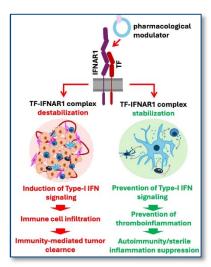


Modulation of Immune Response via Tissue Factor CD142

Cardiometabolic diseases, Autoimmune diseases, Cancer, Immunosuppression, TF, F3, CD142, interferon alpha receptor 1, Therapy, Diagnosis, IFNAR1

DESCRIPTION OF TECHNOLOGY

Immuno-suppression in tumor (immune escape) or immuno-activation in sterile inflammation (autoimmune diseases, cardiometabolic diseases) or infection (Covid-19) lack a unifying mechanism of action. The recently discovered effect that tissue factor ("TF", also called "F3" or "CD142") is modulating the interferon response by inhibiting interferon-alpha receptor-1 (IFNAR1) now provides a new way for modulating (enhancing or reducing!) the immune response of cells in case of several types of illnesses. In addition to it, inhibition of infection/inflammation driven TF-mediated coagulopathy can be achieved.



© Jayakumar Manoharan, Universität Leipzig The newly found modulation pathway allows for development of new API's for therapy as well as new kits for diagnosis. This may – for example – be achieved by way of API's mimicking the TF-IFNAR1 interaction (in case of therapy) and by way of developing probemolecules, directed at the TF-IFNAR1 heterodimer complex, indicating an active interaction of TF and IFNAR1.

AT A GLANCE ...

Application Fields

- Cardiometabolic diseases
- Autoimmune diseases
- Cancer
- Sepsis
- Viral infection
- Kidney diseases
- Coagulopathy

Business

- Pharmacy
- Medical research
- Diagnosis

USP

- Simultaneous targeting infection and coagulopathy
- Modulating the immune response positively or negatively
- May also be applied for diagnostic purposes

Development Status

- The Signaling pathway is identified and proven.
- Developing suitable API's has already been commenced.

Patent Status

Priority application no. EP23218581.9 was filed on Dec. 20th 2023 at the European Patent Office.

APPLICATION FIELDS

The broad involvement of interferons with numerous types of illnesses allows the therapeutic usage of the newly discovered way of modulating the immune response in case of many illnesses, especially with respect to cardiometabolic diseases, autoimmune diseases, sepsis, viral infection, and cancer.

Additionally to the therapeutic usage, diagnostic applications may also be developed, based upon the effect of the found TF/IFNAR1-interaction.

ADVANTAGES OVER THE PRIOR ART

Applying the newly found interaction pathway between TF and IFNAR1 is possible for therapeutic purposes as well as for diagnostic purposes. Because it is an interaction pathway, unknown heretofore, it can open the way to new (alternative) methods for therapy and diagnosis of cardiometabolic diseases, immune diseases and cancer.

STATE OF THE PRODUCT DEVELOPMENT

Currently the invention resides on an early stage of development, but further development, being aimed at the development, synthesis and testing of active pharmaceutical ingredients ("APIs") for making use of the discovered effect is already under way. Development partners are therefore especially welcome for intense joint cooperation.

MARKET POTENTIAL

The study "High and Rising Mortality Rates Among Working-Age Adults" in U.S. shows that, collectively, cardiometabolic diseases were responsible for more than 4.8 million deaths among the U.S. working-age (ages 25–64) population between 1990 and 2017. The contribution of cardiometabolic mortality to the rise in all-cause working-age mortality is considered to be due to the net increases in mortality from cardiometabolic diseases after 2010.¹

Prevalence (case numbers) of all these diseases is expected to be further increasing worldwide², showing that there is existing a huge market potential on global scale.

COOPERATION OPPORTUNITIES

On behalf of Universität Leipzig, TransMIT GmbH is looking for cooperation partners for further development or licensees in Germany, Europe, US and Asia.

https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(23)01301-6/fulltext and https://acsjournals.onlinelibrary.wiley.com/doi/10.3322/caac.21660, all viewed on Jan. 11th 2024.

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 $^{^1}$ Cf. https://www.ncbi.nlm.nih.gov/books/NBK571925/, viewed on Mar. 18th 2024. 2 Cf. https://pubmed.ncbi.nlm.nih.gov/37572826/,