Inducible and specific modulation of interferon-beta expression for targeted treatment of inflammatory and autoimmune diseases

Technology #cu13321

Interferons are anti-viral agents produced by nearly all cells as part of the body’s defense system. Interferons act to alert neighboring cells to the presence of a virus, causing them to heighten their anti-viral defenses. These signals are essential for the elimination of infectious agents. However, over-expression or aberrant expression of interferon-beta has been implicated in several inflammatory and autoimmune diseases. For example, overproduction of interferon has been recognized as the major cause of systemic lupus erythematosus. This technology utilizes cardiac glycosides that specifically bind to ion channels on the cell membrane, acting to alter the intracellular ion concentration and subsequently alter expression of interferon-beta. Regulating the levels and duration of interferon production is critical to the optimization of antiviral activities, while minimizing the detrimental effects associated with over-production or prolonged expression. This technology therefore utilizes inducible and specific interferon expression as a potential treatment modality with wide applicability to a number of diseases.

Cardiac glycoside delivery, an FDA-approved arrhythmia treatment, is shown to alter interferon-beta expression via ion channel blockage and may improve treatment outcomes for a variety of diseases

This technology takes advantage of the discovery that interferon gene expression can be regulated by modulating intracellular ion concentrations. Cardiac glycosides, such as ouabain and digoxin, specifically bind to the sodium-potassium pump on the plasma membrane of human cells, leading to a large increase in intracellular sodium concentration. Both of these drugs are FDA-approved for the treatment of congestive heart failure and cardiac arrhythmias, and are widely used. Current treatments for inflammatory and autoimmune diseases do not target interferon expression with specificity and do not have the capability to modulate the expression of interferon. Additional studies show that tumor necrosis factor (TNF) signaling is also negatively affected by cardiac glycoside treatment. Both interferon-beta and TNF are key cytokines implicated in inflammatory and autoimmune diseases, and experiments validating this technology suggest the possibility that cardiac glycosides could be used to treat these diseases.
The mechanism of action of this technology has been validated in vitro using various human cell lines.

**Lead Inventor:**

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**Applications:**

- Targeted interferon modulation as a therapeutic for autoimmune and inflammatory diseases
- Interferon up-regulation as a therapy for the immuno-compromised
- Research tool

**Advantages:**

- Targets interferon expression with specificity
- Modulates interferon expression
- Employs FDA-approved cardiac glycosides
- Applicability to a wide variety of diseases including: neurodegenerative, autoimmune, and inflammatory diseases.

**Patent Information:**


Tech Ventures Reference: IR CU13321

**Related Publications:**


**Inventors**

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