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IMAGES OF INTEREST

Gastrointestinal: *Mycobacterium avium paratuberculosis* and Crohn's disease

Uncovering the cause of inflammatory bowel disease appears to have become the holy grail of gastroenterology. For Crohn's disease, the most popular hypothesis is that the disease results from loss of immunological tolerance to bacteria or other microorganisms that are normally present in the bowel lumen. However, several other hypotheses exist including a role for infectious agents such as atypical mycobacteria, *Chlamydia* species, *Listeria* monocytogenes, cell-wall deficient *Pseudomonas* species, *Mycoplasma* species and a number of viruses including measles virus. Interest in atypical mycobacteria was recently rekindled by observations reported by Dr S Naser and others in an article published in *Lancet* in 2004. Using a novel method, the group was able to culture *Mycobacterium avium* subspecies *paratuberculosis* (*MAP*) from the peripheral blood in 50% of patients with active Crohn's disease. This has raised the possibility that *MAP* may be the cause of Crohn's disease in humans in a similar way to Johne's disease in livestock.

Recently, our group has been able to replicate the above findings in a number of patients with active Crohn's disease. We used Ziehl-Neelsen staining to show what we believe are spheroplastic phase forms of *MAP* (purple spots) within the cytoplasm of a macrophage (Fig. 1) and persisting in modified TB broth after 4 months of culture (Fig. 2). To confirm the identify of these acid-fast organisms, we performed a duplex polymerase chain reaction designed to detect the unique insertion (L1 and L9) sites of the *MAP*-specific insertion element IS900. The amplified L9 site was sequenced and then compared to a reference DNA of *MAP*. Alignment of our sequence against the reference sequence showed a perfect match, thereby confirming that the *Mycobacterium* found in our Crohn's disease patients was indeed *MAP*. These observations support the association between *MAP* and Crohn's disease. However, further research is required to determine whether *MAP* is involved in the pathogenesis of Crohn's disease or whether the detection of *MAP* in peripheral blood simply reflects translocation of organisms through a leaky epithelial barrier.

Contributed by
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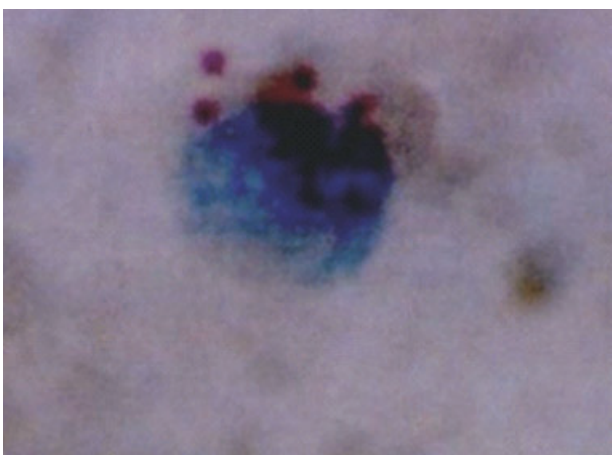

Figure 1

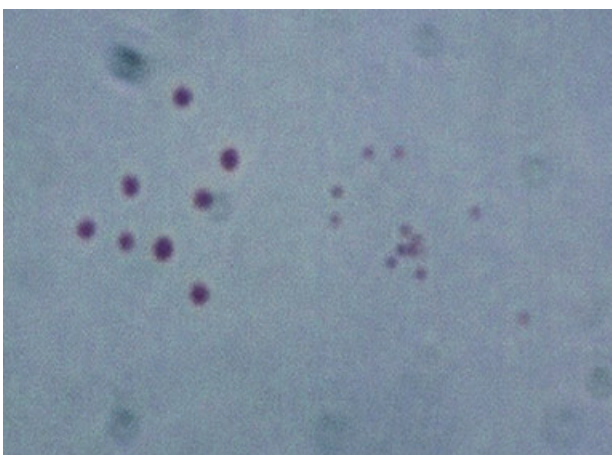

Figure 2

Contributions to the Images of Interest Section are welcomed and should be submitted to Professor IC Roberts-Thomson, Department of Gastroenterology, The Queen Elizabeth Hospital, Woodville South, South Australia 5011, Australia.