Natural Killer Not Functioning to Combat Non-Presentation of MHC Due to Downstream Malfunction of Toll Like Receptor

A lesson in adaptive parasitology, and free parking.

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Abstract

The recent discovery of the car park cell, and its symbiotic relationship with a new microorganism the "car" has been useful in detecting a new defect in the immune system. TLR 9 defects may lead to a loss of defect in NK activity.

Introduction: Endosymbionts have been of major interest to biologists. Endosymbionts have been thought to evolve alongside their hosts from parasites, eventually deriving mutual benefits and prospering together. The Biscuit group would like to report on an event which runs the counter to the norm.

The car produces energy and hazardous pollutants in the process of its normal mobility function. However, cars when stopped are vulnerable to attack by parasitic organisms known as parking inspectors (according to the literature shows Today Tonight and A Current Affair). The carpark cell offers protection from car parasites, and in return a primitive proxy for cellular products (money) is given to the cell by the car. However, defective non-money releasing strains have been discovered. The mechanism for this mutation was studied closely, and it appears that the fault lies with a defective host immune response.

Methods: Facts were made up post-hoc in line with tabloid journalism standards.

Results:

Figure 1: Toll Like Receptor (TLR) 9

Figure 2: TLR 9 Unbound complexed CpG motif

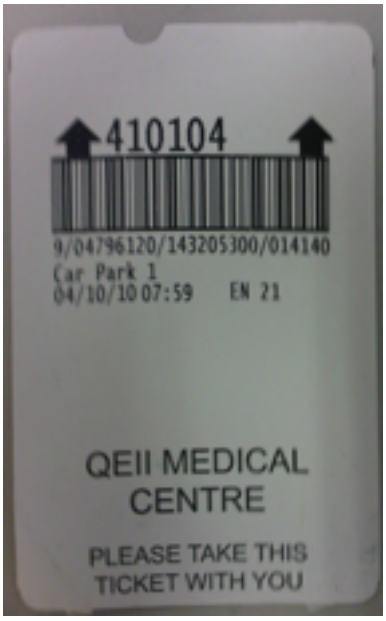

Figure 3: Putative, inactivated modifying complex of CpG motifs

Figure 4: Money-Hand Complex (MHC)

Figure 5: Natural Killer (NK) cells

Disgust: TLR 9 will normally recognise the uncomplexed CpG and prevent exit of the vehicle from the carpark. Presentation of the complexed CpG elements prevent binding and the upward motion of the observed yellow gate like mechanism. Deviant TLR9 was found to not bind uncomplexed CpG and allow free passage of vehicles out of the carpark. Triplicated daily single car studies were used to determine the state of the TLR 9 mechanism.

Further research determined that interaction with the MHC and the NK cells, prevented the release of devastating economic sanctions (fines), also known as poorins. The mechanism for the inhibition of poorins is affected by co-stimulation of bound TLR 9. Normal host car park CpG sequences are complexed and therefore, upon the later encounter of the TLR, are not bound, leading to no further stimulation of the NK cell, releasing poorins, mediated by a protein known to cause systemic shock, uncontrollable rage, and severe inconveniences based on trivial inconveniences, the carpark warden.

If the TLR mechanism failed to bind inadequately complexed CpG it was safe for cars to escape without adequate MHC presentation. It is believed that this mechanism has led to the evolution of a parasite that down regulates MHC production, and thus favours parasite proliferation and sequestration of money by the parasite, prior to its escape. Therefore, in the absence of TLR 9 co-stimulation, it has been found that some strains of carpark parasites have evolved to prevent any mutualistic symbiosis, essentially taking without providing anything for the maintenance of the carpark cell.

It has been proposed by Libertarian economists that localised IGA may act as a defensive mechanism against carpark cell invasion by the parasites via a sketchy mechanism known as “financial interest”.