## Immune Therapeutics Inc, Provides Scientific and Update and Links To Peer Review Publications (OTCMKTS: IMUN)

Orlando, Fl., January 16, 2019 Immune Therapeutics Inc (OTC-BB IMUN) today announced two peer-reviewed articles on the clinical application of . Methionine enkephalin (MENK are now available online.

**Orlando, Jan 15, 2019** (<u>Issuewire.com</u>) - "Peer-reviewed journals are an important part of the scientific process, which requires intensive scrutiny by other experts in the field of any manuscript submitted before publication. This external validation of the science behind MeNK is very important as we move forward to commercialize of this product," stated Immune CEO, Noreen Griffin Therapeutics today announced two peer-reviewed articles on the clinical application of methionine-enkephalin (MENK) are now available online.

The novel mechanism of anticancer effect on gastric cancer through inducing G0/G1 cell cycle arrest and caspase-dependent apoptosis in vitro and in vivo by ethionine enkephalin

Gastric cancer (GC) is the second cause of cancer-related deaths. Methionine Enkephalin (MENK), an endogenous opioid peptide, has immunological and antitumor activity This October 18, 2018 publication reports The results indicate that MENK could significantly inhibit the growth of human GC cells SGC7901 and HGC27 in a concentration- and time-dependent manner, decrease the number of cell colonies, and arrest cell cycle in the G0/G1 phase by causing a decrease in Ki67, cyclin D1, and c-myc mRNA. Furthermore, MENK could induce tumor cell apoptosis associated with the upregulation of Bax, a corresponding downregulation of BCL-2 and survive, and activation of caspase-3 and PARP. Moreover, MENK upregulated the expression of opioid receptors (OGFr) in SGC7901 and HGC27 cells. The interaction between MENK and OGFr in SGC7901 and HGC27 cells appears to be essential for the antitumor activity of MENK.: We conclude that MENK may be a potential drug for the treatment of GC.

https://www.dovepress.com/the-novel-mechanism-of-anticancer-effect-on-gastric-cancer-through-ind-peer-reviewed-article-CMAR

 Rules to activate CD+T cells through regulating subunits of opioid receptors by methionine-enkephaline (MeNK)

The current work indicates that MENK could promote the proliferation and functions of CD8+T cells through up-regulating mu and delta opioid receptors, and this effect of MENK could be canceled while one or both of the receptors would be blocked. Such observation suggests that different types of opioid receptors may combine as a whole to participate in the egulation of immune cells. Of course, the mechanisms need to explore in depth.

(https://www.sciencedirect.com/science/article/pii/S156757691830746X)

## ABOUT IMMUNE THERAPEUTICS, INC.

Immune Therapeutics Inc. is a late-stage clinical-stage biopharmaceutical company focused on the development and commercialization of our highly innovative immunotherapies. IMUN is a biotechnology company developing T-Activation immunotherapies to achieve immunomodulation in patients with

autoimmune and inflammatory disease and cancer and infectious

## Forward-Looking Statements

This release may contain forward-looking statements. Actual results may differ from those projected due to a number of risks and uncertainties, including, but not limited to, the possibility that some or all the matters and transactions considered by the Company may not proceed as contemplated, and by all other matters specified in the Company's filings with the Securities and Exchange Commission. These statements are made based upon current expectations that are subject to risk and uncertainty. The Company does not undertake to update forward-looking statements in this news release to reflect actual results, changes in assumptions or changes in other factors affecting such forward-looking information. Assumptions and other information that could cause results to differ from those set forth in the forward-looking information can be found in the Company's filings with the Securities and Exchange Commission (www.sec.gov), including its recent periodic reports.

888-613-8802

http://www.immunetherapeutics.com/

## **Media Contact**

Immune Therapeutics.com

noreen.griffin@immunetherapeutics.com

1-888-613-8802

36 North Orange Ave Suite 607

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