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Opinion

Antibiotic Resistance

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The problem is that most antibiotics were discovered but the bacteria has learnt in a clever manner how to undo the said actions and neutralize the antibiotic itself. So now we are at a place where those little infections that we could easily kill with a pill are ready to kill us again.

I just had a small thought in mind, that what we eat today as cure has a hard work of almost 50 years behind it involved. The things though difficult then but were far simpler in terms of techniques used and results and also far less competition. The people I feel actually did hard work then.

Now do we have that kind of time? And efforts.

My opinion was may be we start looking at some alternative techniques as well, the first and foremost that I believe in very strongly is resorting to plants. But again the drawback is geographical distribution and variation of active compounds. Besides, the process of individual screening is very time consuming.

Yet another option in my opinion can be the use of poisons secreted by microbes externally. Exotoxins are delivered by pathogenic microbes, without which the microorganism does not evoke manifestations in the tainted person. Numerous bacterial poisons are discharged into nature and are in this way amiable to immune response treatment.as an example would like to mention about the use of the as of late FDA and EMA-affirmed human monoclonal counter acting agent (mAb) Bezlotoxumab (Merck) ties to the *C. difficile* poison B and is shown to avert repetitive *C. difficile* disease (CDI) in danger grown - *Staphylococcus aureus* harbors numerous VFs to encourage tissue grip, resistant avoidance, and host cell damage. Similarly certain secretions can be used. Type III emission frameworks (T3SS) are intricate structures installed in the bacterial internal and external films of numerous Gram-negative microorganisms that are utilized to convey destructiveness effector proteins into host cells and encourage the foundation furthermore, dispersal of diseases.

Type III emission frameworks (T3SS) are intricate structures installed in the bacterial internal and external films of numerous Gram-negative microorganisms that are utilized to convey destructiveness effector proteins into host cells and encourage the foundation furthermore, dispersal of diseases. The interference of poison discharge or auxiliary proteins of T3SS, particularly the needle-tip protein get together is a focal point of ebb and flow look into for little particle inhibitors.

Recognition and attachment of bacteria to the uroepithelium plays a key role in anti-virulence strategies that target uropathogenic *E. coli* (UPEC) and lower urinary tract infections (UTIs). Adhesion is mediated through the expression of pili and their tip adhesion/FimH that binds to mannosylated residues on the bladder epithelial surface . Several strategies to block adhesion have been pursued including developing mannose analogs that bind within the mannose-binding pocket of FimH and block pilus binding of FimH to host receptors and thus prevent attachment of UPEC. Other FimH inhibitors were effective in the mouse model, but it is not known if these animal models are predictive and whether data from them will translate into clinical efficacy.

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