"Atavism, Extinction Theory and the Treatment of Cancer"

A plenary conference by **Mark D. Vincent**, Cancer Care, Ontario, Canada

Wednesday, the 8th of June 2022, 15h-16h

at Laboratoire TIMC, pavillon Taillefer, room R31, Domaine de la Merci, La Tronche, France

and via Zoom (link below)

Mark D. Vincent is a medical oncologist at the London Regional Cancer Centre, a part of Cancer Care Ontario, where his activities include the management of lung and gastrointestinal cancer, and laboratory research into the reversal of cytotoxic drug resistance by means of gene therapy. He is also an Associate Professor at the University of Western Ontario in the Department of Medicine, in the division of Medical oncology. His interests include the following subjects:

- translation of basic research into the clinic, and the design and conduct of clinical trials in lung and colorectal cancer,
- toxicity minimisation/avoidance and resistance reversal by means of gene-directed therapy and small molecule chemopotentiation.

Abstract. Evidence from phylostratigraphy and other sources has steadily mounted in support of the atavism theory of cancer, which holds that malignant transformation represents the re-emergence of an originating eukaryote protist, dating back to the Proterozoic eon. One corollary of this idea is that the peculiar traits of the malignant phenotype, including hypoxia tolerance, milieu acidification, Warburg metabolism and bloom-like growth, are inherently primitive and/or adaptations suited to the specific geochemistry of the Proterozoic. These and other features act as markers or signatures, serving to distinguish cancer cells from the normal tissues of the host of origin; as such, they may be the basis for novel therapies whose primary objective is to eradicate the cancer cells without intolerable toxicity to the patient. Key attributes of good cancer therapies include not only efficacy mechanisms to shrink the cancer, but also selectivity mechanisms to avoid damage to normal cells. Such cancer treatments may, but are not obliged to target molecular drivers of the cancer, if they cancer be identified; however, highly effective therapies can and have been designed based solely on markers or signatures confined to the cancer and not expressed or manifest in normal cells.

While the atavistic features of cancer cells are indeed candidate markers or signatures for this purpose, additional insights into the design of new and better treatments may also be obtained from a deeper understanding of extinction theory. It is well-accepted that at least five mass extinctions have occurred since the Cambrian, and the forces causing them are at least partially understood. There is much less information available on extinctions that may have occurred prior to that, during the Proterozoic, but it is reasonable to assume (and in fact known to some extent) that similar ultimate causes were operating then as later, including volcanism, bolide impacts, extraterrestrial radiation, continental drift, glaciation, and other manifestations of climate change. Clearly, at least some of the early eukaryotes had to have survived not only these existential threats, but also the generally harsh conditions of the Proterozoic.

Extinction theory provides insights into both killing and survival mechanisms, and in this respect may serve as a paradigm for cancer therapies which have the same goals: exterminate the cancer cells, protect the normal cells. One of the key insights from mass extinctions is that the ultimate causes most efficiently operate in concert (‘multi-causality’). In fact, some established cancer regimens have discovered this principle empirically, and are extremely effective; others are less effective and could be much improved by exploiting this insight. In particular, combinations of driver oncogene inhibition and acausal marker-constrained destructive agents may be particularly interesting, including also the re-primitivization markers and signatures mentioned above.

In this way, one might contemplate a unification of atavism, extinction and treatment theories into a broader and generalizable framework that is not only intellectually satisfying but also practically useful in helping to address the many serious and ongoing unmet medical needs in cancer.

Link for the visio-conference :

URL : https://univ-grenoble-alpes-fr.zoom.us/j/91204287393?pwd=TE9HRGxwd1c1Uyt2NHUvSDJnMVhOUT09
ID de réunion : 912 0428 7393
Code secret : 325347
Une seule touche sur l’appareil mobile
+13462487799,,91204287393# États-Unis (Houston)
+16465588656,,91204287393# États-Unis (New York)

Composez un numéro en fonction de votre emplacement
+1 346 248 7799 États-Unis (Houston)
+1 646 558 8656 États-Unis (New York)
+1 669 900 9128 États-Unis (San Jose)
+1 253 215 8782 États-Unis (Tacoma)
+1 301 715 8592 États-Unis (Washington DC)
+1 312 626 6799 États-Unis (Chicago)
ID de réunion : 912 0428 7393
Trouvez votre numéro local : https://univ-grenoble-alpes-fr.zoom.us/u/ayX9XeUBT

Participer à l’aide d’un protocole SIP
91204287393@zoomcrc.com

Participer à l’aide d’un protocole H.323
162.255.37.11 (États-Unis (Ouest))
162.255.36.11 (États-Unis (Est))
213.19.144.110 (Amsterdam Pays-Bas)
213.244.140.110 (Allemagne)
Code secret : 325347
ID de réunion : 912 0428 7393