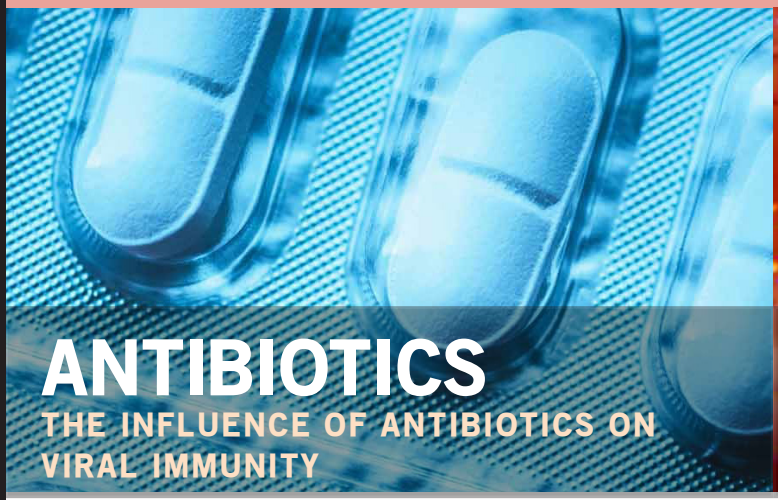
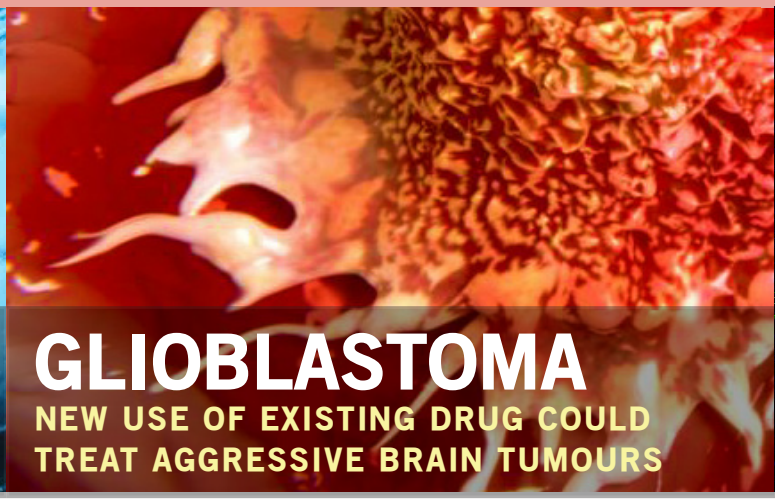


ANTIBIOTICS

THE INFLUENCE OF ANTIBIOTICS ON VIRAL IMMUNITY



GLIOBLASTOMA

NEW USE OF EXISTING DRUG COULD TREAT AGGRESSIVE BRAIN TUMOURS

SHADI SADEGHIAN

Recent *in vivo* experiments have demonstrated that, upon treatment with oral antibiotics, mice become increasingly susceptible to West Nile (WNV), Dengue (DNV), and Zika (ZIKV) viral infections.[1] Using ampicillin for as little as three days yielded exaggerated WNV symptoms.[2] Specifically, the experimental data demonstrated that, after antibiotic exposure, there was a decrease in the number of murine immune cells in gut-distal lymphoid tissue. Specifically, there were fewer CD8+ T-cells relative to antibiotic-free controls.[2] In previous experiments, the transfer of CD8+ T-cells has been said to protect alymphoid mice against a lethal WNV injection.[3] To test whether unprimed CD8+ T-cells can provide protection against lethal WNV, splenic CD8+ T-cells were transferred into mice, along with a lethal dose of WNV.[3] While all control recipients died, 75% of those who received a transfer of CD8+ T-cells survived.[3] This survival rate suggests that naïve CD8+ T-cells could provide protection against WNV. In addition to this finding, mice were treated with a broad-spectrum antibiotic cocktail for 14 days pre- and post-inoculation with WNV, ZIKV, and DNV.[2] While 80% of the control mice survived at least 21 days post-inoculation, only one experimental mouse survived this time frame.[2]

The authors argued that reducing the robustness of the microbiota is a factor in compromising immunity. This reasoning is consistent with previous studies that have examined this relationship. One experiment demonstrated that commensal bacteria-derived signals stimulate the innate immune system to attain optimal antiviral immunity.[4] The specific mechanisms are poorly understood, but it is speculated that commensal bacteria influence the activation threshold of innate antiviral response pathways, such as the interferon signaling pathway.[5]

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SOPHIE ZARB

Glioblastoma is an aggressive form of brain cancer that is often fatal within 18 months of initial diagnosis. These malignant, web-like brain tumours develop from astrocytes, a specific type of brain cell. The tumors spread rapidly from the supportive tissue throughout the brain in finger-like projections.[1] As there is currently no cure for glioblastoma, treatment is palliative in nature, consisting mainly of chemotherapy, radiation, and/or surgery.[1]

In a recent study conducted at the University of Georgia's Regenerative Bioscience Center and funded by the National Institutes of Health, researchers found that surfen, a compound molecule used to optimize insulin delivery, can potentially be used as an effective treatment to block tumour growth in the brain.[2] Given the positive charge of surfen, researchers wanted to test whether it could be used to block negatively charged sugars in brain tissue that are known to aid in cancer proliferation.[2-4] When tested in lab animals, the surfen-treated specimens developed smaller, less branched tumours, suggesting that the molecule hindered the tumour cells' ability to spread.[2]

These findings suggest that surfen treatment may make it easier to isolate and surgically remove glioblastoma tumours. If surgical intervention is pursued at an earlier stage, the need for other treatment protocols including chemotherapy and radiation may be reduced.[2] Researchers are hopeful that the proven safety and beneficial nature of surfen will result in an accelerated path to drug approval, paving the way for the medical community to change the prognosis of this life-threatening disease.[5]

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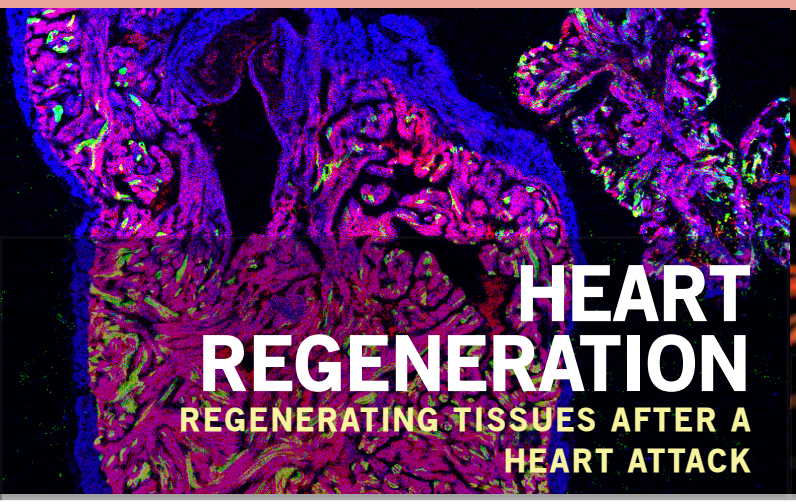

HEART REGENERATION

REGENERATING TISSUES AFTER A HEART ATTACK

MEERA CHOPRA

When human heart cells die during a heart attack, a cellular process to replace lost cells is initiated by cytokines. To prevent mitosis, an enzyme called protein tyrosine phosphatase 1B (PTP1B) can halt this signalling pathway and avert the regeneration of cardiac tissue.[1] In contrast, when cardiac tissue dies in aquatic animals, an allosteric inhibitor named MSI-1436 hinders PTP1B, leading to cell regeneration.[1,2] Furthermore, mice treated with MSI-1436 following a myocardial infarction had 600% more heart tissue regrowth than those without the treatment.[2]

The regeneration of dead tissue begins once cytokines bind to receptor tyrosine kinase (RTK) on the cell membrane, triggering a signal transduction pathway.[2] Generally, this process is terminated in humans by the enzymatic inactivation of RTK by PTP1B.[1,2] However, when MSI-1436 binds to the allosteric site of PTP1B, the enzyme is unable to bind to RTK, allowing signal transduction to proceed.[1,3] This pathway ends with the activation of a protein called CDK1 cyclin-B that enters the nucleus to stimulate DNA replication. Organelles then duplicate in the G2 phase of the cell cycle and the cell undergoes mitosis and cytokinesis.[3] This process is repeated until all required tissue cells are replaced.

Using MSI-1436 is a promising technique for tissue regrowth. This compound has undergone comprehensive toxicity testing and it is believed that injection into organs would not cause significant side effects.[2,4] Unlike other regenerative treatments, this technique has a lower risk of electrical impulses from regenerated tissue misaligning with the innate cardiac rhythm.[2] Thus, MSI-1436 shows promise for the future of biotechnology and could improve treatment for various heart diseases.

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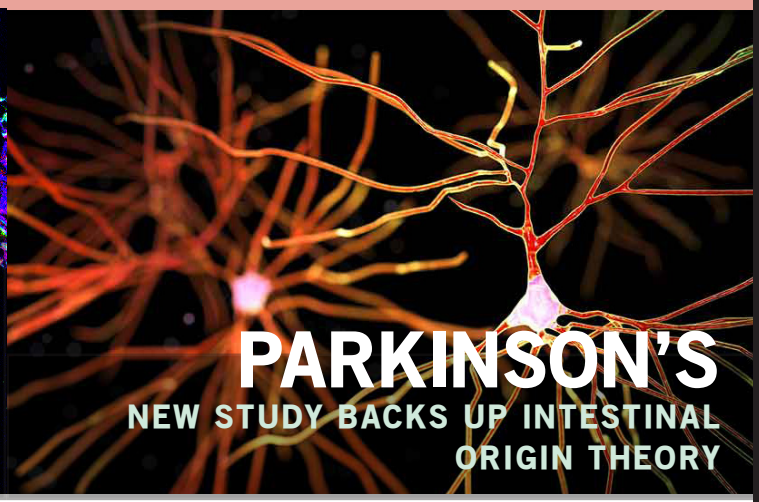

PARKINSON'S

NEW STUDY BACKS UP INTESTINAL ORIGIN THEORY

ADRIAN WONG

Parkinson's disease is a neurological disorder involving various symptoms, including slow movement, tremors of the limbs, stiffness, and deformities in posture.[1] The disease currently affects approximately 100,000 Canadians.[2] Parkinson's is characterized by a reduction of dopamine in the striatum of the brain due to a loss of dopamine-releasing neurons. However, the exact cause remains unknown, leading to many theories regarding this matter.[3]

One such theory, proposed in 2003, states that Parkinson's may originate from the gut and move to the brain.[4] A recent study conducted by Van Den Berge et al. presents evidence supporting this theory.[5] The researchers engineered transgenic rats that express extra copies of the *SNCA* gene, the gene most involved in early-onset Parkinson's.[5,6] These rats were used to study the movement of preformed alpha-synuclein fibrils —the intermediate form of which may cause neuronal death by affecting synapses and other targets— to the brain.[5,7] After injection of fibrils into the duodenum of the rats, alpha-synuclein was found in the major structures of the sympathetic and parasympathetic routes to the brainstem, including the substantia nigra, where the loss of dopamine-releasing neurons occurs.[4] These findings suggest that alpha-synuclein spread from the duodenum to the brain.[5]

This research may have implications for the treatment of Parkinson's, providing scientific evidence towards an origin for the disease.[4] While treatments for Parkinson's exist, they only replace dopamine in the brain to suppress symptoms and cannot halt the disease.[8] A better understanding of the way Parkinson's arises may allow the development of treatments that can stop it before it causes harm to a patient.[4]

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