J Clin Cell Immunol 2017, 8:5 (Suppl)
DOI: 10.4172/2155-9899-C1-040

Site attachment inhibition therapeutics

Simon Raymond
Melbourne University, Australia

The concern with respect to antimicrobial resistance and the associated health threat has gained increasing attention and there has been difficulty in gaining traction globally. Given the lack of success by the two pathways established to date which have focused on: 1) "replication of infective agent" and 2) "immune system enhancement," the current researcher has conceptualized and developed the new, or third, mode of action pathway represented by "*site attachment inhibition" (or negation of cellular attachment by infective agents).*" The current author anticipates *site attachment inhibition therapeutics* to include drug (medication) based therapies, stem cell based treatment (including prenatal and earlier) incorporating new generation immunization methods, and waveform (E.g. electromagnetic radiation) based treatment. With respect to viruses, support for the likely success of the new mode of action pathway: A) the known CCR5-Δ32 mutation achieves resistance (immunity) against HIV through negation of cellular attachment; B) other areas of medicine use analogous receptor antagonism (E.g. beta blocker therapy); C) advanced IT uses analogous site attachment inhibition to remove viruses. With respect to bacteria, support for the likely success of the new mode of action pathway: A) advanced IT uses analogous site attachment inhibition to remove IT infections; B) glycoproteins are key receptors for attachment and analogous to glycoprotein IIb/IIIa medications which inhibit (negate) platelet aggregation and thrombus formation, it seems reasonable to pursue antagonism or blockade of other glycoprotein receptors in order to prevent bacterial attachment to human cells (note: this is also relevant to viral infections); C) the human immune system coats infective agents in an attempt to negate cellular attachment, therefore this mode of action represented by site attachment inhibition makes scientific sense. Attention must be directed toward correctly identifying the target receptors and appreciating the difference between association and causation. Looking at mutations noticed in the human population and connecting this to the innate resistance they possess to certain infections is not enough as this may simply represent association as opposed to causation. Even the known CCR5-Δ32 mutation has not been completely confirmed as direct/causative of the inhibition of attachment observed in research analyses. Future research by the current author will likely include delineation of the application of quantum physics to medicine and surgery, starting with neurology and immunology, and in what circumstances this is appropriate. In addition the merger between fields including immunology, neurology, IT and advanced physics (quantum physics) appears likely to commence. Furthermore, detailed delineation of new generation immunization methods to be developed based on site attachment inhibition. In addition, the details regarding the unique new mode of action pathway (site attachment inhibition) with the only previous related research (or, minority research) more focused on aspects such as masking foreign entity identification and related methods. In conclusion, this paper presents the new, or third, mode of action pathway in antimicrobial therapy represented by *site attachment inhibition therapeutics*.

simonraymondcontact@gmail.com