Graduate School of Biomedical Sciences

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Children with severe burns are known to have a massive rise in their metabolic rate, also associated with significant muscle wasting. We found that a three-month aerobic and resistance exercise training program was safe and did not exacerbate their metabolic rate, whilst helping to improve their muscle mass and strength.
I investigated the negative effect of high fat maternal nutrition and preeclampsia on the offspring's cardiovascular health as an adult. Pre-pregnancy weight was also shown to be a strong risk factor for preeclampsia. I showed that maternal obesity strongly influences an offspring's future health, and if present with preeclampsia has an enhanced detrimental effect.
I examined the difference in survival rates among black and white women with late stage breast cancer. I found that disease complications directly related to the advanced cancer and its treatment, immobility and depression do not explain the observed differences in survival between groups.
I studied the potential effectiveness of a Nutrition Education Program on food choices and dietary self efficacy (DSE) for adolescents at risk for Type 2 diabetes. While the Program did not alter the food selection by these at risk adolescents, there was a highly significant improvement in their DSE.
KLF4 is a protein involved in maintaining the structure of the normal human colon, and is important in the development of colorectal cancer. I focused on how KLF4 regulates expression of genes as well as how it modulates a signaling pathway within cells that is critical in the development of colorectal cancer.
Titin is a mechanically important, chain-like protein in muscles. Some segments of titin are tightly wound up like coiled rope. Some of these ‘coils’ require stronger tugs to unravel than others. I used several computer programs to compare the ‘coils’ and found differences that might explain why some require stronger tugs to unravel.
Lower muscle mass is associated with poorer outcomes in many clinical conditions and with aging. My research focuses on understanding how diet modification and exercise can be used to increase or maintain muscle mass during such conditions to improve clinical outcomes and the overall quality of life.
Anthrax toxins are capable of disrupting cell signaling. This disruption has serious effects on the immune system. I determined that these toxins altered the properties of B cells. My results showed that these toxins were an important way that anthrax disables the immune system in producing disease.
Bacterial pneumonia has a disproportional effect on the elderly. Hospitalizations and pneumonia inpatient mortalities are higher among males -75+. Among this group, Hispanics have more hospitalizations than other ethnic groups. Increasing % of Hispanics in a county led to lower hospitalizations and pneumonia inpatient mortalities. Other influencing factors are median income and hospital size.
Stroke mortality rates are reported to be lower for Hispanics than non-Hispanic Whites. My research explored the role of immigrant status in stroke incidence and mortality. I also investigated the impact of cause of death ambiguity and the role of the misreport of ethnicity on death certificates.
Neuroblastoma is a devastating cancer that usually occurs in children under age three. My research focused on how the hormone GRP uses particular cancer signaling pathways to regulate neuroblastoma growth. This research has led to a better understanding of the influence of hormones on cancer and may lead to improvements in neuroblastoma therapy.
Burned children may be predisposed to developing hyperthermia during exercise. We examined core body and skin temperature during exercise in the heat. While temperature responses are altered, hyperthermia did not occur. However, these children showed intolerance to exercise in the heat.
My research project used national nutrition survey data to compare dietary patterns across race/ethnic subgroups of the US population and to examine dietary influences on race/ethnic differences in type 2 diabetes.
HIV infects the brain and can cause cognitive impairment. HIV-infected brains showed changes in proteasomes, which are responsible for protein degradation. These changes, associated with HIV encephalitis, cognitive impairment, and viral loads, were shown in neurons. Findings suggest that the proteasome changes contribute to HIV-associated cognitive impairment.
Yersinia pestis is the bacteria that causes the bubonic plague. Rickettsia typhi, another bacteria, causes a murine typhus. Both are transmitted to humans and other mammals by fleas. This research investigated mechanisms used by these bacteria to enhance their transmission.
In my research I examine the reasons why nerves cannot reconnect in the spinal cord after they are injured. I studied impaired bladder function after spinal injury and found that a known neurotrophin, Artemin, improved bladder function by facilitating nerve regeneration and overcoming a known inhibitory barrier to regeneration.
My dissertation focused on the energetics and kinetics of NTP binding and hydrolysis by the *E. coli* DnaB helicase-replication factor DnaC complex in the absence and presence of nucleic acid. My results show that a DnaB-DnaC-ssDNA complex containing six DnaC molecules is the recognition complex in the initiation of DNA replication.
My project examined changes in the proportion of older adults with cognitive impairment in the US (1993 – 2004). The prevalence of cognitive impairment declined over this period, due to increases in education of the population. Declines were greater for blacks and Hispanics and lower socioeconomic groups.
Interferon is important for the clearance of virus infections. Viruses can escape this protective interferon response by blocking specific cellular proteins. I have shown that although SARS, Hep B and influenza viruses interact with the same pattern recognition receptors, they evade the interferon response by different mechanisms.
My research reveals important knowledge of the process of cardiovascular diseases. I demonstrated that a particular cell orchestrates a new amplification loop of inflammation that accelerates its progression. This novel mechanism highlights a “backdoor” pathway that could be targeted for potentially revolutionary treatments of aneurysms, aortic dissections, and atherosclerosis.
I investigated how immunity develops against the parasite Leishmania braziliensis, which causes a deadly and disfiguring disease in South America. We determined that the responses of dendritic cells and monocytes (cells of the immune system) play an important role in controlling this parasite.
The purpose of this study was to explore reasons homeless youth turn to substance abuse. Thirteen metropolitan homeless youth were studied. The findings fit five categories of my grounded theory centered around a core category called Using, consisting of three phase in the process, namely pre, active, and post substance use.
Bacteria of the genus *Rickettsia* possess the ability to invade host cells and quickly escape the host cell defense mechanisms in order to survive. My research led to the discovery of at least two ways that *Rickettsia* are able to perform this complex task involving a phospholipase enzyme and a hemolysin protein.
My research evaluated RepliVAX West Nile, a new vaccine for West Nile virus. I showed that this vaccine protected animals from West Nile infection. Studies also showed the role various components of the immune system play in the development of immunity from this promising vaccine.
Cerebral palsy (CP) results in stiffness of muscles and decreased motion of the knee and ankle, making walking difficult. I assessed knee and ankle motion and walking ability, following Selective Percutaneous Myofascial Lengthening Surgery (SPML) in children with spastic CP.
I studied the mechanisms involved in phencyclidine-induced neurotoxicity in developing rat brain as a model of schizophrenia. I showed that phencyclidine inhibited two key cellular signaling pathways important for neuronal survival and that both of which were involved in protection against toxicity by lithium and a known brain neurotrophic factor.
Injury to a nerve may cause pain. High levels of molecules called reactive oxygen species may help produce pain by affecting certain neurons, termed GABAergic, that are normally important for pain suppression. My studies showed that the function of these neurons in the spinal cord is decreased by oxidative stress and that this decrease contributes to the stress induced pain.