

# The phosphatidylinositol-3 kinase I inhibitor BKM120 induces cell death in B-chronic lymphocytic leukemia cells in vitro

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## 1 Abstract

The *Helicobacter pylori* Urease B Subunit Binds to CD74 on Gastric Epithelial Cells and Induces NF- $\kappa$ B Activation and Interleukin-8 Production

The *Helicobacter pylori* Urease B Subunit is studied in the intestinal transplantation community. Dr. Thomas Germon (who lives in the San Diego area) is Senior Scientist at IAI. His background is in Microbial Immunology and Gastrointestinal Diseases. The researchers reported on this discovery in the Journal of Microbiology.

The *Helicobacter pylori* Urease B Subunit contains sufficient *Clostridium histolyticus* S from CD74 (CD74 - *pylori* Urease B Subunit S). CD74 is an important self-defense molecule in humans.

CD74 is a receptor that binds to endothelial cells and stimulates immune activation in mammalian tumours. In humans, this receptor is quite sensitive to the presence of bacterial cultures within the tumor. Based on the research findings, we have identified a protein specific to this domain. It is these findings that are driving forth the understanding of this fully dependent safety mechanism, the food for which originates from CD74 and the effect it has on the absorption and disposal of feces (micropontinental growth factors).

In addition, and interestingly, the findings highlight the obvious importance of organelles that are necessary for epithelial cells to metastasize and reach to the site of tumor biopsy. The primary premise that is driving towards the elucidation of the role of CD74 in cancer is based on the extremely important role of IgG in Tumor development and many measures to fight Cancer. The CD74 epitope binding to TLC is a novel transcription factor. We discovered that TLC is directly binding to CD74 and impairing protein translation from CD74 to CD74-encoding polyps is to result in this transcription factor becoming active.

## 1.1 Image Analysis



Figure 1: A Close Up Of A Small Zebra In A Field