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Biomarkers and psoriasis

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Psoriasis is a chronic inflammatory skin disease mediated by the cells and molecules of both the innate and adaptive immune systems that involves red elevated patches and flaking silvery scales. Despite intensive research, psoriasis pathogenesis remains unknown. Our purpose was to study the role of nickel in psoriasis using microarray gene expression data. The unexpected outcome from this study was the role of metronidazole in the treatment of psoriasis. Six psoriasis microarray assays were downloaded from the GEO database. Statistical tests have been done on both normalized and nonnormalized data. We used KEGG, Reactome and Biosystems for pathway analysis and RGD for gene-chemicals interactions. Nickel upregulates the top upregulated genes in psoriasis including AKR1B10, IL36G, SERPINB4, KYNU, SERPINB3, TCN1, DEFB4A, HPSE, PI3, SPRR2C, SPRR3, VNN3 and several S100 family members without downregulating any of those upregulated genes. Nickel also downregulates the top downregulated genes such as KRT77, ID4, BTC, CCL27, CHP2, IL37 and RORC without

upregulating any of those downregulated genes. The strongly upregulated pathways included immune response, defense response (e.g., amebiasis), cell cycle and metabolic pathways and the top downregulated pathways included inflammatory bowel disease, keratins, ErbB, Chemokine, cytokine and TGF-beta pathways. Based on the best of our knowledge, this is the first study that highlighted the role of nickel in psoriasis pathogenesis using microarray gene expression data. The significant and unique effect of nickel in upregulating the upregulated genes and downregulating the downregulated genes in psoriasis and a strong affinity between the imidazole ring were an indication of a possible dramatic effect of metronidazole on improving psoriasis skin inflammation in our limited case study. Using microarray data, we showed that recognition of the role of abnormal nickel concentration could point the way to greater understanding of psoriasis pathogenesis.

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