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Opinion Article

Leprosy metabolic, not infectious

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California research group detects link to atherosclerosis

A versatile team of 15 researchers linking chemists, biologists, microbiologists, immunologists, molecular geneticists, cardiologists and dermatologists (namely D Cruz, AD Watson, CS Miller, D Montoya, MT Ochoa, PA Sieling, MA Gutierrez, M Navab, ST Reddy, JL Witztum, AM Fogelman, TH Rea, D Eisenberg, J Berliner and R L Modlin) form the universities and institutes of California have joined forces to research into the pathogenesis of leprosy.

This aim led to an extremely valuable work, recently published under the title Host-derived oxidized phospholipids and HDL regulated innate immunity in human leprosy", *Journal of Clinical Investigation,* Volume 118, Number 8, August 2008; pages 2917-2938.

At least two conclusions deserve to be highlighted from this immensely valuable study:

a. Metabolic alterations of the oxidative type from determined lipids of the organism condition the immune capacity (of genetic origin) to acquire leprosy.

b. These metabolic alterations are surprisingly similar to those observed in atherosclerosis; the authors suggest that this similarity could guide the search for new treatments for both diseases.

These conclusions reaffirm the metabolic nature of leprosy, as we have been expressly repeating for decades, an idea which we have developed at length in our book *Metabolic Theory* of *Leprosy,* Diorki publishing house, Madrid, 1998, 214 pages.

My comment

The curtain has come down on the whole show built up around leprosy as a result of this empirical contribution of the California researchers to the pathogenesis of leprosy, definitely and finally placing leprosy within the metabolic illnesses. Even if so late in the day, enough of myths and legends, of ignorant descriptions because as from today leprosy, we repeat, are to be included within metabolic pathology.

On bringing down the curtain, the enigmatic character of this ancient human plague, which has existed since Biblical times, vanishes forever.

All this leads one to consider (metaphorically) that the "dictatorship of the Hansen bacillus" has come to its end and that the idolatry of this germ should be consigned to history.

Enough already of taking about an "incubation period" of two, 20 or "years, enough of speaking of contagion, isolating leper patients of discriminating against those patients.

Enough of seeking antibiotics and/ or chemotherapy to treat lepers, enough of seeking

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preventive vaccines against the "infection" caused by the Hansen bacillus, enough of the preventive and/or therapeutic measures recommended by the World Health Organization (WHO), which has always considered leprosy as an infectious illness caused by the Hansen bacillus. Now the WHO recommendations do not count for anything. The Hansen bacillus as collapsed and with it, all the preventive/ therapeutic structure created around it.

The contribution of these United States researchers to our knowledge of the pathogenesis of leprosy is of immeasurably enormous value and deserves to be crowned with a Nobel Prize.

This metabolic focus of leprosy has always been upheld by us since 1947 until the present but we could never advance in persuading scientific orthodoxy and social structures which always considered leprosy to be an infectious disease, despite all our efforts. Our work on leprosy, published between 1947 and 2008, thus shows it.

Many of statements in the paper have not been accepted by the reviewers. To respect the right of author, this is being published as such in Opinion Article category -Hony Editor