



<b>Title</b>	<b>The role of c-Myc in phagocytosis of mycobacteria in human macrophages</b>
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oxide. We aim to demonstrate these mechanisms by treating GVHD murine models with human MSCs.

**Methods:** Balb/c host mice were subjected to 800 cGy dose of gamma irradiation and given donor T lymphocyte depleted bone marrow cells and CD4+ T lymphocyte from donor C57BL/6N mice via tail vein. Some mice were treated with  $0.8 \times 10^6$  human MSCs. Survival rate and symptoms of GVHD were monitored. At day 6 post HSCT, single cell suspensions, tissue lysates and paraffin sections were prepared from target tissues for flow cytometry to check for donor T lymphocyte engraftment, enzyme linked immunosorbent assays to check for levels of proinflammatory cytokines, chemokine and regulatory pathway testing, western blot for inducible nitric oxide synthase (iNOS) expression and imaging by coherent antistokes raman spectroscopy (CARS).

**Results and conclusion:** The immunosuppressive functions of MSCs were documented in our GVHD models by:

1. Observing protection of GVHD mice from death. This may be due to the decreased engraftment of donor T lymphocyte with concomitant decrease in levels of TNF- $\alpha$  and IFN- $\gamma$  and decreased RANTES expression which is a chemokine ligand marker in target tissues.
2. Noting an increase in levels of iNOS in MSC treated mice, which is a known immunosuppressive soluble mediator.
3. Suppression in the phosphorylation of STAT 5A/B proteins in MSC treated mice which indicates arrest of the T cell cycle.
4. Finally the presence of MSCs in target tissues is documented by recording differences in chemical bond vibratory signals between control and MSC treated samples using CARS.

### **The Role of c-Myc in Phagocytosis of Mycobacteria in Human Macrophages**

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Mycobacterium tuberculosis is an intracellular pathogen and the causative agent of the disease tuberculosis. Macrophages are the major immunocytes to initiate host immunity against mycobacteria. Among the multiple strategies employ by macrophages to defence against mycobacteria, phagocytosis is the first step. Through

phagocytosis, macrophages could not only clear the pathogens from infection sites, but also present antigens derived from the engulfed bacteria to lymphoid cells.

c-Myc is a transcription factor that regulates a variety of target genes. It can form a complex with Max and bind to the enhancer box sequences of the promoter to mediate the transcription. Recently, our group revealed that c-Myc has a potential role in regulating the antimicrobial responses in macrophages.

Here, we further revealed that c-Myc may play a positive role in phagocytosis and contribute to host defense to mycobacteria. Pretreatment of c-Myc inhibitor, 10058-F4, could significantly reduce the amount of mycobacteria internalised by macrophages. The acidification of phagolysosome in mycobacteria infected macrophages was also inhibited by 10058-F4. Further investigation showed that macrophages phagocytose mycobacteria in a PI3K/Akt independent pathway. And the action of c-Myc inhibitor does not affect the expression levels of Rho family GTPases. However, we found that 10058-F4 could significantly inhibit phosphorylation of ERK1/2 kinase, which has been indicated to play a role in FcR mediated phagocytosis in macrophage. In conclusion, c-Myc may play a role in phagocytosis of mycobacteria through regulating phosphorylation of ERK1/2.

### **Symptomatic Meckel's Diverticulum in Children: A 5-Year Review**

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**Background:** Despite being the commonest congenital gastrointestinal anomaly, Meckel's diverticulum (MD) is notoriously difficult for diagnosis due to the diversity of presentations. We aim to review the MD patients in our hospitals, focusing on their various presentations.

**Method:** Hospital notes of patients aged  $\leq 18$  with diagnosis of MD from 2008 to 2013 were retrieved. The demographic data, clinical presentations, treatments and pathological results were analysed. Adult MD patients during the same period were also recruited for analysis.

**Result:** Thirty children with the diagnosis of MD had operation done (age 1 day to 17 years). Three patients were born with persistent omphalomesenteric duct. In 4 children, MD was an incidental finding during operation only.