האוניברסיטה העברית בירושלים
THE HEBREW UNIVERSITY OF JERUSALEM
FACULTY OF MEDICINE הפקולטה לרפואה
Dr Eitan Shaulian ד"ר איתן שאוליאן

July 31, 2019

To The Steven A. Melman Foundation,

Dear Sir/Madam,

The funds received from your donation were directed to the following project; The gene JUN is involved in the development and progression of many cancers. It relays numerus signals to enhance of proliferation, metastasis, and resistance to drugs. Accordingly, it is regulated both by factors that induce cellular proliferation and by stress signals. Previous studies demonstrated that some tumors will not develop in its absence. Currently there is no inhibitor for JUN. We found a gene that regulates the expression of JUN. Using your donation we studied the nature of this gene and the consequences of its inactivation. It turned out that the gene does not translate to protein but acts as RNA and therefore belong to a growing family of long noncoding RNAs. It was found to be induced by the DNA damages and stress signals that induce JUN and moreover, to be important for JUN expression. In its absence Jun expression is reduced. The consequences of its inhibition were studied in the model of melanoma, a type of skin cancer in which JUN plays a role both in proliferation and drug resistance. It turned out that repression of the long noncoding RNA expression resulted in three effects that may have a beneficial clinical effect; 1. The cells were less motile a fact that may impair their metastatic capacity. 2. The cells were more sensitive to chemotropic drugs. 3. Eventually, 80-90% of the tumor cells in which the long noncoding RNA was transiently repressed died. We believe that permanent (stable) silencing will kill all the tumor cells. Future planed work will test how specific is the death to cancer cells and to melanoma in particular, to identify what other genes beside JUN are regulated by the this long noncoding RNA and weather it's silencing can prevent tumorigenicity in animals. We thank you for your donation.

Sincerely,

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