Vitamin B6 decreases skin inflammation in a zebrafish model of psoriasis

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Psoriasis is a skin inflammatory disorder that affects 1-3 % of the human populations. Although several therapies based in the neutralization of proinflammatory cytokines have been used with relative success, they have important side effects. Therefore, additional treatments are required. We have developed a zebrafish model of skin inflammation based in the transient genetic inactivation of the Tnfa/Tnfr2 axis. Using a metabolomic approach in this model, we have found reduced levels of vitamin B6 and increased ones of cystathionine, a marker of vitamin B6 deficiency.

The main objective of this study was determining if vitamin B6 was able to inhibit skin inflammation in Tnfr2-deficient zebrafish larvae.

Tnfr2-deficient zebrafish larvae were treated by bath immersion with 0-10 μM Vitamin B6 at 24hours post-fertilization (hpf) in the presence of 1% DMSO to facilitate adsorption. Skin inflammation was then evaluated at 72 hpf by checking neutrophil dispersion and the expression of genes encoding pro-inflammatory mediators by RT-qPCR.

We observed that vitamin B6 was able to reduce neutrophil dispersion of Tnfr2-deficient larvae in a dose-dependent manner. Studies are in progress to evaluate the mechanism involved in the regulation of skin homeostasis by vitamin B6.

The results of our study point out to vitamin B6 and cystathionine as prognosis markers for psoriasis and suggest that dietary intake of vitamin B6 may have a profound impact on this disease.