

Mycobacterium tuberculosis and its Affect

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COMMENTARY

Drug resistant tuberculosis is increasing. Its treatment is costly and lengthy. New rapid methods of detecting drug resistance (e.g. PCR), are helpful but too costly to be used in developing countries. Several tools with improved sensitivity and speed of diagnosis have been endorsed by the World Health Organization (WHO) [1,2], as they allow early epidemiological and therapeutic interventions. However the current process of endorsement is necessary but insufficient [3].

The International Standards for tuberculosis Care discourage the use of serological tests in routine practice and no international guidelines recommend their use. Development of new drugs is also very costly and not seen as a priority in the market orientated economics of the pharmaceutical industry. The development of multidrug-safe *Mycobacterium tuberculosis* (MDR-BT) and widely tranquilize safe *Mycobacterium tuberculosis* (XDR-BT) strains has made a large number of the as of now accessible enemy of tuberculosis drugs insufficient. Tuberculosis caused by *Mycobacterium tuberculosis* complex remains one of the major public health problems, especially in developing countries. Late resurgence of aspiratory tuberculosis in created nations like United States represented a danger to the clinical network because of safe strains. Consequently WHO looked to traditional medicine. Tuberculosis is these days the most deadly disease in the World.

Several signature compounds for *Mycobacterium tuberculosis* and *Mycobacterium bovis* have been identified which may offer a solution, if the compounds can be shown to reliably characterize infection through sampling of human breath. Electronic odorant detection systems so far appear inadequate for the task of TB detection. A recent study by researchers at Christchurch's New Zealand Institute for Plant and Food Research Limited shows that the bees can detect even traces of sweet-smelling volatiles produced by *Mycobacterium tuberculosis*. They concluded that potential exists for trained honeybees in non-invasive diagnostic tests for TB. Avicenna just about 1000 years back suggested nectar as one of best cures in the treatment of tuberculosis. Nectar with against tubercular treatment limits the unfavorable medication responses prompted by Anti-TB tranquilizes in recently analyzed sputum corrosive quick bacilli positive pneumonic positive tuberculosis patients. It has been demonstrated that the growth of mycobacteria from positive cultures and from positive smears of affected patients was inhibited by honey. Yet, the component by which nectar act is hazy. A lot littler changes in oxygen pressure influence the anti-microbial executing of bacterial persisters (e.g. *M. tuberculosis*). Despite the fact that these little decreases in oxygen strain don't modify the murder motor of the bigger anti-toxin defenseless populace, these progressions significantly influence the size and endurance capacity of the littler persister subpopulation.

This subpopulation remains vulnerable to the common antibiotic-induced hydroxyl-radical-mediated death pathway if sufficiently high freeradical concentrations can be maintained. Generation of hydroxyl radical is a common property of honeys of European, North and South American origin. The hydroxyl radical-based

instrument of nectar activity didn't separate between anti-microbial delicate and anti-microbial safe microorganisms. Hydrogen peroxide (H₂O₂), one of the Reactive oxygen intermediates generated by macrophages via the oxidative burst, was the first identified effector molecule that mediated mycobactericidal effects of mononuclear phagocytes. It is well known that honey when diluted is a glucose/glucose oxidase generating H₂O₂ system. TLRs (Toll like Receptor) are involved in cellular recognition of mycobacteria, expression of TLR2 or TLR4 conferred responsiveness to both virulent and attenuated *M. tuberculosis*. Honey has been found to stimulate monocytes *in vitro* to release TNF- α .

This was determined to be due to a 5.8 kD component in honey which acts via the TLR-4 receptor. Mycobacterial infection and pro-inflammatory cytokines increase surface expression of TLR-2. Interestingly it has been shown that Honey stimulates inflammatory cytokine production. The above referred to components may act synergistically they may likewise be embroiled in a similar procedure with various response time courses.

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