

LETTER TO THE EDITOR

CORRELATION BETWEEN SERUM IgE AND SALIVARY CORTISOL LEVELS IN SUBJECTS WITH TYPE I ALLERGIC DISORDERS

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This study compares the relationship between serum IgE and salivary cortisol levels in 42 normal and in 18 type I allergic subjects. Levels of serum total IgE and salivary cortisol were determined with the UniCAP system and ELISA respectively. In the type I allergic subjects, there was a significant correlation between serum IgE and salivary cortisol levels ($p < 0.01$). In the normal subjects, on the other hand, no correlation was found. These findings suggest that there may be an association between stress and allergic disorders.

Defined as “immune-mediated tissue injury,” allergic reactions fall into four categories (1). We compare the relationship between serum IgE and salivary cortisol levels in normal and in type I allergic subjects, who showed typical allergic responses resulting from IgE-dependent degranulation of mast cells. This type of reaction is also referred to as immediate or anaphylactic, reaginic or atopic allergy and presents as atopic dermatitis, allergic rhinitis, bronchial asthma and other ailments.

MATERIALS AND METHODS

For this study, we recruited 60 male workers (mean $\pm$ SD, 44.7 $\pm$ 9.3 yrs) employed at a Japanese metal plant. Eighteen (44.2 $\pm$ 9.1 yrs) were type I allergic subjects who had symptoms or positive histories of atopic dermatitis, allergic rhinitis, bronchial asthma or urticaria. None presented evidence of any other diseases, and reported the regular use of any medication.

To avoid any effect of injection stress on cortisol

levels, saliva sampling was conducted prior to blood sampling. The saliva samples were collected directly into test tubes for two minutes, after which, blood samples were obtained from forearm veins. All samples were collected between 9 and 10 a.m. and were stored at -80°C until the assay.

The salivary cortisol levels were determined with a commercial enzyme immunoassay kit (CIRON, Tokyo, Japan) (2). The serum total IgE levels were determined with the UniCAP system (Pharmacia & Upjohn Inc., Tokyo, Japan) according to previously described methods (3).

The statistical analyses were performed using Student's *t*-test, Pearson's correlation coefficients, and χ -square test.

RESULTS

Table I shows the relationship between serum IgE and salivary cortisol levels in each group. In the type I allergic and the normal subjects, the salivary cortisol levels (mean $\pm$ SD) were 0.22 $\pm$ 0.13

Key words: type I allergy, IgE, cortisol, stress

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0394-6320 (2007)

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Table I. Relationship between salivary cortisol and serum IgE levels in subjects with type I allergies and in normal subjects.

	n	Salivary cortisol levels, µg/dl	Serum IgE levels, IU/ml	Pearson's correlation coefficients
Type I allergic subjects	18	0.22±0.13	584.6±849.5	0.61**
Normal subjects	42	0.37±0.47	189.9±387.6	-0.05
<i>p</i> value (Student's <i>t</i> -test) ^a		0.07	0.07	

Values are expressed as mean±SD.

** $p < 0.01$.

^aType I allergic subjects vs. normal subjects

and 0.37±0.47 µg/dl, and the serum IgE levels were 584.6±849.5 and 189.9±387.6 IU/ml, respectively. There was no significant difference in salivary cortisol or serum IgE levels between allergic patients and controls.

In the type I allergic subjects, there was a significant correlation between salivary cortisol and serum IgE levels ($p < 0.01$). In the normal subjects, on the other hand, no correlation found.

DISCUSSION

An abrupt increase in the cumulative prevalence of allergic diseases in individuals with IgE levels of more than 400 IU/ml has been reported (4) and, in the present study, we found that many (7 of 18, 38.9 %) of the type I allergic subjects had IgE levels of more than 400 IU/ml (normal subjects, 3 of 42, 7.1 %; $\chi^2 = 9.14$, $p < 0.01$). Nonetheless, there was no significant difference in serum IgE levels between allergic patients and controls ($p = 0.07$). This may have resulted from low sample size, especially for the allergic patients. Larger populations are needed for future studies.

In the present study, the salivary cortisol levels were to some extent ($p = 0.07$) lower in the type I allergic subjects than in the normal subjects. It is possible that their adrenal glands do not function normally, resulting in deficient secretions of steroids (5). Moreover, other studies indicate that endogenous glucocorticoids suppress inflammatory responses, including IgE-dependent allergic reactions (6).

Our findings suggest that, in type I allergic subjects, although the salivary cortisol levels tend to be suppressed, within the suppressed levels, they correlate with the serum IgE levels. This may be mediated by stress. The stressor-stress response results in activation of the HPA

axis, which can be detected physiologically by measuring cortisol levels. Moreover, recent progress in psychoneuroimmunology has revealed a relationship between type I allergic disorders and psychological disturbances (7). Other studies have indicated that allergic reactions may be enhanced by a direct effect of peripheral corticotropin-releasing hormone or mast cells (8). Such findings suggest that there may be a relationship between allergic disorders and dysfunction of the HPA axis. Our present findings suggest that, if individuals can be made aware of stress and learn how to control their stress reactions, they may be able to reduce the severity of their allergic disorders.

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