J Clin Cell Immunol 2017, 8:3(Suppl)
DOI: 10.4172/2155-9899-C1-037

Validation of *Candida* colonization associated with anamnestic response of anti-enolase IgG at early stage of invasive candidosis by memory B-cell ELISPOT

Fang-qiu Li, Xiu-juan Shang, Xiao Chen, Yu-an Hu and **Li-ning Shi**
Nanjing University, China

Objective: The rapid elevation of IgG antibody against *Candida* enolase has been observed in the patients at early stage of invasive candidosis. The aim of the study was to build a comprehension of the rapid increase of specific IgG titers against dominant antigens (such as enolase) at the early stage of invasive *Candida* infection resulted from humoral immune anamnestic response associated with previous *Candida* colonization.

Methods: The oral *Candida* colonization mouse model was established, and the immuno-competent and immuno-compromised *Candida* colonized mice were challenged by intraperitoneal injection of *Candida* spore. The numbers of enolase-specific memory B-cells in the spleen were measured by ELISPOT and compared with the levels of specific IgG, IgM and IgA antibodies in the peripheral blood.

Results: The burst of enolase-specific memory B cells was detected in both immuno-competent and immuno-compromised mice at day 7 post-invasive infection; which was followed by a strong increase in specific antibody titre in the *Candida* colonized mice. The Eno-IgG antibody was positively correlated with the antigen specific Bm ($r=0.737$, $P<0.01$).

Conclusion: It was confirmed that the *Candida* colonization associated with anamnestic response of IgG against dominant antigen at early stage of invasive candidosis and the rapid elevation of specific-IgG would suggest a diagnosis of invasive infection.

njlifq@163.com

The quinoline-3-carboxamide paquinimod prevents development of diabetes in the non-obese diabetic (NOD) mouse

Sahar Tahvili[1], **Marie Törngren**[2], **Dan Holmberg**[1], **Tomas Leanderson**[1] and **Fredrik Ivars**[1]
[1]Lund University, Sweden
[2]Personalmatsalen Active Biotech, Sweden

The NOD mouse spontaneously developed type 1 diabetes (T1D). At the age of 3-4 weeks, there was detectable infiltration of mononuclear cells in the pancreatic islets of Langerhans of these mice. This process known as insulitis, causes selective cell death of the insulin producing β-cells in the islets. Female NOD mice displayed severe insulitis at about 15 weeks of age and developed hyper-glycaemia at around 15-30 weeks of age. We have previously shown that the immunomodulatory compound paquinimod can reduce the influx of monocytes to sites of inflammation. Since monocyte-derived macrophages are known to be involved in pathogenesis in NOD pancreas, we have in here investigated the impact of paquinimod treatment on the development of T1D in the NOD mouse. In cohorts of mice treated between weeks 10 to 20 of age and followed up until 40 weeks of age, we observed dose-dependent reduction of incidence of disease as well as delayed onset of disease. Further, in mice treated with paquinimod from 15 weeks of age, most of the treated mice had not developed glycosuria at 30 weeks of age and displayed strongly reduced insulitis. Importantly, in these treated mice there were significantly more non-infiltrated islets than in untreated controls. Collectively, these data indicate that paquinimod treatment inhibits progression of insulitis to overt diabetes in the NOD mouse.

sahar.tahvili@med.lu.se