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Short Communication

# In a Thai population, HLA Markers of Drug Hypersensitivity

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## ABSTRACT

SCARs, such as Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and drug reactions with eosinophilia and systemic symptoms (DRESS), are potentially life-threatening cutaneous reactions triggered by a number of medications. Due to the hemolytic anemia caused by ribavirin, which increased the number of blood transfusions, this therapy has been restricted to thalassemia and SCD patients. HLA alleles, which encode human antigen-presenting proteins, have recently been discovered to be valid pharmacogenetics markers for predicting these life-threatening reactions.

**Keywords:** SCARs; Pharmacogenetics; Markers

## INTRODUCTION

Type A and type B adverse drug reactions are the two main forms of adverse drug reactions. Adverse drug reactions of type A are usually linked to the drug's mechanism of action and dosage. Type B adverse drug reactions, on the other hand, are unpredictable reactions that occur only in susceptible individuals and are usually unrelated to the drugs' mechanism of action. Despite the fact that type B adverse drug reactions are less common, they are more serious than type A adverse drug reactions [1].

The most common adverse drug reactions in type B are cutaneous adverse drug reactions. The severity of cutaneous reactions triggered by drugs can vary from mild cutaneous reactions like maculopapular rash and urticaria to life-threatening Extreme Cutaneous Adverse Reactions (SCARs) like Stevens–Johnson Syndrome (SJS), Toxic Epidermal Necrolysis (TEN), and drug reactions with eosinophilia and systemic symptoms like Stevens–Johnson syndrome (SJS), toxic epidermal necrolysis, and drug reactions with eos [2]. SJS and TEN are cutaneous reactions of the same etiology, but the degree of skin detachment relative to body surface area (BSA) differs only in SJS, where it is restricted to less than 10% of BSA, while in TEN it is widespread, accounting for more than 30% of BSA. Several lines of evidence indicate that human leukocyte antigen (HLA) gene polymorphisms can play a significant role in an individual's susceptibility to these life-threatening SCARs. [3].

Several HLA alleles have been found to be closely linked to SCARs, and some have been suggested as legitimate genetic markers for predicting these life-threatening reactions. Since the associations between SCARs and HLA alleles are unique to occlusion, hepatic sickle cell cholestasis, liver ischemia and specific alleles of the HLA gene, high resolution DNA typing is an effective tool for identifying these pharmacogenetics markers. The frequency of these pharmacogenetics HLA alleles, particularly 2-field data (4-digit resolution) [4], are critical parameters for estimating the size of the population at risk for drug-induced SCARs. Allergies and autoimmunity are examples of hypersensitivity reactions caused by the normal immune system. These reactions are generally referred to as an immune system overreaction, and they can be negative, unpleasant, or even fatal. A pre-sensitized (immune) state of the host is needed for hypersensitivity reactions. The most commonly used concept of hypersensitivity is Gell and Coombs, which identifies four forms of immune responses that cause tissue harm in bystanders. An immune-mediated reaction to a drug is known as drug hypersensitivity. Rashes, anaphylaxis, and serum sickness are among the moderate to extreme symptoms. The diagnosis is made clinically, with the aid of skin testing on occasion. Drug discontinuation, supportive care (e.g., antihistamines), and occasionally desensitization are all options for treatment. Some protein and large polypeptide drugs (for example, insulin and therapeutic antibodies) can stimulate antibody development directly. Many drugs, including peptides embedded in major histocompatibility complex (MHC) molecules; act as haptens, binding covalently to serum or cell-bound proteins. The binding makes the protein immunogenic, causing the formation of antidrug antibodies, T-cell responses to the grug, or both. Haptens can also bind directly to the MHC II molecule, causing T cells to become activated. Some medications have a prohapten effect. Prohaptens become haptens when they are metabolized; for example, penicillin is not antigenic in and of itself, but its key degradation product, benzylpenicilloic acid

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can combine with tissue proteins to form benzylpenicilloyl (BPO), which is a major antigenic determinant [5].

## CONCLUSION

Drug hypersensitivity is a natural occurrence, but it can be a daunting problem for most physicians. Many clinicians have concluded that the only appropriate choice for drug-reactive patients is permanent and absolute avoidance of putative criminals because immunodiagnostic tests for drug allergy are small in number and require some sophistication to interpret. Patients with multiple drug hypersensitivity syndromes have been known to be abandoned by their primary care physicians or ordered to stop taking all medications. Reactions to emerging drugs such as quinolone derivatives and radiocontrast media are becoming more common.

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