UC Merced Center for Excellence on Health Disparities Undergraduate Student Project Abstracts

Year 3 Cohort

Examining the Validity of Public Data to Describe Unincorporated Areas: A Case Study with a Rural Latino Community in California

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Unincorporated areas nationwide house populations with socio-economic challenges correlated with many disparities in quality of life. In California, an estimated 438 unincorporated areas house over 7 million people, or approximately 1 in 5 Californians. Publicly available data sources are important first steps in the analysis of the health of unincorporated areas. However, limited research exists to understand the validity of public secondary data for unincorporated communities. A case study using a small rural town in the San Joaquin Valley of California (Lost Hills) was conducted to examine the validity of public data in describing the population.

Existing critiques of the U.S. Census and the American Community Survey (ACS) were used to identify threats to their internal validity. Subsequently, these threats were examined using the data available for Lost Hills to understand their relevance for this unincorporated area. The findings indicated that both the Census and the ACS may undercount the overall population of Lost Hills, in particular the Latino population. Limited English proficiency and lack of citizenship also threaten the valid count of the largely immigrant and migrant population of Lost Hills. The data and methods of the ACS may offer a more valid representation of this unincorporated community. These findings may be generalizable to other small communities with a proportionally larger percentage of Latino residents.

Contribution of adipose RAS proteins to metabolic syndrome in OLETF model rats

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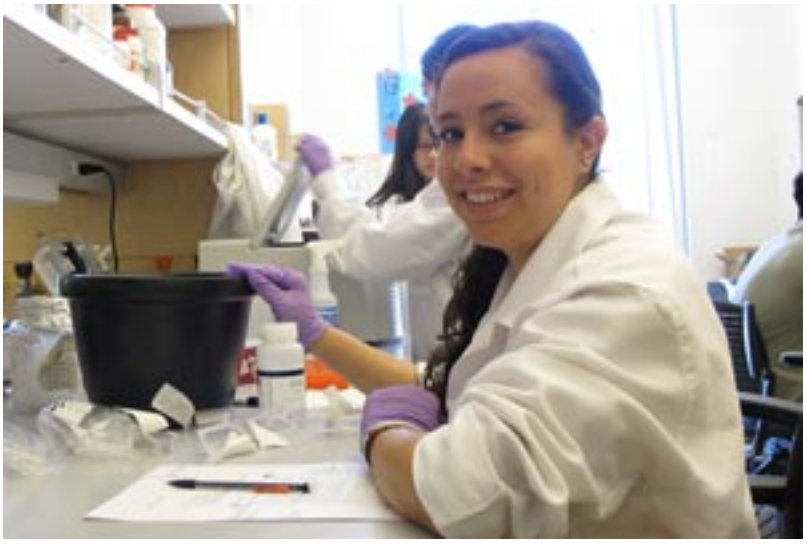
Activation of the renin-angiotensin system (RAS) has been shown to contribute to metabolic syndrome.Â Hyptertension is a key component of metabolic syndrome and is regulated by RAS.Â A preferable model used to evaluate the effects of such conditions is the Otsuka Long-Evans Tokushima Fatty (OLETF) rat because their pathogenesis closely resembles that of the progression of human insulin resistance, metabolic syndrome, and type 2 diabetes. However, the contribution of adipose RAS to metabolic syndrome is not yet well defined.

To address the hypothesis that adipose RAS activation is increased with insulin resistance, rats were divided into two groups (n=10-12/group): 1) lean strain-control LETO 2) Obese insulin resistant OLETF.Â Systolic blood pressure (SBP) was measured weekly for 24 weeks and insulin resistance index (IRI) was determined at 9 and 24 weeks. Tissue samples were collected from each group (n=5-6/group) at 15 and 24 weeks. Mean SBP from OLETF was 28% and 27% greater compared to that of the LETO after 15 and 24 wk respectively. Mean IRI was greater at both time periods as well, and we exacerbated in 24 wk OLETF compared to 15 wk. By 24 wk, adipose angiotensinogen (angiotensin precursor) expression had increased in 24 wk OLETF suggesting that activation of adipose RAS was increased. The data suggest that increased activation of RAS may contribute to the increased metabolic disorders (SBP and IRI) commonly associated with metabolic syndrome.



Chlamydia pneumoniae in Gingival Epithelial Cells Respond by Secreting Proinflammatory Cytokines

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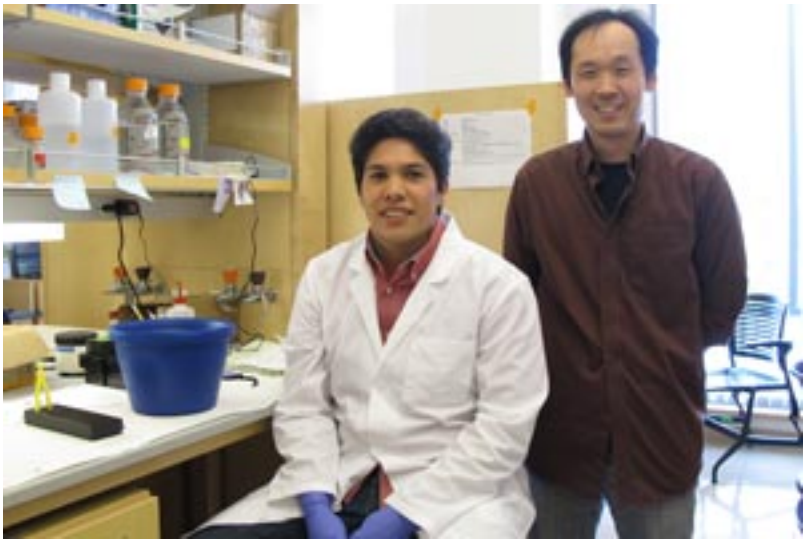
Chlamydia is one of the worldâ€™s most common sexually transmitted disease as well as the leading cause of blindness. Recently there has been more of an interest in Chlamydia pneumoniae, an intracellular bacteria that infects humans and has been known to cause lung complications, bronchitis, and pharyngitis. However, little is known about whether if C. pneumoniae can cause oral disease

Chlamydia infections are usually characterized by inflammation, which is an innate immune response. NLRP3 inflammasome is one of the most well studied type of inflammasome due to its keen immune response to microbial molecules. NLRP3 inflammasome triggers the secretion of proinflammatory cytokines acting as a defense against infection. Knowing this, there is a belief to be a connection between NLRP3 inflammasomes and gingival epithelial cells. Gingival epithelial cells (GEC) are beneficial when studying the oral mucosa and in order to understand if C. pneumoniae is causing oral diseases we will study infected GEC.

Through this study we hope to see that the C. pneumoniae will adhere to the GEC which then can help determine if C. pneumoniae will activate NLRP3 inflammasome during infection. Respectively, there is an expectation to see growth inhibition of C. pneumoniae in the gingival epithelial cells through an increase of proinflammatory cytokines.



Developing C2C12 Cell Model for IBMPFD to Study TDP-43 and Ubiquitin Pathology

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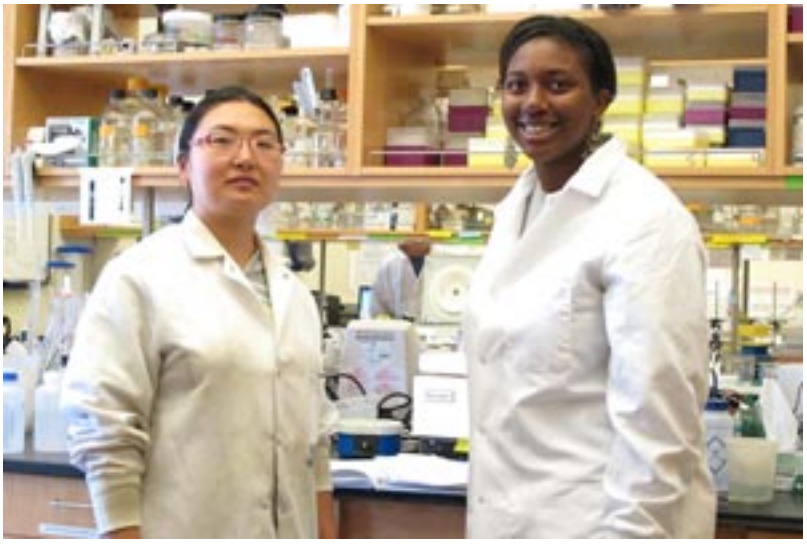
Inclusion body myopathy associated with Pagets disease of bone and frontotemporal dementia (IBMPFD) is a degenerative genetic disorder caused by mutations in the valosin-containing protein (VCP) gene. Approximately, 80% of patients carrying VCP mutations develops inclusion body myopathy. Although the molecular mechanisms underlying IBMPFD pathology remain unknown, mislocalization of TAR DNA binding protein 43 (TDP-43) in cytoplasm and the accumulation of ubiquitinated proteins have been implicated as important culprits in the mutant VCP-mediated myodegeneration. We sought to investigate mechanisms by which disease-associated mutations in VCP triggered TDP-43 redistribution to cytoplasm and the accumulation of ubiquitin inclusions in the cell. Currently, there is no in vitro model that recapitulates the disease phenotypes. In this study, we developed a cell culture model overexpressing human wildtype or disease-relevant mutant VCP.Â Murine myoblast C2C12 cells were used as a model for skeletal muscle cells and transfected with disease-causing mutant forms of human VCP, R155H, A232E, and a dominant-negative mutation, K524A.Â We established optimal conditions for the transfection in C2C12 cells and examined pathological hallmarks using immunocytochemical and biochemical approaches, both of which were optimized in the laboratory.

Our preliminary results suggest that mutant VCP expressions slightly altered TDP-43 localization in the cytoplasm and aggregation of ubiquitinated proteins, but accurate comparisons to wildtype forms have yet to be quantified. Nuclear and cytoplasmic fractionations were achieved, and will be used to analyze distribution of TDP-43 and ubiquitin aggregates. Taken together, we have developed optimal techniques that will be used to study IBMPFD pathology in C2C12 cells.



Optimizing Existing HIV Entry Inhibitors

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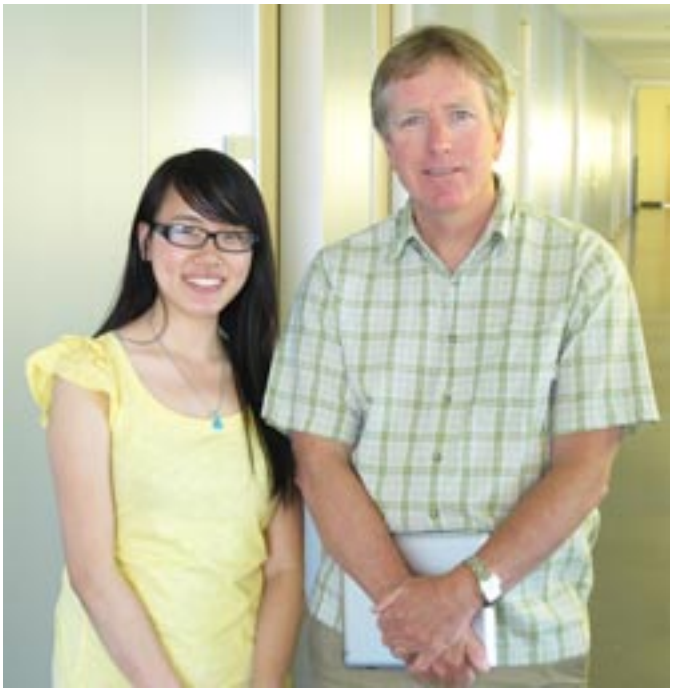
HIV entry inhibitors have been a groundbreaking discovery in the search for successful HIV drugs, and in particular to stop initial infection. Currently, Fuzeon (T20) is a drug approved by the FDA for treating and decreasing viral load in HIV-infected persons, which has the mechanism of binding to HIV and preventing HIV entry into cells. Cell entry is mostly due to two major glycoproteins on HIV-1â€™s surface: gp41 and gp120. Since the introduction of T20, a first-generation inhibitor, many laboratory and wild-type strains of HIV have become resistant to this specific inhibitor. This has led to further studies implementing different proteins or molecules to identify a stronger inhibitor to block HIV entry.

One method of stopping HIV is by blocking gp120. Griffithsin, a protein derived from algae, has been discovered as a very potent entry inhibitor. It acts by targeting the glycoprotein gp120, and, works effectively on its own, or coupled with another inhibitor. In our previous study, we discussed the success of the chemokine variant inhibitor 5P12-linker-C37. By blocking HIV entry into the host cell by binding gp41 and host cell proteins, these powerful yet inexpensive inhibitors potentially provide a remedy for the ever-increasing number of HIV infections. With the recent success of a new third generation gp41-binding fusion inhibitor, T1144 (Pan, C., Cai, L. (2011) *Journal of Biological Chemistry)*, newer inhibitors seem to carry potential for an improved anti-HIV drug. By linking Griffithsin with T-1144, and 5P12 RANTES with T-1144, it is hypothesized that HIV inhibition could be improved.



Comparing Indicators of Quality Early Care and Education from State and National Sources

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Research shows that children who experience higher quality early care and education programs (ECE) perform significantly better in school than do children from lower quality programs. In order to objectively assess and improve ECE, a quality rating and improvement system (QRIS) with research-supported quality indicators is crucial. California is in the process of developing QRIS, following 23 states nationwide, which have already adopted statewide QRIS. A comparative analysis of national and state indicators was conducted to understand the potential importance of existing indicators for California's QRIS. Indicators from a 2010 national compendium of 23 states' QRIS and California's CAEL QIS 2010 report were organized into a matrix and reviewed to identify similarities and differences between California and other states. Five of California's proposed indicators (ratios and group size, teaching and learning, family involvement, staff education and training and program leadership) matched 12 of 13 indicators identified nationally. A nationally-compiled indicator on accreditation by a national accrediting body was not included in the California set.

One potential strength in the California QRIS that was largely absent nationwide was the definition of quality criteria for different types of ECE (preschool and family child care). This comparison of nationwide and California indicators of quality ECE demonstrated that the majority of ECE quality indicators were consistent between the two reports. Results from this study may inform conversations on the development of California's QRIS and application of the quality indicators to county- and city-level efforts to ensure high quality early care and education.