypes have been shown to have clinical relevance: 1. Infiltrative type (infiltrative aggressive type)-all recurrent tumor. 2. nodular type (NBCC)-head & neck region (chronic sun-exposure). 3. superficial multicentric type (SBCC)-female & younger patients, occurring mainly on the trunk (male) & leg (female).

The high rate of BCC mandates that clinicians have a clear understanding of the tumor's patterns of presentation & behavior. Goal of any form of treatment includes establishment of the proper diagnosis & eradication of the lesion with a reasonable aesthetic result. While many BCCs demonstrate a single histologic subtype, roughly 40% change in their microscopic appearance at the subclinical extension (eg from nodular type to infiltrative type) & this finding has the potential to alter therapy.

Different tumors have been associated with BCC (coincidence of arise from the same origin of hair follicle). These include wart, neurofibroma, dermatofibroma, nevi sebaceus, seborrheic keratosis, malignant melanoma, squamous cell carcinoma (SQCC) & Keratoacanthoma. The differential diagnosis (D/D) includes SQCC, trichoepithelioma (TE), malignant melanoma.

A.Immunohistochemical stain (IHC) & B. electron microscopy were utilized for the D/D & the differentiation stage of BCC.

A. IHC: The common useful IHC stains include 1.p53 & 2.bcl2. 1.Increased expression of p53 has been found in the majority of BCCs. (SBCC: 50% (+); NBCC: moderate intensity with some peripheral accentuation; IBCC: intense p53 nuclear staining with peripheral accentuation). 2.Most BCCs demonstrate strong, diffuse cytoplasmic labeling with bcl2. 3.Other IHC stains include AE-3 & Ber-EP4 (show intense & diffuse positivity for BCC), CD44 (related to the metastatic potential), moesin (one of the ERM family members, directly associated with cytoplasmic domain of CD44). UEA1 & AE-1 show negative in BCC. Cytokeratins can also show the differentiation stage of BCC (K1, K2, K9, K10-11). B. EM: Electron microscopically, hemidesmosomes were poorly organized in BCC & hair follicles & the lamina densa was incomplete in BCC & follicular matrix.

The IHC & EM comparative studies revealed morphologic similarities between BCC & follicular matrix cells & the coexistence of melanocytes in the BCC tumor nests strongly suggest the differentiation of BCC toward the follicular matrix cells.

Metastatic BCC (MBCC): BCC usually invades locally but rarely metastasizes, occurring in only 0.0028-0.55% of all BCCs. Patients may present with lymphadenopathy, ulcerations, anemia, bone pain or muscle weakness, related to the site of metastasis.

Risk factors for MBCC include radiation, large & invasive tumors & a history of recurrence. The average survival time for localized lymph node metastasis in BCC is 3.6 years.

Treatment: 1.resection. 2.high-energy pulsed CO2 laser: for SBCC & CIS SQCC (with low risk of scarring). 3.Immunotherapy: eg Curcumin (a potent antioxidant & chemopreventive agent) induce a P53-dependent apoptosis in a dose-and time-dependent manner. 4.Electrochemotherapy (ECT): an effective alternative to excision for treatment of primary BCC.

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Comparative Pathology Case 132

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Clinical history: This 8-month-old male, Husky dog was presented to our hospital with more than 20 hard masses (5-8 cm in diameter), they were found subcutaneously over the whole body. These masses were observed by the owner 2-3 weeks ago. He had normal appetite, and treated with Amoxicillin, Prednisolone and eventually Oncovin. Masses got smaller after the treatment and some of them disappeared, but 6 months later, one new mass was observed on his right side of abdomen.

Diagnosis: Canine transmissible venereal tumor (CTVT)

Pathological findings: Grossly, more than 20 hard masses, ranging from 5 to 8 cm in diameter, were found subcutaneously over the whole body. No evidence of penetrating wound was seen over the nodules. They were in the superficial surface of flexor muscles of the forelimb and hindlimb and also the semitendinosus and sternohyoideus muscles. The cytoplasm was pale blue, agranular, many of the cells had few small, clear, round vacuoles near the distinct cell boundary. Microscopically, the nuclei of tumor cells were pale, with aggregated chromatin, a single, prominent eccentric nucleolus was seen. The nuclei were large. Mitotic figures were occasionally observed.

Discussion: Canine transmissible venereal tumor (CTVT) must be differentiated from other round cell tumors, namely mast cell tumor, histiocytomas, and lymphomas. Cytological features alone will not able to distinguish CTVT from other round cell tumors. Karyotyping and transmission studies have sometimes been used to confirm the diagnosis. Chemotherapy should then be the treatment modality since surgical removal alone usually resulting in recurrence. CTVT is the only contagious neoplasm of dogs that can be transplanted spontaneously with intact viable cells across major histocompatibility (MHC) barriers among dogs and even other Canidae such as foxes, coyotes and jackals. After two to four months of progressive growth, the tumor regresses in adults, but may metastasize in immunosuppressed dogs and neonatal pups. This tumor affects the external genitalia of both sexes and is transmitted by coitus, damaged mucosa or wounds. Experimentally, the CTVT can be transmitted by the of living tumor cells and considered to be an allograft. The mechanisms of how the tumor cells manage to overcome histocompatibility barriers so successfully for such a long period and yet succumb later are not known. Tumor cells have been shown to contain a tumor-associated antigen (TAA). In contrast to TAA expression, cells from progressor tumor lacked the expression of either Class I or Class II MHC antigens whereas 30 to 40% of those

from early regressor tumors expressed both Class I and Class II MHC antigens. This in turn may provoke additional immune reactions of the host to speed up the rejection process and resulted in the tumor mass to regress in two to three weeks. Tumor growth in adult dogs are smaller but infiltrated with greater numbers of lymphocytes than are the larger tumors growing in pups. The intensity of the lymphocyte blastogenic response during the progressive phase of tumor growth was shown to correlate with the course of the disease.

A high response could be detected in lymphocytes from dogs in which the CTVT eventually regressed, and metastatic spread occurred in dogs in which the lymphocytes were nonreactive. Elucidation and exploitation of the underlying mechanism which occurs in the body will be of great significance and practical importance.

Diagnostic criteria:

1. The nuclei of tumor cells contain in most time a single, prominent basophilic nucleolus.
2. The cytoplasm was pale blue, agranular, and has few small, and clear vacuoles near the distinct cell boundary.
3. The nuclei were large in relation to cell size.
4. Karyotyping and transmission studies have sometimes been used to confirm the diagnosis.

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<p style="text-align: center;">中華民國比較病理學會 第一次至第十五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	Pseudomembranous colitis	省立新竹醫院
14. Dog	Dirofilariasis	台灣省家畜衛生試驗所
15. Human	Pulmonary dirofilariasis	台北榮民總醫院
16. Squirrel	Toxoplasmosis	台灣養豬科學研究所
17. Pig	Toxoplasmosis	屏東技術學院獸醫學系

第三次比較病理學研討會病例（84年8月27日於國立台灣大學舉行）：

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

第四次比較病理學研討會病例（84年11月19日於新光吳火獅紀念醫院舉行）：

28. Human	Nocardiosis	台灣省立新竹醫院
29. Largemouth bass	Nocardiosis	屏東縣家畜疾病防治所
30. Dog	Demyelinating canine distemper 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 type 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 cryptococcal infection. Liver: metastatic carcinoma. Adrenal gland, right: carcinoma	三軍總醫院
48. Wild rodents	Adiaspiromycosis	國立台灣大學獸醫學系
49. Human	Echinococcosis	台北榮民總醫院
50. Piglet	Porcine cytomegalovirus infection	台灣省家畜衛生試驗所
51. Human	Pneumocystis carinii pneumonia	台北病理中心
52. Goslings	Aspergillosis	屏東縣家畜疾病防治所
53. Human	Intracavitary aspergilloma and cavitary tuberculosis, lung.	羅東聖母醫院
54. Human	Fibrocalcified pulmonary TB mixed actinomycosis and aspergillosis lung infection with abscess DM, NIDDM.	林口長庚紀念醫院
55. Broilers	Infectious laryngo-tracheitis	國立屏東技術學院獸醫學系

(Herpesvirus infection)

第八次比較病理學研討會（86年3月2日於台中榮民總醫院第一會議廳舉行）：

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

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

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

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

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|-------------|---|-----------------------|
| 76. Cat | Polycystic kidney bilateral and renal failure | 美國紐約動物醫學中心 |
| 77. Human | 1.Xanthogranulomatous inflammation with nephrolithiasis, kidney, right.
2.Ureteral stone, right. | 羅東聖母醫院 |
| 78. Chicken | Marek's disease in native chicken | 屏東縣家畜疾病防治所 |
| 79. Human | Emphysematous pyelonephritis | 彰化基督教醫院 |
| 80. SHR rat | 1.Glomerular sclerosis and hyalinosis, segmental, focal, chronic, moderate
2.Benign hypertension | 國防醫學院 & 國家實驗動物繁殖及研究中心 |
| 81. Human | Angiomyolipoma | 羅東博愛醫院 |
| 82. Human | Inverted papilloma of prostatic urethra | 省立新竹醫院 |

83. SD rats	Phagolysosome-overload nephropathy	國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 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	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
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

中華民國比較病理學會
第一次至第十五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
74.	Hepatocellular carcinoma induced by aflatoxin B1	Wistar strain rats	台灣省農業藥物毒物試驗所
81.	Angiomyolipoma	Human	羅東博愛醫院
82.	Inverted papilloma of prostatic urethra	Human	省立新竹醫院
84.	Nephrogenic adenoma	Human	國泰醫院
86.	Multiple myeloma with systemic Amyloidosis	Human	佛教慈濟綜合醫院
87.	Squamous cell carcinoma of renal pelvis and calyces with extension to the ureter	Human	台北病理中心
88.	Fibroepithelial polyp of the ureter	Human	台北耕莘醫院
90.	Clear cell sarcoma of kidney	Human	台北醫學院
93.	Mammary gland adenocarcinoma, complex type , with chondromucinous differentiation	Dog	國立台灣大學獸醫學系
94.	1.Breast, left, modified radical mastectomy, showing papillary carcinoma, invasive 2.Nipple, left, modified radical mastectomy, papillary carcinoma, invasive 3.Lymph node, axillary, left, lymphadenectomy, papillary carcinoma, metastatic	Human	羅東聖母醫院
95.	Transmissible venereal tumor	Dog	國立中興大學獸醫學系
96.	Malignant lymphoma, large cell type, diffuse, B-cell phenotype	Human	彰化基督教醫院
97.	Carcinosarcomas	Tiger	台灣養豬科學研究所
98.	Mucinous carcinoma with intraductal carcinoma	Human	省立豐原醫院
99.	Mammary gland adenocarcinoma, type B, with pulmonary metastasis, BALB/cBYJ mouse	Mouse	國家實驗動物繁殖及研究中心
100.	Malignant fibrous histiocytoma and paraffinoma	Human	中國醫藥學院

	102.	Pleomorphic adenoma (benign mixed tumor)	Human	佛教慈濟綜合醫院
	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
	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	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
細菌	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
	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
病毒	113	Tuberculous meningitis	Human	羅東聖母醫院
	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
黴菌	130.	Fowl pox and Marek's disease	Chicken	國立中興大學獸醫學系
	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
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 <i>Toxocara canis</i> larvae migration	Dog	臺灣養豬科學研究所
原蟲	4	Cryptosporidiosis	Goat	台灣養豬科學研究所
	15	Amoebiasis	<i>Lemur fulvus</i>	台灣養豬科學研究所
	16	Toxoplasmosis	Squirrel	台灣養豬科學研究所
	17	Toxoplasmosis	Pig	屏東技術學院獸醫學系
	51	<i>Pneumocystis carinii</i> pneumonia	Human	台北病理中心
	57	Cecal coccidiosis	Chicken	國立中興大學獸醫學系
	65	Cryptosporidiosis	Carprine	台灣養豬科學研究所
立克次體	70	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
	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

中華民國比較病理學會第一次至第十五次比較病理研討會

各單位提供討論病例次數統計

(八十三年十月三十日至八十八年四月十一日)

單位名稱	提供討論病例數
台灣養豬科學研究所	11
國立台灣大學獸醫學系	11
國立中興大學獸醫學系	11
美國紐約動物醫學中心	11
國家實驗動物繁殖及研究中心	8
屏東縣家畜疾病防治所	7
花蓮佛教慈濟綜合醫院	7
省立新竹醫院	6
長庚紀念醫院	5
台北病理中心	5
羅東聖母醫院	5
新光吳火獅紀念醫院	4
國立屏東科技大學獸醫學系	4
彰化基督教醫院	4
省立豐原醫院	3
台北榮民總醫院	3
台北醫學院	3
台北耕莘醫院	3
頭份為恭紀念醫院	2
台中榮民總醫院	2
台灣省家畜衛生試驗所	2
中國醫藥學院	2
三軍總醫院	2
羅東博愛醫院	2
台北市立仁愛醫院	1
國泰醫院	1
台北馬偕醫院	1
華濟醫院	1
大甲李綜合醫院	1
高雄醫學院	1
國防醫學院	1
國立嘉義農專獸醫科	1
台灣省農業藥物毒性試驗所	1
合 計	132

會員資料更新服務

各位會員：

您好！如果您的會員資料有更新或誤刊情形，麻煩

您填妥表格後寄回學會秘書處或電話連絡：

中華民國比較病理學會秘書處

苗栗縣竹南郵政信箱 23 號

病理生物系 邱慧英 小姐

Tel: (037) 672352轉505

Fax: (037) 687803

e-mail address: hic01@mail.prit.org.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）寄苗栗縣竹南郵政信箱23號，中華民國比較病理學會秘書處王□真小姐，電話：037-672352轉506，傳真037-687803。