第三十三屆生物醫學聯合學術年會

論文投稿規則、範例及摘要表格

一、所有欲發表之論文投稿截稿日期：**2017 年 12 月 31 日 17:00PM 止**。逾時不予處理。

二、已在其他期刊發表過的文章，恕不採用。

三、文字：摘要內容限以英文撰寫。題目需英文。作者姓名須中、英文並列（責任作者加*）。

四、字數：內文限英文 2500 字元（含空格）以內，不得跨頁；未按規定者不予接受。

五、字體及行距行高：中文 – 標楷體；英文 - Times New Roman；字體大小 - 12 號字。
行距 – 最小行高，行高– 12；文件格線被設定時，貼齊欄位勿打勾。
英文篇名每字字首均統一為大寫。

六、所有投稿論文一律採線上繳交

請注意！凡是有意願參加「大會主題口頭論文競賽」或「學會口頭論文競賽」者，除了上傳摘要 word 檔之外，仍然需要以 PDF 的形式上傳 manuscript，以利評審進行審查，檔案大小不可以超過 20 MB。

(1)「大會主題口頭論文競賽」manuscript 需有指導教授推薦信函之合併 PDF 檔案。

(2)「學會口頭論文競賽」依各學會規則。

存檔：檔名為 2018 學會名稱-第一作者姓名.（例：2018 細分學會-陳小美）。

七、投稿方式：
1. 至網站線上投稿區下載投稿專用表格
2. 詳細填寫投稿資料並上傳摘要檔案
3. 至網站投稿名單確認檔案上傳成功

八、摘要撰寫格式範例：（下一頁：投稿摘要表格。填寫完畢後請另存下頁表格，進行上傳。）

1.英文題目 → 2.中文姓名 → 3.英文姓名 → 4.英文服務單位 →	***Xist* reduction in breast cancer upregulates AKT phosphorylation via HDAC3-mediated repression of PHLPP1 expression** 黃彥淞[1]，張哲菖[2]，李思碩[3]，周玉山[1,3]，施修明[*1,2,3] Yen-Sung Huang[1], Che-Chang Chang[2], Szu-Shuo Lee[3], Yuh-Shan Jou[1,3], Hsiu-Ming Shih[*1,2,3] [1]Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan [2]Graduate Institute of Translational Medicine, College of Medical Science and Technology, Taipei Medical University, Taipei, Taiwan [3]Program in Molecular Medicine, National Yang-Ming University and Academia Sinica, Taipei, Taiwan
5.摘要內文： → 一律用英文。不分段。2500 字元（含空格）以內。右方為可參考樣本。	Long noncoding RNAs (lncRNAs) dysregulated in cancer potentially play oncogenic or tumor-suppressive roles. While the X inactivate-specific transcript (*Xist)* lncRNA is important for X-chromosome inactivation in female cells, very little is known about the role of *Xist* in human breast cancer in modulating cellular pathway(s). Here, we show that *Xist* expression is significantly reduced in breast tumor samples and cancer cell lines. *Xist* knockdown or overexpression resulted in increased or decreased levels, respectively, of AKT phosphorylation and cell viability. Further studies revealed an inverse correlation between *Xist* and phospho-AKT levels in breast cancer samples. Additionally, *Xist* knockdown-elicited increase of cell viability was attenuated by AKT inhibitor. These results suggest that *Xist* negatively regulates cell viability via inhibition of AKT activation. Interestingly, decreased *Xist* expression in breast cancer samples was associated with reduced levels of *Jpx* RNA, an lncRNA that positively regulates *Xist* promoter activity. Accordingly, *Jpx* knockdown enhanced AKT activation and cell viability. We also demonstrate that knockdown of *Xist* or SPEN, an intermediator protein to link *Xist*, SMRT co-repressor and HDAC3 complexes for X-chromosome inactivation, decreased expression of PHLPP1, a phosphatase to remove AKT phosphorylation, via increased HDAC3 recruitment to the PHLPP1 promoter, correlating with increased AKT phosphorylation. Our findings elucidate the tumor suppressor role of *Xist* in breast cancer and provide the molecular basis of *Xist* in downregulating AKT activation.