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Poster presentation

Entry of *M. leprae* into Schwann cells: old studies revisited

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All leprosy is neural and the role of Schwann cells as preferential host cells for *M. leprae* is undoubted. It is within these that *M. leprae* grow and exhibit their dual roles of nerve damage and immune subversion. These roles have been dissected by the innovative use of experimental mouse models and murine Schwann cell culture extensively used at FMR in the last two decades. It is debatable whether the entry of *M. leprae* into Schwann cells (Sc) is specific. The evidence suggests that early entry into Sc is mediated only by viable *M. leprae* though no evidence of a single specific bacterial structure mediating bacterial entry was obtained. The role of weakly phagocytic cells (like Sc) in *M. leprae* pathogenesis was expanded in intervention studies in mice wherein actively phagocytic cells were killed by ingestion of injected silica. Since *M. leprae* growth in both foot pad and nerve damage was not affected, it implied that the weakly phagocytic cells were responsible for these features. Additionally murine tissue culture studies demonstrated that active entry of *M. leprae* into Sc resulted in deposition of bacterial antigens within the Sc membrane blocking cellular communication and nerve regeneration. Evidence for breakdown of cellular communication was obtained from the down regulation of NgCAM and NGF receptor which matched the pattern of limited/progressive nerve damage in murine experimental models involving C57Bl/6 and Swiss White mice. The antigen deposits paradoxically served as a focal point for either (i) functional tissue damaging lymphocytic stimulation or (ii) their death via apoptosis – a representation of immune evasion.

Cumulatively mechanisms amplified in experimental models and nerve tissue culture could provide insights into precipitation of immune-mediated lepra reactions and for the silent phases of nerve damage in leprosy, which are non immune in nature. Unfortunately the insights provided have not been mainstreamed into the understanding of leprous neuropathy. The observations will be therefore amalgamated with those of recent studies towards identifying fresh approaches to intriguing questions in leprous neuropathy.

References

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