Calcipressins as lead targets for autoimmune, inflammatory and cardiovascular disease

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Technology description

MARKETS ADDRESSED:
The development of more selective immunosuppressive agents to mitigate transplant rejection and autoimmune diseases requires effective strategies of blocking signaling pathways in T-cells. Current immunosuppressive strategies use cyclosporin A (CsA) or FK506 to inhibit calcineurin, which dephosphorylates and promotes the nuclear import of nuclear factor of activated T-cells (NFAT) transcription factors. These nuclear NFATs then transactivate cytokine genes that regulate proliferative responses of T-cells. Both CsA and FK506 have debilitating side effects, including nephrotoxicity, hypertension, diabetes, and seizures, that argue for the development of alternative or complementary agents.
Researchers at Harvard University have discovered a novel group of endogenous inhibitors that competitively inhibit the activity of calcineurin. This discovery has important implications for the development of next-generation immunosuppressive agents that mitigate the side effects associated with current treatments.
The applications of this invention involve the development of next-generation compounds for the treatment of the inflammatory response, such as those related to autoimmunity and organ transplant rejection.

Advantages

The invention is a novel family of endogenous calcineurin inhibitors, termed calcipressins (Csp), which modulate the pattern of calcineurin-dependent transcription. Csp1-deficient mice showed abnormalities in lymphocyte development, had higher endogenous amounts of calcineurin, and underwent premature cell death in comparison to wild-type cells expressing Csp1. Additionally, a Csp1 peptide binds competitively to calcineurin and inhibits NFAT localization to the nucleus.

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