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# Strong Doubts on the Causative Role of Cold Medicines

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## Editorial

After 60 years of work, there is now, for the first time, data to suggest that the high fatality rates of EN can be reduced by adding cyclosporine A to sophisticated supporting measures in specialist settings. 2-4 This suggests that going forward, the goal in the therapy of EN patients should not only be life but also "survival without severe disability." Actually, the most common serious consequences of EN are ocular lesions. They can continue to be active for years, and they frequently cause severe disability like blindness. With decreasing mortality in the acute phase of EN, efforts should now focus on risk factors, mechanisms, prevention, and treatment of sequelae, especially those affecting the eyes. The same team was the first to note that young patients with EN linked to "cold medicines" had more serious ocular lesions and that, in the Japanese population, the risk was linked to polymorphism of enzymes involved in the "prostaglandin cascade," [1].

HLA-A*02:06 and HLA-B*44:03 in particular. The present publication also reports, in another population, that more severe chronic ocular complications are observed among EN cases attributed to NSAIDs or “cold medicines.” It does not include investigation of possible HLA associations. The most significant findings of the current and earlier articles are that younger patients had a higher risk of severe ocular sequelae, at least in Japan, with an HLA relationship that needs to be verified in other countries. We do not question these findings in any way. We vehemently contest the notion that most of these patients' cases of EN were caused by "cold drugs." Appropriate causality determination is crucial in many ways for drug-induced disorders in general, especially for the more severe ones like EN. Patients need to be shielded against the possibility of repeat exposure. Correct risk estimation is necessary for regulatory organisations and drug corporations to assess the "benefit/risk" ratio of pharmaceuticals. Only after identifying the substance most likely to cause widespread epithelial cell death is it possible to analyse drug-specific immune cytotoxicity *in vitro* [2,3].

All recent developments on HLA and the risk of EN (e.g., carbamazepine and HLA B*15:02 in Southeast Asia, allopurinol and B*58:01 globally) called for the correct identification of the drug that causes the EN. For more than 30 years, we have contributed several extensive studies to the evaluation of drug causality in EN. In 1984, in an early series of 50 cases, the 3 drugs most often taken by the patients before hospital admission were phenylbutazone (PBZ), sulfamethoxazole (SMX), and acetyl-salicylic acid (ASA).9 Prior reports had suggested that PBZ and SMX could induce EN, but that was not the case for salicylates [4].

Additionally, the pattern of exposure to these drugs prior to the commencement of the disease was noticeably different. For PBZ and SMX, the median exposure time was 15 days; for ASA, it was less than 3 days. The authors put forth the theory that ASA might be an unwitting bystander, utilised as a result of early signs like fever or pain before a disease fully manifests itself. One of the first requests from the methodology team was the use of strict criteria for the definition of disease onset, blinded to any information on exposure to medications. The same team was the first to report that young patients with EN attributed to “cold medicines” had more severe eye lesions and that, in Japanese population, the risk was associated with HLA-A*02:06 and HLA-B*44:03, and with polymorphism of enzymes implicated in the “prostaglandin cascade” [5].

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## Conflict of Interest

The Author declares there is no conflict of interest associated with this manuscript.

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