Examples of Lay Abstracts

The number of new cases of kidney cancer, along with the death rate, has been increasing steadily for the last 65 years in the United States, and approximately $1.9 billion is spent annually on its treatment. For patients who are not suitable to be treated with traditional surgery, cryoablation (killing the tumor by freezing it) therapy is one of the most promising and prevailing alternatives. In this therapy, multiple needles are inserted in to the patient's body with their tips at the place of the tumor. When the freezing starts, an iceball begins to grow near the tips of the needles and gradually cover the entire tumor to kill it. During this freezing period, the patient is scanned repeatedly using, for example, Magnetic Resonance Imaging (MRI) to monitor the progress of the therapy. Currently, this progress is assessed visually by scrolling back and forth through numerous 2D images. To improve safety and effectiveness in this work, we developed computer software that automatically identifies the needles and the growing iceball in 3D images. This can aid in better understanding the coverage of tumor by iceball during the freezing period of the therapy.

Taken from http://bwhresearchday.partners.org/poster-session-call-for-abstracts/

We all accumulate mutations within our mitochondrial DNA (mtDNA) which encodes proteins necessary for our energy production. The more mutations we accumulate, the less we can produce energy, the less we can maintain the health of our tissues. This mitochondrial aging is believed to be one of the main reasons behind aging and age-related diseases. mtDNA mutations can be inherited or caused by inflammation and oxidative stress. However, certain drugs such as the ones used to treat HIV are believed to also induce mtDNA mutations. Evidence is accumulating that HIV-infected people age faster than non-infected people. They have diseases normally seen in older people earlier in life. It is unclear whether this is because of their HIV virus (which can cause chronic inflammation) or because of the side effects of the antiretroviral drugs they take to treat their disease. It is technically challenging to study mtDNA mutations because there are hundreds of copies of mtDNA in each cell. We have developed a deep sequencing-based methodology to detect low frequency mtDNA mutations and distinguish them from background PCR and sequencing errors. We will use a cell culture model to determine whether commonly used HIV drugs can indeed induce mtDNA mutations. We will also test samples collected from HIV+ and HIV- individuals, young and old, treated with antiretroviral or not, as well as umbilical cord progenitor cells from infants born to HIV+ or HIV- mothers, to investigate the relationship between mitochondrial aging, age, HIV infection and exposure to HIV therapy. It is important to figure out what may accelerate aging in HIV+ individuals: the virus or the medications?

Taken from http://pathology.ubc.ca/files/2012/05/Research-Summary-and-Lay-Abstract_2012.pdf

Autism is a complex disorder, in which there is a strong familial predisposition. Many studies have been performed examining behaviour, anatomy and chemistry of the central nervous system, and the inheritance (genetics) of the spectrum of autistic disorders. One initiative, the Autism Tissue Program, has banked brain samples donated after death, for research use. Our study has extracted DNA from these brain samples, and analyzed them for millions of genetic ''markers'', in hope that this information can provide insight into the findings of other studies. We have also identified some

rare changes in the DNA that may be associated with autism susceptibility, and point to specific genes
that may be involved. Nevertheless, our main focus is in providing an ongoing resource of genetic data to the autism research community, so that other researchers can correlate their findings with genetic variation that we have described. All of these data are available to researchers through a peer-reviewed approval process used by the Autism Tissue Program, to ensure appropriate and ethical use of this information.

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Cardiovascular disease is the leading cause of death in the United States accounting for about 40% of all deaths. A class of drugs called statins has been developed to help reduce the risk of death from cardiovascular disease. These drugs were primarily developed to reduce the negative effects of lipids or fats on the cardiovascular system. Recently, it appears that these drugs might help reduce cardiovascular disease in ways that do not involve lipids or fats such as lowering blood pressure. Animals or blood vessels from animals used in this study will be given or treated with different statins. We will then examine changes in the cells, which may explain why statins have beneficial effects independent of their effects on fats. The results of this study will help us understand how statins can provide protection against cardiovascular disease and may help us develop better drugs for cardiovascular protection or indicate new uses for statins.

Taken from http://www.georgiahealth.edu/research/animal/archivepages/laysummary.html

The signature feature of muscular dystrophy as a disease is the absence of a key muscle fiber membrane protein called dystrophin. One of the most promising therapeutic strategies for countering muscular dystrophy is to replace in the diseased muscle the missing dytrophin protein with its "twin sister" protein, utrophin, which is found in small amounts in the muscles of people that have muscular dystrophy. We have provided exciting new evidence showing that utrophin is under the control of another muscle protein, calcineurin, an enzyme that we have shown to be an orchestrator of muscle growth. Indeed, we showed that when calcineurin is turned on within a muscle fiber, utrophin is enticed to appear in abundance all over the muscle fiber in areas usually reserved for dystrophin. By virtue of this replacement calcineurin is thus capable of rescuing fibers that are damaged by muscular dystrophy. Within the time frame of this proposal, we plan to further define the role of calcineurin in this rescuing of damaged dystrophic muscles and plan to identify other players in this symphony to help us to better understand and develop strategies and interventions to reverse its damaging effects.

Taken from http://www.cihr-irsc.gc.ca/e/43205.html