A CLEC12B antagonist for treating hyperpigmentation disorders (BIO16201)
A CLEC12B ANTAGONIST FOR TREATING HYPERPIGMENTATION DISORDERS (BIO16201)

Product factsheet

- **Target:**
  - CLEC12B (C-type lectin domain family 12 member B)

- **Product:**
  - A CLEC12B antagonist such as an antibody or a peptide

- **Application:**
  - Hyperpigmentation disorders

- **Rational / POC:**
  - CLEC12B is specifically expressed in the skin by melanocytes
  - CLEC12B is expressed at the membrane surface of the melanocytes and in the cytoplasm
  - Decreasing CLEC12B expression significantly reduce the transfer of melanin to the keratinocytes.
  - Expression levels of CLEC12B are correlated with key proteins involved in the pigmentation
  - Therefore Decreasing the expression of CLEC12B or preventing the interaction between CLEC12B with keratinocytes is a specific and effective way to decrease pigmentation in the skin.

- **Patent and publication:**
  - EP17305681.3: METHODS AND COMPOSITIONS FOR TREATING HYPERPIGMENTATION DISORDERS

September 2020
Proof of concept

Silencing of CLEC12B using lenti-shRNA increases the production of melanin in Normal Human Melanocyte (NHM)

Cells images (A) and cells lysates (B) of NHM transduced with control or CLEC12B lentiviral shRNA. (C) Quantification of melanin in NHM transduced with control or CLEC12B lentiviral shRNA normalized to protein content.
Proof of concept

Overexpression of CLEC12B using lentiviral vector decreases the production of melanin in Normal Human Melanocyte (NHM)

Cells images (A) and cells lysates (B) of NHM transduced with control or CLEC12B lentiviral particles. (C) Quantification of melanin in NHM transduced with control or CLEC12B lentiviral vector normalized to protein content.

September 2020
Silencing of CLEC12B using lenti-shRNA increases microphthalmia-associated transcription factor (MITF) and melanogenesis gene expression of DCT and Tyrosinase.

Overexpression of CLEC12B using lentiviral vector decreases microphthalmia-associated transcription factor (MITF) and melanogenesis gene expression, decreases cAMP-responsive element-binding protein (CREB) and SHP2 phosphorylation and increases P38 and ERK phosphorylation.
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