Dendritic cells (DCs), are key elements of the immune reaction cascade as they initiate adaptive immunity by migrating from peripheral tissues to lymph nodes. In this context, injection of DCs to patients has been used for tumor-associated antigen delivery. However, the DCs injected to patients migrate inefficiently to lymphoid organs, a process required to trigger antitumoral immunity. The present invention provides a new method to modulate migration of DCs cells in vivo and ex vivo by activating the lysosomal calcium channel Mcoln1.

**APPLICATION**

- **In vivo method** using an activator of Mcoln-1 for **vaccination**, treatment of **autoimmune disease**, treatment of **infection**.
- **In vivo method** using an inhibitor of Mcoln-1 to **treat cancer and to prevent or decrease metastasis**.
- **In vitro screening method** to identify molecules capable of modulating cell migration.

**PROBLEM ADRESSED**

DCs are central regulators of both immunity and immune tolerance, enlightening their great therapeutic potential. To date, two main approaches have been used to treat cancer patients with DC-based therapy: (1) transfer of syngeneic DCs differentiated and loaded with tumor-associated antigens in vitro and (2) injection of antigens into patients. In both cases, DCs must migrate to lymph nodes to present tumor antigens to T lymphocytes and trigger anti-tumor immunity. There is a need to identify and develop compounds able of modulating DCs migration to improve treatment of autoimmune diseases and cancer.

**COMPETITIVE ADVANTAGES**

The present invention provides new compounds that facilitate the migration of immature and mature DCs for the treatment of autoimmune diseases and cancer.

- **Targeted therapy based on DC cells migration.**
- **Targeted delivery of active molecules.**
- **Low toxicity.**

TRML1 activation via MLSA1 in immature cells increases cell motility.
The stimulation of the DC migration by activating the lysosomal calcium channel, Mcoln1 (also known as trpml1) has been validated **ex vivo** and **in vivo**. The activity of mcoln1 can be increased by different means including by treating DCs with the small compound MSLA1. The next ongoing step is to determine whether MSLA1 can be used to stimulate anti-tumor activity in mice, alone and in combination of checkpoint inhibitors.

**IP STATUS & OWNERS**


**WHAT ARE WE LOOKING FOR ?**

**Licensing** and **collaboration** for development, exploitation and commercialization of products targeting Mcoln1.

**PUBLICATIONS**


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