



**CALIFORNIA STATE SCIENCE FAIR  
2012 PROJECT SUMMARY**

<b>Name(s)</b> Gwendolyn G. Lee	<b>Project Number</b> <b>S0512</b>
<b>Project Title</b> <b>Impact of vRNA and M1 Protein on the Structure and Budding Mechanism on the Influenza A Virus</b>	
<p style="text-align: center;"><b>Abstract</b></p> <p><b>Objectives/Goals</b> The purpose of this research is to elucidate the structure and budding mechanisms of the influenza A virus using cryo-electron tomography. This project also aims to compare budding in mutant strains with that in wild-type viruses in order to highlight budding defects that may serve as potential targets for future antiviral therapy. With this information, vaccines and medications that target specific components of the budding process, thereby inhibiting viral proliferation, can be developed.</p> <p><b>Methods/Materials</b> The influenza virus samples were prepared using the influenza A/Udorn/72 (H3N2) strain, as well as the influenza A/WSN/33 (H0N1) strain. The budding mutant was created using the M1[R101A] mutation in the WSN strain. The samples were maintained in Madin-Darby canine kidney (MDCK) cells. Samples were flash-frozen in preparation for cryo-electron microscopy. Cryo-electron tomography (cryoET) was used to analyze these virus samples.</p> <p><b>Results</b> The tomographic reconstructions of the influenza A/Udorn/72 (H3N2) strain display three major morphologies: filamentous particles, spherical particles, and chains of particles resembling beads on a string. Some particles were completely devoid of vRNA. The aberrant chains of particles were linked by viral membranes, forming junction points, or by interlocked surface proteins. The reconstructions of the influenza A/WSN/33 mutant strain reveal a major aberration, in which the middle section of certain particles collapsed, forming only a thin layer.</p> <p><b>Conclusions/Discussion</b> The lack of vRNA in certain virions suggest that RNPs are not required in the budding process. The chains of particles indicate budding "hot spots" that are particularly conducive to budding. These locations can serve as targets for antiviral drugs. The M1 protein appears to play a role in the structure of the virus. Furthermore, there appears to be a correlation between the presence of M1 and that of surface proteins.</p>	
<b>Summary Statement</b> In order to elucidate the structure and budding mechanism of the influenza A virus, samples of wild type and mutant influenza virus were cultured, imaged using cryo-electron microscopy, and analyzed using tomography reconstruction technique	
<b>Help Received</b> Dr. Hong Zhou allowed me to use laboratory equipment and taught me how to use computer reconstruction software. Dr. Nayak allowed me to use his laboratory space and equipment.	