

## Role of L-Fucose in modulating leukocytes behavior and reducing infection recurrences

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Leukocytes enter inflamed tissue through a cascade of molecular interactions that mediate leukocyte adhesion to the Lendothelium as well as leukocyte activation. Selectin family members largely mediate initial tethering and rolling of leukocytes on vascular endothelium, whereas integrin and Ig family members are essential for leukocyte firm adhesion. The rolling of leukocytes on the endothelial surface, is mediated by E-selectin (CD62E) and P-selectin (CD62P), which are expressed on the surface of activated endothelial cells, and L-selectin (CD62L), which is constitutively expressed on most leukocytes including granulocytes, monocytes and lymphocytes.

The objective here is to discuss the ability of L-Fucose to modulate leukocytes behavior via restoring CD62L expression and its clinical benefit in reducing frequency of infections. We describe three patients with recurrent infections and significant leukocytosis with neutrophilia suggesting leukocyte adhesion defect (LAD1 and LAD3 were rolled out). All of them have normal neurodevelopment and expression of CD15s. However, CD62L was deficient in all patients. Other immunedisorders including CGD were rolled out in all patients.

Flow cytometry analysis of peripheral blood leukocytes was performed on all three patients before the therapy and while patients are on L- Fucose therapy (50-100 mg/kg/day) at 1-week, 4-weeks and periodically. L-Fucose therapy corrects defective CD62L, normalization total WBC counts, neutrophil counst and significantly reduced the frequency of infections in all three patients. *In conclusion*, the adhesion molecules and their ligands are all required for immune defense against microbes. CD62L is coordinated molecular present in many human cells and characterized by its dynamic interactions with cells and tissues. Isolated L-selectin (CD62L) deficiency causing primary immunodeficiency was not previously described and L-Fucose is capable to correct defective CD62L and reduce infections.

## **Biography**

Daifulah Al Zahrani, MD, FAAP, ABAI. Received bachelor's degree of Medicine and Surgery from King Saud University in 1994. Pediatric Residency training Program between July, 2000 -June, 2003 at University of B.C. Vancouver, Canada. He received Fellowship training in Bone Marrow Transplant and Primary Immunodeficiency at Hospital for Sick Children, University of Toronto, Canada. He also received American Board of Allergy and Immunology (ABAI) in Oct 2006. Since 2007 till present he worked as Consultant allergy, immunology and BMT at King Abdulaziz Medical City-WR. Jeddah Saudi Arabia.

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