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Burkholderia pseudomallei Down-Regulates Host Defense Gene Expression in Non-phagocytic A549 Cells

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Melioidosis is an endemic disease found in many tropical countries of South East Asia and Northern Australia. This disease is caused by *Burkholderia pseudomallei*, a Gram-negative soil saprophytic bacterium known to invade, survive and multiply in both phagocytic and non-phagocytic cells. Currently, limited information is available regarding the mechanism of host response against this bacterium. In this study, we have utilized the microarray technology to understand the complex interplay between *B. pseudomallei* and its host. A549 human lung epithelia cells were exposed to *B. pseudomallei* clinical isolate R15/05 as well as a less virulent animal isolate Sheep 4523/98 with a M.O.I of 10:1 for 3 and 18 hours. Using the Illumina Sentrix Human-8v2 Expression BeadChip, we monitored changes in gene expression in host cells after exposure to *B. pseudomallei*. Analysis of microarray data showed 39 genes differentially expressed in both the clinical and animal isolate, 3 and 18 hours post infection. Among these 39 genes, many of those involved in the host defense response were down-regulated. These included several cytokines, chemokines as well as genes involved in the NFkB and STAT signaling pathways. The down-regulation of these defense genes might be attributed to the host's response to prevent inflammation in order to survive the pathogen invasion. In addition, this phenomenon might also reflect the ability of *B. pseudomallei* to suppress host cell's defense response, either by manipulating the host innate defense system or interfering with associated signaling pathways. Analysis of the microarray data has helped to shed new light on the *B. pseudomallei* infection process and survival strategy inside its host cell.

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Relationship Between IgeE Antibodies to the *Staphylococcus aureus* Enterotoxin B (SEB) with the Severity of Atopic Dermatitis in Children

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Background: The skin of patients with atopic dermatitis (AD) exhibits a striking susceptibility to colonization and could function as classic allergens, inducing production of functionally relevant specific IgE antibodies. The aim of this study was to determine the relationship between IgE antibodies to the *Staphylococcus aureus* enterotoxin B (SEB) with the severity of Atopic Dermatitis in children.

Methods: In a cross-sectional study of 30 children with atopic dermatitis, AD is diagnosed based on standard criteria Hanifin and Rajka, clinical severity of AD was determined by the SCORAD index. Specimen for *S. aureus* culture was isolated from 3 different areas of the skin. Total serum IgE and IgE specific to SEB was measured by ImmunoCAP system.

Results: Thirty children, 19 male and 11 female, aged from 3 months to 8 years with AD entered this study. Five of 30 children were sensitized to SEB. The degree of disease severity correlated to a higher extent with the presence of SEB-specific antibodies. Among patients 13.7% were colonized with *S. aureus* producing staphylococcal enterotoxins B. Children colonized with toxigenic S. aureus strains had higher disease severity [SCORAD index of 65.1 ± 18.5 in positive SEB versus 23.9 ± 16.9 in negative SEB group ($P = 0.005$)].

Conclusion: Our results demonstrate a relationship between severity of disease in AD patients and IgE antibodies to SEB. Sensitization to *S. aureus*-derived superantigens may be involved in disease exacerbation. The presence of SEB-specific antibodies had additional explanatory value for disease severity and therefore may be helpful in the characterization of children with severe atopic dermatitis. It is recommended that all Atopic Dermatitis patients be considered for *Staphyloccocus aureus* culture, *Staphyloccocus aureus* Antibody evaluation and prophylactic antistaphylococcal treatment especially in severe cases.

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Pre- and Intra-Operative Risk Factors Which Influence Early Outcome in Infective Native and Prosthetic Aortic Valve Endocarditis. A Single Center Study

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Background: In-hospital mortality in patients suffering for infective native and prosthetic aortic valve endocarditis is still high. Purpose of this study was to identify pre-operative and intra-operative predictors of early outcome.

Methods: Between January 2004 and December 2006, 75 patients, mean age 61.6 ± 14.1 years (range 19-85 years), underwent surgical treatment for infective native or prosthetic aortic valve endocarditis. Patients were identified after the modified Duke and Renzulli criteria for infective endocarditis. Pre- and intra-operative variables were analy-