Ebola...or African Strains of Tuberculosis?

The Ebola virus currently ravaging several West African nations has striking similarities with two strains of tuberculosis which are just as deadly and cause Ebola-like symptoms. Yet health authorities are in denial about this link and don't consider it in the design of diagnostic tests.

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The US Centers for Disease Control and Prevention (CDC) recently declared: "Diagnosing Ebola in a person who has been infected for only a few days is difficult, because the early symptoms, such as fever, are nonspecific to Ebola infection and are seen often in patients with more commonly occurring diseases, such as malaria and typhoid fever."[1]

Only a sin of omission, then, would explain why anyone or any group would *not* want to mention specifically the most commonly occurring cause of infectious death in Africa—tuberculosis (TB)—whose sky-high rates in West Africa make Ebola look like a dropperful of water squeezed into the Mississippi.

If by October 2014 Ebola had laid claim to what the World Health Organization (WHO) claimed was well over 3,338 deaths[2] since its March outbreak, certainly this ought to be weighed in the light of the approximately 600,000 Africans slain by TB during the same time window.[3] Furthermore, by 20 October 2014, worldwide Ebola mortality stood at 4,868, with just over 81 per cent of these deaths recorded in Liberia and Sierra Leone.[4] While Liberian health officials warned as early as 2009 that TB was skyrocketing out of control[5], a mixed scientific coalition from Sierra Leone and Germany cautioned that Sierra Leone's own tuberculosis level was not only the highest in West Africa but was filled with resistant strains of TB and tuberculous *Mycobacterium africanum* which had "reached an alarming level…raising the question of possible consequences" for a future new TB epidemic.[6]

Indeed, almost half of all TB cases in the West African Ebola zone are caused by *Mycobacterium africanum*—an unusual yet just as deadly member of the tubercular family, exclusive to West Africa, which is rapidly becoming a microbe of great public, and now possibly global, concern. That tubercular M. *africanum* can and has already caused TB in the USA is a matter of record.[7] Furthermore, there is a body of evidence that M. *africanum* requires more sustained contact, even among household members[8]—certainly mirrored in the current outbreak. Meanwhile, health officials continue to insist that "casual contact" cannot transmit Ebola—precisely the same claim that they've long made about TB.

Surely the CDC is aware that there is not a sign or symptom of Ebola, including its haemorrhagic tendencies, that cannot be found in acute disseminated miliary (blood-borne) tuberculosis, once called "galloping consumption"—the single most feared form of the disease *ever*. Most likely it is also aware that such tuberculosis has its own virus-like forms, some of which can simulate the Ebola virus. Such viral TB is generally acknowledged to be TB's preferred form—as a survival strategy to storm any inclement conditions in which the microbe might find itself.[9] Then why did the CDC not mention tuberculosis, by name, in its short list of possibilities that could cause Ebola-like symptoms? If such oversight stopped there it would be unremarkable, but it seems to have been carried over in the very design of the most recent diagnostic tests issued to detect Ebola.

Discovery of the Ebola Virus

In September 1976, a team—including a 27-year-old medical graduate training as a clinical microbiologist at the Institute of Tropical Medicine in Antwerp, Belgium—received a blue thermos from Zaire. It contained two 5.0-mL clotted blood specimens from an African-based Flemish nun. The Belgian physician who sent it, Jacques Courteille, practising in Kinshasa, included a note saying that he was at a complete loss as to the nun's mysterious, yet deadly, illness. He also asked if the samples could be tested for yellow fever. This thermos had travelled from Zaire's capital city of Kinshasa on a Sabena commercial flight to Belgium—inside its deliverer's hand luggage. When the samples were received, Peter Piot, the 27-year-old medical graduate, and his colleagues placed the blood samples under an electron microscope. To be sure, Piot's interest was virology, and a virologist he would soon become—best known for his work on theorising the "viruses" behind Ebola and AIDS. To this effect, he contributed heavily to the voluminous literature that HTLV-1 had a role in AIDS, which it did not. Piot had this to say about the Ebola virus:

> *We saw a gigantic worm-like structure—gigantic by viral standards. It's a very unusual shape for a virus; only one other virus looked like that and that was the Marburg virus.*[10]

To some, it might be considered "worm-like"; to others, serpentine (see figures 1–4). But the new "virus" needed a name. Piot related the interesting tale of how Ebola came to be named Ebola:

> *On that day our team sat together late into the night—we had also had a couple of drinks—discussing the question. We definitely didn't want to name the new pathogen 'Yambuku virus', because that would have stigmatised the place forever. There was a map hanging on the wall and our American team leader suggested looking for the nearest river and giving the virus its name. It was the Ebola river. So by around three or four in the morning we had found a name. But the map was small and inexact. We only learned later that the nearest river was actually a different one. But Ebola is a nice name, isn't it?*[11]

Depends how you look at it.

Piot's specimens proved negative for yellow fever. He mentioned that the tests for Lassa fever and typhoid were also negative. What, then, could it be? Piot said that to "isolate any virus material", small amounts of the blood samples were injected into Vero cells and into mice. Several of these mice subsequently and abruptly died—"a sign that a pathogenic virus was probably present in the blood samples…used to inoculate them".[12] The fact that the mice died did not mean that it was at the hands of a "pathogenic virus".

Piot's boss at the Institute of Tropical Medicine, Professor Stefaan Pattyn—who Piot admitted "could be a bit of a bully"—supposedly specialised in the study of mycobacteria (tuberculosis and leprosy) yet he seemed unaware of the haemorrhagic consequences of acute TB. Nor had he taken the time to use special stains and cultures to detect its virus-like cell-wall-deficient forms. Instead, Pattyn followed his current passion, shared by Piot. Pattyn had previously worked in Zaire for six or seven years, and exotic viral illnesses were now "right up his alley".[13] So Pattyn's team likewise never really considered a strain of acute miliary TB or its viral cell-wall-deficient forms in his rule-outs for an acute haemorrhagic or epidemic fever—among them *Mycobacterium tuberculosis* and *Mycobacterium africanum*.

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The Galloping Spread of Tuberculosis

The Ebola of its day on steroids, "galloping" acute consumptive tuberculosis could kill in days—the mere memory of which, just a few generations ago, brought terror to the faces of those who had witnessed and were describing it. Dubos and Dubos made it clear that "galloping consumption" was not an isolated but a frequent diagnosis in the 19th and early 20th centuries.[14]

Despite persistent myths to the contrary, in the early phase of any new TB epidemic from a new and virulent strain, tuberculosis manifests itself as an acute disease and only much later as the chronic pulmonary tuberculosis that we know in today's western world. An example of this can be found in the high mortality during the 1918 "influenza" pandemic, when African Americans were brought to fight in France during World War I, large numbers dying from a fast-tracked tubercular "galloping consumption".

Many often underestimate the speed, contagiousness and ferocity of a TB epidemic. Khomenko and Muratov's 1993 study[15] should have cemented the notion that the explosive contagiousness of just such Ebola and influenza-like viral forms of tuberculosis are exactly the stuff that previous epidemics and pandemics could have been made of. But it didn't.

In the USA, the CDC and the National Institutes of Health (NIH) seemed to feel differently, ignoring the historic possibility. There was much the same viral passion at that time over "influenza" when, in 1990, a new multidrug-resistant (MDR) tuberculosis outbreak took place in a large Miami municipal hospital. Soon thereafter, similar outbreaks in three New York City hospitals left many sufferers dying within weeks. By 1992, drug-resistant tuberculosis had spread to deadly mini-epidemics in 17 US states, and was reported not by the American but the international media as out of control.

Viral forms of swine, avian and human TB can be transmitted from one species to another. So can exotic strains of tuberculosis and *Mycobacterium africanum*, imported into the United States through countries such as Liberia. By 1993, the World Health Organization had proclaimed tuberculosis a global health emergency.[16] That emergency has never been lifted.

Anderson pointed out that such acute, untreated disseminated, "galloping", blood-dispersed TB could kill in hours or days[17], its mortality, according to Saleem and Azher, even today approaching 100 per cent.[18] Ebola itself can take up to a month to kill its victims, said Ben Neuman[19], an expert in viruses at the University of Reading in the UK—although there are many cases that also kill in hours or days.

Not only were tubercular haemorrhaging and fever both mentioned by Fox[20], but also haemorrhaging of or into the serous cavities, gums, nose, joints, skin and bowel. Appleman[21], discussing massive spontaneous haemorrhages into the vitreous humour, mentioned that Axenfeld considered acute tuberculosis an important possibility in the rule-out for bleeding into the eye. Coughing up blood has always been a well-known scenario for TB. Haemorrhages of significance from the ear, secondary to tuberculous otitis media, are also on record.[22] There is the well-documented possibility of acute disseminated tuberculosis attacking the bone marrow and, through fibrosis, causing a partial shutdown of platelets—changing the very morphology of those platelets as well as interfering with their function. These combined effects create a clear and present haemorrhagic danger.

Still to this day, bone marrow biopsy is at times a valuable diagnostic test for tubercular involvement. In addition, extrapulmonary (outside of the lungs) tuberculosis is the most frequent cause of a prolonged fever of unknown origin (FUO) and has been for a long, long time.[23]

A fact initially carefully minimised by certain Ebola "authorities" and recently clarified by Feldmann is that in the current Ebola outbreak "less than half" of the people infected showed visible haemorrhaging.[24] This was just enough to prompt some virologists to rethink Ebola's designation from "Ebola haemorrhagic fever "to the "Ebola virus disease". So much for "haemorrhagic fever". Yet even then, in a 1978 WHO *Bulletin* regarding the 1976 Ebola outbreak in Zaire, it was admitted that haemorrhaging, although from "multiple sites", was "principally [from] the gastrointestinal tract".[25] However, patients with TB spread to the gastrointestinal tract can also have fever and abdominal pain, and can have the same gastrointestinal or rectal bleeding that patients with Ebola can have.

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Fatality Rates on the Rise

Nor do the parallels stop here. In September, as the CDC justifiably warned against nonessential travel to Sierra Leone, available data from the two Ebola facilities in that country came in with case fatality rates (CFRs) for Ebola that ranged between 50 per cent and 72 per cent. Although considerably higher than the 37.7 per cent CFR that Sierra Leone's Ministry of Health was reporting[26], this averages out to an agency-reported fatality rate of 61 per cent—not much different from the approximately 67 per cent mortality given for the untreated active tuberculosis that currently rages in West Africa and many other places around the world.[27]

Meanwhile, WHO recently summed up that although the rate of Ebola infections was picking up speed at an alarming rate in West Africa, the fatality rate was 53 per cent overall, ranging from 64 per cent in Guinea to just 39 per cent in Sierra Leone.[28] If this 53 per cent figure was designed to make the situation more bearable, it hardly achieved its goals.

Problems with Diagnostic Tests

Moreover, the designs of present diagnostic tests for Ebola don't meet the sniff test in certain respects. An article in the *Los Angeles Times* of 7 August 2014 mentioned that an unapproved Ebola test-tube diagnostic assay, developed by the US military, had just been approved for use in the US under a "special emergency-use provision".[29] Critics claim that the two PCR systems to be used for Ebola testing in such "emergency situations" are unapproved. But there's more.

While an instruction booklet issued by the US Food and Drug Administration[30] showed impressive results for detecting and thereby being positive for known "Ebola" samples, it sadly failed in its inadequate selection of those pathogens that might be cross-reacting and therefore making for false-positive Ebola tests. The instruction booklet, Version 2.0, which accompanied the new Ebola assay, mentions:

> 9.2.2.2 *Bacterial Cross-Reactivity: Bacterial cross-reactivity of the* EZ1 *assay was evaluated by testing purified nucleic acid of bacteria that potentially could be infecting the majority of the*

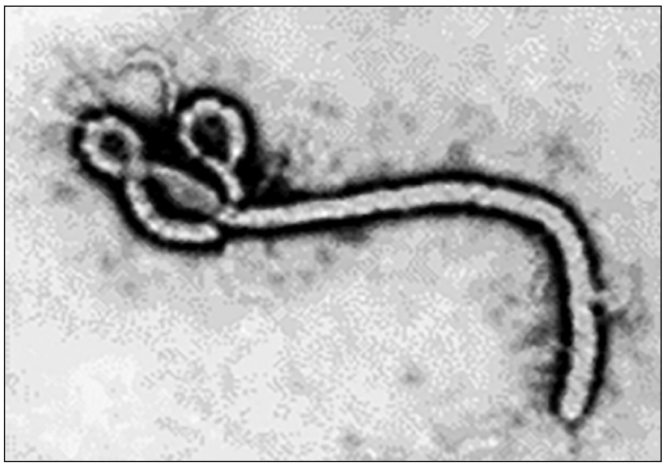

Figure 1: The serpentine form of the Ebola virus (magnification ~60,000x)
(Micrograph from Dr F. A. Murphy, University of Texas Medical Branch, Galveston, Texas, courtesy CDC)

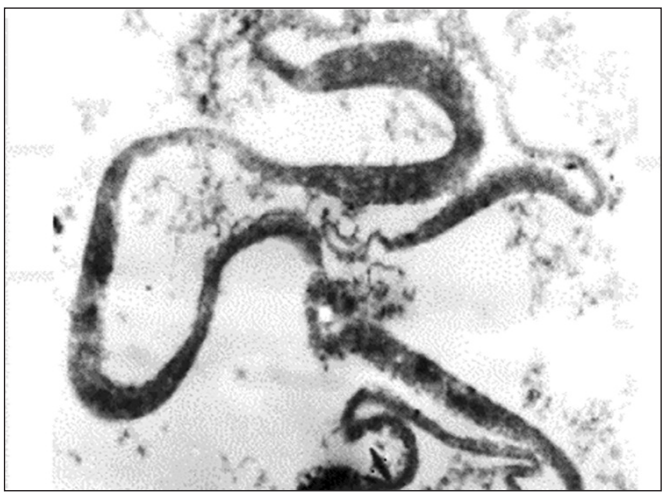

Figure 2: L-forms (or cell-wall-deficient forms) of tuberculosis under the electron microscope
(Source: Michailova, L. et al., "Morphological variability and cell-wall deficiency in *Mycobacterium tuberculosis* 'heteroresistant' strains", *Int. J. Tuberc. Lung Dis.* 2005 Aug; 9(8):907-14, 911)

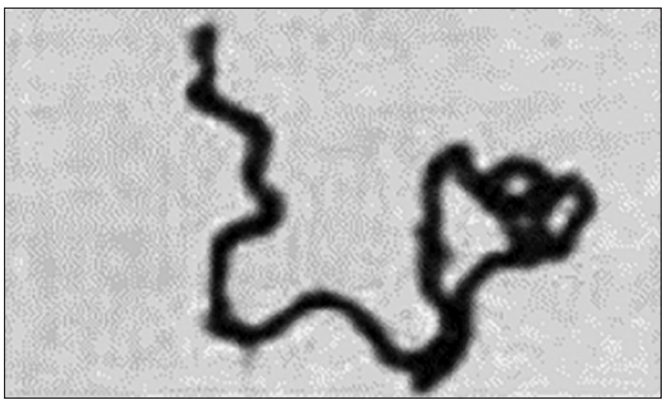

Figure 3: Serpentine form of "cords" of lethal tubercle bacilli
(Source: Darzins, E., *The Bacteriology of Tuberculosis*, University of Minnesota Press, Minneapolis, 1958, p. 296)

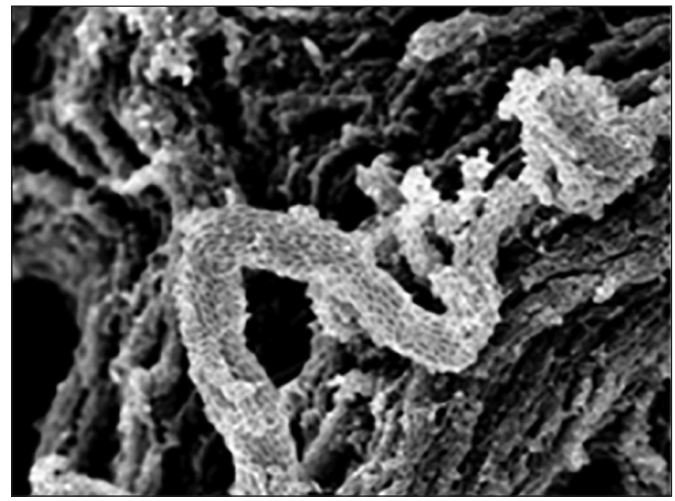

Figure 4: Worm-like cords from an atypical TB under the electron microscope (Source: Julián, E. et al., "Microscopic Cords, a Virulence-Related Characteristic of Mycobacterium tuberculosis...", *J. Bacteriology* 2010 Apr; 192[7]:1751-60, 1756)

population. ***No cross-reactivity was observed in the human DNA or any of the bacteria tested (Table 51)***.[31] [Bold print is in original.]

Yes, the only problem being that a glance at table 51 shows practically every bacteria in existence except for the one subset of pathogens "that potentially could be infecting the majority of the [West African] population", and those pathogens are, again, M*ycobacterium tuberculosis* and its related M*ycobacterium africanum*.

Such diversion is no trivial point. As time went by, it became obvious that attempts were in the pipeline to link the pathogenesis of Ebola and AIDS, right down to their sexual transmission (a poorly kept secret is that TB can also be sexually transmitted[32]). So mistakes made during the AIDS probes would have to be avoided with Ebola.

For example, in the past, as veterinarian Myron "Max" Essex—the first scientist to propose human

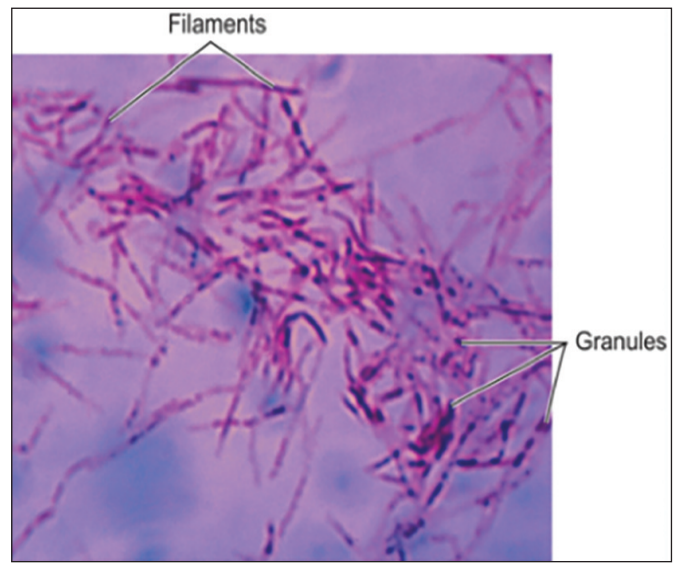

Figure 5: Filamentous cell-wall-deficient forms of *Mycobacteria tuberculosis*

immunodeficiency virus (HIV) testing—knew, tuberculosis and its allied mycobacteria gave a false positive for HIV in his tests in almost 70 per cent of cases. Such cross-reactivity between HIV and tuberculosis was so significant that it forced Essex and his protégé, Congo physician Oscar Kashala, to warn that the HIV screening test, the enzyme-linked immunosorbent assay (ELISA) and the western blot results "should be interpreted with caution when screening individuals with M. *tuberculosis* or other mycobacterial species".[33] This, of course, automatically meant throwing away HIV serum diagnostics for, according to WHO, at least a third of the people in the world who presently harbour tubercular infection.

So why, then, was *Mycobacterium tuberculosis* noticeably excluded from the CDC's Table 51 and not included in those pathogens tested for cross-reacting and therefore possibly giving false-positive tests for Ebola? Did the original panel (Version 1.0) chosen by government scientists actually include *Mycobacterium tuberculosis* and related microbes in its design—only to find that indeed these mycobacteria caused positive tests for Ebola as in the HIV affair? Did they feel that such results might muddy the waters or be too difficult to explain, and so subsequently remove them? This is not known.

A group of researchers from the University of Oxford, UK, and the University of Leuven, Belgium, have just determined that HIV is "almost certain" to have begun its spread from Kinshasa, now the capital of the Democratic Republic of the Congo.[34] Whether this research bears out or doesn't, Kinshasa itself has long been a hotbed for tuberculosis—and now Ebola. On top of that, a doctor in rural Liberia, swarmed with Ebola patients, says he's had extremely good results with HIV treatment—albeit such treatment was borne out of admitted desperation.[35]

The US National Institute of Allergy and Infectious Diseases, having got wind of this, is carefully looking into the use of some of these HIV-antiretrovirals to control Ebola. This just might work, but will it answer the reason as to *why* it works? The NIH, for example, has long known, through mechanisms not yet clearly worked out, that HIV treatment suppresses both the tuberculosis and the fowl tuberculosis that are currently the leading causes of infectious death in AIDS. To this effect, the NIH decades ago recruited university laboratories to look into the reason why.[36]

Therefore, are the HIV drugs working against an "Ebola virus" which is estimated to have killed over 3,300 Africans from March to the beginning of October 2014, or TB which killed 600,000 Africans in that same window? There still remains much work ahead to determine this. Antiretrovirals have major side effects.

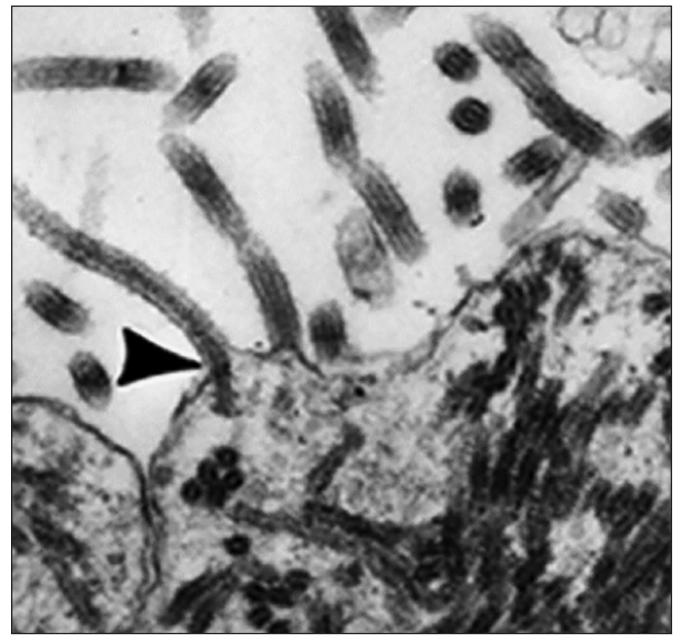

Figure 6: Filamentous forms of the Ebola virus (magnification ~40,000x) (Source: Dr F. A. Murphy, University of Texas Medical Branch, Galveston, Texas)

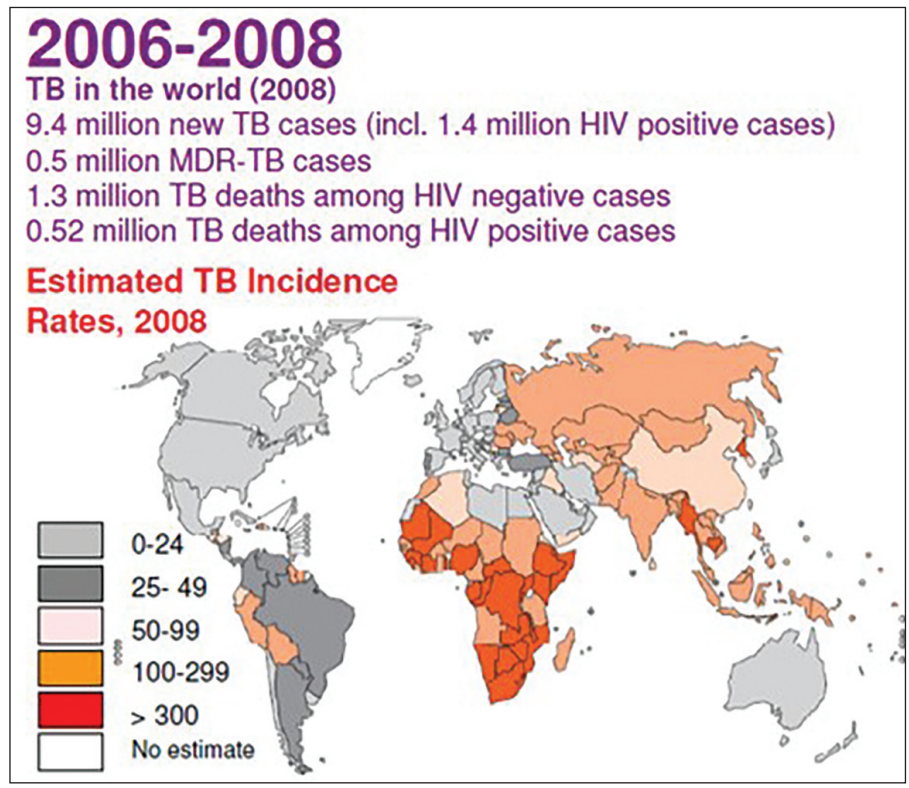

Figure 7: Map showing the severity of the tuberculosis problem, compiled in 2006–2008—the last period before WHO's TB statistics plummeted as a result of including many TB cases in the questionable wastebasket of "AIDS-defining illness". Note that the darker-highlighted regions are mostly in Africa. (Source: WHO)

Continued from page 36

His two-part article "Influenza and the TB Connection" was published in NEXUS, vol. 19, nos 1 and 2. His two-part article "The Untold Truth About Cancer" was published in NEXUS, vol. 17, nos 1 and 2.

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