$Q LGHDO VDPSOH UHVHDUFK ZRXOG EH VWDWHPHQW ZLWK WKH JXLGDQF
5,6(PHQWRU JLYLQJ D JHQHUDO EDFNJURXQG WR IUDPH WK
\RX ZRXOG EH LQYHVWLJDWLQJ VWUDWHJLHV IRU REWDLQL
\RXU UHVXOWV ZRXOG SURYLGH DQ LPSDFW LQ \RXU ILHOG

Research Project Description

On a broad scale, my research project on hCMV with Dr. ; focuses on discovering WKH URO

H RI FKURPDWLQ VWUXFWXUH LQ WKH YLUDO OLIH F\FOH RI KXPDQ F\WRPHJDORYLUXV

infection. hCMV enters the nucleus of early myeloid cells and endothelial cells, where it gets chromatinized and genetically repressed. This is the called the latent phase of the hCMV life cycle, where few immediate early genes are expressed, but no viral particles are made. Previous research on hCMV reveals that a cellular protein complex known as PRC2 regulates the chromatinization of the hCMV genome. However, hCMV somehow circumvents complete repression, as indicated by some expression of immediate early genes. The hCMV genome is eventually able to rid itself of a closed chromatin structure through unknown processes, resulting in a lytic infection defined by considerable viral particle assembly and cell death. Therefore, it is believed that the chromatinization regulation of the

of cells that are models for eliminated JARID2 protein. For cell lines, we will use THP1, NT2D1, and MRC5. THP1 and NT2D1 are cell lines that are an effective model of a latent infection. I will test THP1 and NT2D1 in parallel to see which is more efficient at maintaining