



## The 65th ASH Annual Meeting Abstracts

## POSTER ABSTRACTS

**623.MANTLE CELL, FOLLICULAR, AND OTHER INDOLENT B CELL LYMPHOMAS: CLINICAL AND EPIDEMIOLOGICAL****Inhibition of CDC20 Suppressed the Development and Progression of Mantle Cell Lymphoma**Yingtong Chen<sup>1</sup>, Ping Yang<sup>1</sup>, Jing Wang<sup>1</sup>, Shuang Gao<sup>1</sup>, Xiaoyan Ke<sup>1</sup>, Hongmei Jing, MD<sup>1</sup><sup>1</sup>Peking University Third Hospital, Beijing, China

**Background:** Cell division cycle 20 homologue (CDC20) is an important factor regulating the cell mitosis phase. CDC20 is highly expressed in several types of cancers, which is related to the clinicopathological characteristics and prognosis of cancer patients. This study aimed to explore whether CDC20 could affect the development and progression of mantle cell lymphoma (MCL) and the underlying mechanism.

**Methods:** The expression level of CDC20 was detected in peripheral blood mononuclear cells (PBMCs), bone marrow mononuclear cells (BMNCs) and pathological tissues of MCL patients and MCL cell lines (Z138, Mino, Rec1). The CDC20 inhibitor apcin was used to evaluate the effect of CDC20 on the cell phenotypes of MCL cells. The changes in cell proliferation, apoptosis, cell cycle, cell migration and invasion were determined using CCK-8, flow cytometry, and Transwell assays, respectively. The anti-tumor effect of apcin were investigated *in vivo* in the Z138-driven xenograft tumor model. RNA-seq was performed to explore the pathways that changed in MCL cells after apcin treatment, and then these possible changes were verified *in vitro* and *in vivo* by western blot.

**Results:** CDC20 was overexpressed in PBMCs, BMNCs, and pathological tissues of MCL patients and MCL cells compared with their respective control group. In Z138, Mino, and Rec1 cells, apcin could inhibit cell proliferation, migration, and invasion, promote cell apoptosis, and induce cell cycle arrest. Moreover, apcin confirmed its safety and anti-tumor activity *in vivo*. In apcin-treated MCL cells and mice, the PI3K/AKT pathway was significantly inhibited (Figure 1).

**Conclusions:** This study confirms the essential role of CDC20 in MCL tumorigenesis. Anti-tumor therapy targeting CDC20 is expected to be a powerful treatment strategy for MCL patients.

**Disclosures** No relevant conflicts of interest to declare.

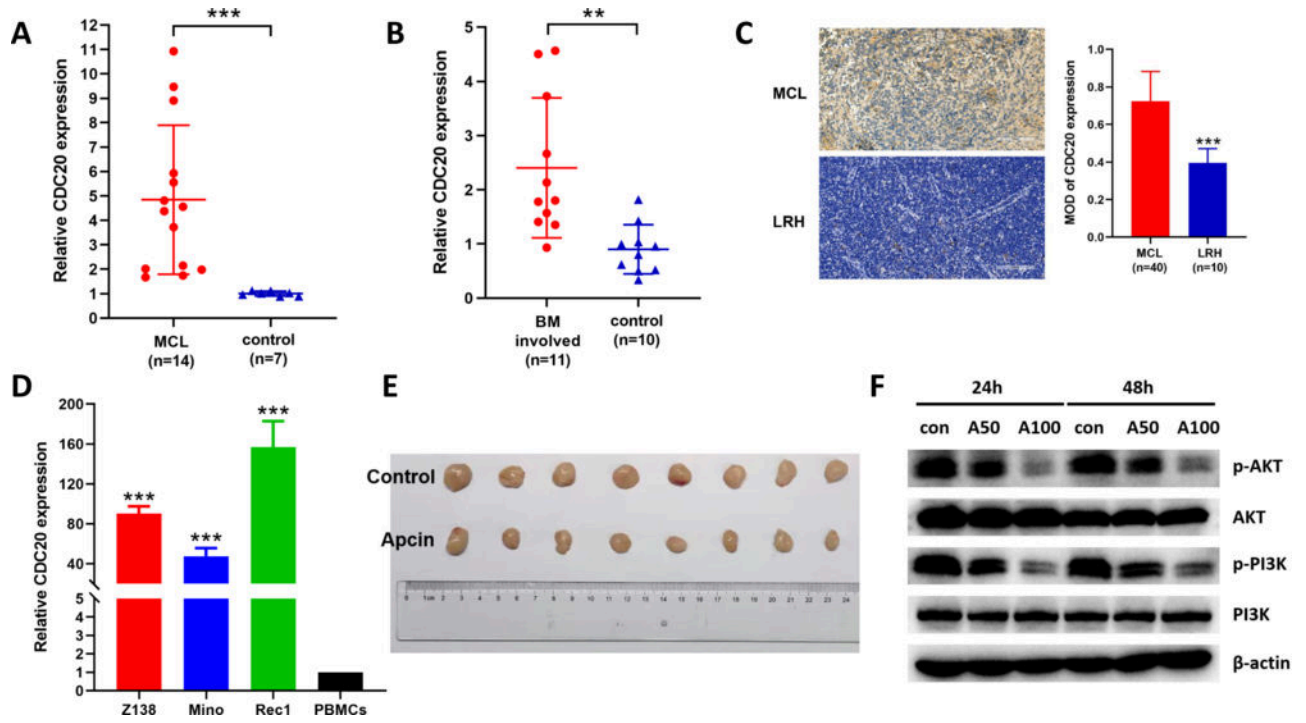


Figure 1

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