Undergraduate Research Symposium May 18, 2018 Mary Gates Hall

Online Proceedings

Session 1C

Blurred Realities, Ethical Questions and Media Critiques

Session Moderator: Barbara Miller, Art History, Western Washington University

MGH 171

12:30 PM to 2:15 PM

* Note: Titles in order of presentation.

You Are What You Eat: Cultivating Conscious Consumption through Informed Consumer Choice

Kathryn C. Kavanagh, Senior, Business Administration (Marketing), Community, Environment, & Planning
Mentor: Kelly Hostetler

Education on the current state of the food system can motivate individuals to create the food systems and generate demand for products they wish to see. With the idea that we are what we eat and how we consume, real food is the best medicine—for individual health, for society, and for the planet. The power of individual freedom does not mean anything goes, but rather informed, responsible choice that transcends to the global aggregate. Thus, respectful and healthy food systems begin with the individual, and have the potential to bring peace to communities and heal human relationships with land and each other, both locally and globally. Knowledge is power, and first starts with attention and awareness. Given the severe lack of transparency and multitude of socioenvironmental externalities within the industrialized food system, there is a need for adequate consumer education on practices and operations. Information on the consequences of these processes can influence attitudes to shift demand and create new vibrant systems. Here, the problem is the solution. To promote holistically healthy and nutritious systems, I created a disruptive advertisement campaign to highlight truths of the industrial system and its social and environmental externalities to inform consumers on cost and value of the products they are purchasing. This project juxtaposed the concepts of ”farm to table” versus ”firm to table,” in additon to the applications of integrative sensory marketing tactics and culinary storytelling. Through the combination of art, music, poetry, and film, I sought to motivate people to think critically about the power of individual choice and what is behind a brand.

Poster Session 3

MGH 206, Easel 173

2:30 PM to 4:00 PM

Interactions between the HIV-1 Env Glycoprotein and DC-SIGN are Influenced by Env Glycan Presentation

Adam Nguyen, Senior, Biochemistry
Mentor: Kelly Lee, Medicinal Chemistry
Mentor: James Williams, Medicinal Chemistry

Interactions between HIV-1 and dendritic cells (DCs) have been suggested to play a role in HIV-1 pathogenesis. DCs are antigen presenting cells that take up, process, and present foreign material to T-cells. Previous research suggests that HIV-1 can exploit this process by binding to DCs, allowing HIV-1 virions to relocate to lymph nodes where infection of T-cells can occur. HIV-1 mediates this interaction through binding of the HIV-1 envelope glycoprotein (Env) to DC-SIGN, a glycan binding protein located on the surface of DCs. Env contains a high amount of oligomannose glycans, enabling Env to act as a binding partner for DC-SIGN. Due to high sequence variation, HIV-1 strains exhibit variability in glycan presentation. A gap remains in our knowledge of where DC-SIGN binding occurs and how glycan presentation across various isolates modulates these interactions. In this study, we applied a combination of biophysical and structural approaches to characterize Env:DC-SIGN interactions across multiple HIV-1 strains. Biolayer interferometry experiments measuring binding affinity demonstrate that DC-SIGN binds with strong affinity towards Env from different isolates. This suggests that DC-SIGN is capable of recognizing different oligomannose glycan presentations and may not have a well defined epitope. However, preliminary results using electron microscopy illustrate that Env glycoprotein structure can be disrupted and altered via binding to DC-SIGN. This appears to be an isolate-specific response, as some strains remain unchanged. Using single particle approaches, we identified potential epitopes in heavily glycosylated regions of Env where additional densities are attributed to DC-SIGN. We infer that in addition to binding HIV Env, DC-SIGN may shield key neutralizing epitopes within these glycosylated regions. Our results provide further insight into the interactions that occur between HIV Env and DC-SIGN, and suggest a mechanism where HIV-1 hijacks DC's normal immune function allowing HIV-1 trafficking while shielding neutralizing epitopes.