Lab Notebook of T.Perez | CSUN 108484650 | COMP490L

10-4-18

Spoke with prof Shapiro re informal mentoring of project, and said he’ll email prof Dewey.

[Mentioned to prof Dewey about the above. ]

It was suggested by prof S that a glossary might be helpful if needed on paper.

“Venue” was discussed. The math’ venue was chosen. \*

\* Since this is the case now, a toned-down version of the Contributions page should be

re-written.

10-5-18

Sent prof D and prof S ;

https://github.com/citationdude/NK-Cell-Simulator link

10-6-18

A toned-down version of the Contributions page was resubmitted. It also was probably an unacceptable format since it included an abstraction of the paper’s future abstract to sort of

“guide in” a pathway (an exposition to the “storyline”) to a somewhat new concept to those whom may review the paper.

10-7-18

**Plan of execution** for reviving and amending older model ‡ ; ; ;

Review prior notes.

Re-read Killer Cell Dyn – Wodarz ‡

10-8-18

LTG (long term goal) graphic;

Conceptual

correlations

Review all Hao Yuan Kueh material

Review

all Wodarz material

WODARZ-KUEH CORRELATION MDL

Mathematical

correlations

Current (10-8-18)

Wodarz mdl.

(Get Qt Creator version working)

Staged Wodarz model

Staged Kueh model

Get solutions via ODE tool (recommended by Prof Shapiro)

Determine Kueh

variables. Using Wodarz dynamic equations\*. Setup\*.

Implement o/p metrics

Test both versions

10-9-18

It must be considered that there exists certain mechanisms that are not included for this research. A relatively important one is transcriptase in terms of it’s proofreading type capabilities.

Since the Wodarz model is LCMV virus based, (key word virus), and his experiments are CD4 centered, many or most viruses are CD4 impaired , as are many cancers. Regarding CD4/CTL, there can be a valid correlation between certain cancers and certain viruses like LCMV. It just happens that the mouse virus (LCMV) is a good model. A well known virus of course the the HIV virus. Here, like cancers being of the retrovirus type, HIV is also a transcriptase problem (reverse transcriptase mechanism). [Wodarz 2007]. Also behaving differently from each other, in terms of immunology, the HIV and HCV are CD4 problematic.

New new discovery has been published regarding this, (ie the concept of [basically] way too many variables that can possibly determine viral or cancerous efficacy). As reported by the Cancer research UK, [cited by James Gallagher – Health and Science correspondent, BBC News – 12, Apr 2018], [sic]the Francis Crick Institute developed a way to analyze a cancer’s history to predict its future. One cancer could kill quickly while a patient with a seemingly identical cancer could live for decades after treatment. They state that [and knowing that , sic] as cancers grow and evolve, they become more mutated and, eventually, different parts of the tumor start to mutate in different ways.

ie to say, this phenomenon is so prevalent, they are intent on pinning down the root causes for this occurance.

10-10-18

[Wodarz variable re-assignments to do this weekend]