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Commentary
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Can Diabetes Mellitus Type-1 be A Transmissible Disease? Revisit to an Earlier Premise

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Commentary

To some diabetalogists/diabeticians, and certainly the majority of the general public, the title of this commentary might come a bit as a surprise. What is known about diabetes mellitus (DM), as a medical condition, that it is a group of linked biochemical-physiological disorders initiated by pathological rise in the circulating blood glucose concentration. This hyperglycemia develops mostly as a consequence to either hypoinsulinemia (DM type-1) or cellular insensitivity to insulin (DM type-2). In extreme cases both causative factors transpire and therefore act together on the body leading to the induction and progression of an aggressive form of the disease. For the time being, DM is unequivocally regarded as an inherent condition since its causes' are innate to the affected person and provoked primarily by life-style and genetics. Although this self-origin manifestation of the condition is certainly true as evident in the multitude of scientific studies and reports, nonetheless other unorthodox cofounders may exist and yet to be revealed and ascertained.

One such further and elusive causative factor of DM could be its possible transmissibility from a diabetic patient to a biochemically-physiologically-genetically susceptible non-diabetic individual. In an article titled "Is Diabetes of Infectious Origin?" published as early as 1927 in The Journal of Infectious Diseases, Eduard Gunderson proposed that DM may be transmissible via blood transfer and/or anomalous contents in consumed meat. For a moment, this assumption might be thought of as a tad farfetched as no scientific evidence exists to support what could be regarded as a bold claim. Nevertheless, in a fairly recent study led by Claudio Soto and published in the Journal of Experimental Medicine it was reported that DM type-2 might be caused by the formation of toxic aggregates of misfolding of islet amyloid polypeptide protein which could result from transmissible proteins.

This commentary proposes that it is highly probable that DM type-1 might also be a transmissible disease, however via bacterial agents. This assumption is supported by an ample and compelling, although be it circumstantial, evidence provided by the findings and observations of considerable and recent related studies. Interest in the transformation of the body, and in particular the gut, microbiota as a cause to the development of numerous human diseases began to attract growing attention and formed the bases for many scientific studies. Examples of such diseases include metabolic, liver, gastrointestinal, respiratory, mental and autoimmune abnormalities, in addition to DM. As far as DM is concerned, human diabetics were shown to possess transformed gut microbiota and, vice versa, chemical induction of DM type-1 distorted the microbiota of rats' gastrointestinal system. The question, among many, that presents itself at this juncture: Can such a transformation in the microbiota of the body, and in particular that of the gut, becomes an intermediary in the transmissibility of DM type-1? To answer this question, first it must be stated that such a hypothesis is sound as it is established that microbial agents are the foremost propagators of infectious diseases. Additionally, and as concluded from the observations of recent studies, if infection and gut microbiota transformation do occur, then it is highly probable that an aggressive attack on the intestinal apical wall will develop leading to epithelial lesions, inflammations and consequently drastic autoimmune responses. Formed erratic antibodies may translocate and even attack neighboring cells and those could be the insulin producing beta-cells due to the close proximity of the pancreas to the most susceptible part of the intestine; the duodenum.

With the lack of direct scientific evidence, is the assumption that DM type-1 be an infectious disease lacks ground? The answer is simply YES. Nonetheless, if bodily material contaminated with

pathogens were to transfer between a diabetic and non-diabetic, and the latter subject was prone to such infections, due for example to compromised immune responses, such a possibility may materialize. This aforementioned suggestion is based on the scientific fact that pathogens, and certainly those that dwell in the gut of patients, can make a healthy individual become sick. Thus, the premise that non-classical live biological agents that exist in the gut of a diabetic might be an additional factor that leads to the development of DM type-1 in the most vulnerable of individuals is highly plausible under the 'right' circumstances. In an attempt to address such an interesting objective we carried out a small and limited pilot study in which we co-habituated healthy and streptozotocin-induced diabetic Sprague-Dawley rats for more than 16 weeks and on biweekly bases monitored their blood glucose. No change was observed in the blood glucose of the normal rats although the streptozotocininjected rats showed progressively the classical symptoms of diabetes. Although this study yielded negative data, still the high rate at which DM type-1 is spreading world-wide, which almost exceeds the international curve for any other epidemic, warrants consideration of every possible etiology of this disease. Accordingly, more focused studies testing versatile combinations of external and internal variables are recommended to test this proposed hypothesis.

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