

## Lysozyme and IgA Values in Patients with Atopic Dermatitis

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**Ten patients with atopic dermatitis had significantly depressed lysozyme levels in saliva, compared with controls, whereas no differences were found in lysozyme activity in serum of patients and controls. The concentrations of IgA in saliva of patients with atopic dermatitis were also significantly lower than in controls, whereas IgA in patients' serum was within normal levels.**

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Atopic patients are predisposed to a number of complications that may result from accompanying T-cell dysfunction and metabolic defects. Patients with atopic dermatitis (AD) are prone to pyoderma due primarily to *Staphylococcus aureus* and beta-hemo-

lytic streptococci. Viral diseases, e.g. herpes simplex, may cause serious complications in patients with AD, and molluscum contagiosum and common warts tend to spread more extensively. Moreover, patients with atopic disease are susceptible to scabies infection (1).

Lysozyme is a bacteriolytic enzyme widely distributed in human tissues and secretions and is assumed to possess a multifarious physiologic role. This enzyme can modulate inflammatory reaction, e.g. induce human granulocytes to increased phagocytosis (2) and influence the function of lymphocytes (3). In vitro investigations have demonstrated that lysozyme can inactivate viruses (4) and also that it has an antifungal potential (5).

The importance of IgA as a surface protective factor in all internal body secretions has been well documented in recent years.

The aim of the present study was to determine the levels of lysozyme and IgA in patients with AD.

## MATERIALS AND METHODS

### Patients

Ten patients, aged 13 to 27 years (average 20.6 years), with typical clinical characteristics of severe AD were studied. Criteria for the diagnosis were those published by Hanifin & Rajka (6). None of the patients was receiving corticosteroids, antihistamines or other systemic therapy or phototherapy at the time of the study. None of the patients had evident systemic disease.

Ten healthy young students with no history of atopic disease served as controls. None was receiving any local or systemic therapy.

### Serum preparation

Blood samples were drawn by venipuncture and, after clotting for 30 min at room temperature, the sera were separated and stored at  $-20^{\circ}$  until required.

### Saliva preparation

Saliva was taken at morning before breakfast and tooth-brushing and were placed in plastic tubes and stored at  $-20^{\circ}\text{C}$ .

### Lysozyme determination

Lysozyme activity was measured by a lyso-plated method previously described (7). Shortly, 40  $\mu\text{l}$  of the solution to be tested were applied in wells in 1% agarose (Litex, Denmark) containing 0.5 g/l dried *Micrococcus lysodeicticus* (Sigma Chemical Company, USA). Both agarose and lysozyme standard were dissolved in 15 M phosphate buffer, pH 6.3. After incubating the agar plates at  $37^{\circ}\text{C}$  for 22 h, the diameters of the zones were compared with a standard curve obtained with hen egg-white lysozyme.

### IgA determination

The determination of the IgA concentrations in the sera and saliva was done using the Beckman Errey Proteins System (ABK TEK Company, Norway).

Statistical significance was calculated using Student's *t*-test.

## RESULTS

The serum and saliva lysozyme values determined in patients with AD and in controls are given in Fig. 1. The lysozyme concentration in AD patients' saliva was 11.2  $\mu\text{g/ml}$  (10.0–12.5  $\mu\text{g/ml}$ ) compared with 13.3  $\mu\text{g/ml}$  (12.0–17.0  $\mu\text{g/ml}$ ) in controls, a statistically significant difference ( $p < 0.01$ ). By contrast, the lysozyme activity in AD patients' serum was 10.8  $\mu\text{g/ml}$  (9.5–12  $\mu\text{g/ml}$ ) compared with 10.6  $\mu\text{g/ml}$  (10.0–11.5  $\mu\text{g/ml}$ ) in controls, which did not reach statistical significance.

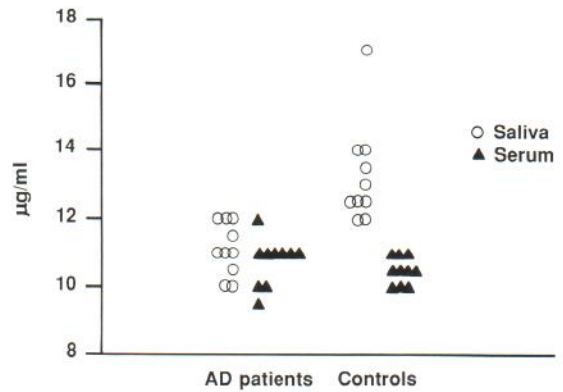


Fig. 1. Lysozyme levels in AD patients vs. controls.

The concentrations of IgA in saliva were 7.48 mg/dl (6.67–9.42 mg/dl) in AD patients, compared with 9.49 mg/dl (6.67–17.3 mg/dl) in controls (Fig. 2), a statistically significant difference ( $p < 0.02$ ). IgA levels in serum of the patients with AD, on the other hand, were within normal limits.

## DISCUSSION

The markedly decreased lysozyme levels found in saliva in patients with AD are hard to explain. Lysozyme found in saliva and other secretions is probably synthesized by glandular cells (8), whereas serum lysozyme in normal subjects is thought to represent enzyme liberated from dying polymorphonuclear leukocytes (9). The fact that lysozyme values in serum of AD patients did not differ from that found in healthy controls, disproves a leakage of lysozyme from the serum to the saliva. The reduced levels in saliva may therefore either be due to a decreased production or may reflect a higher consumption of lysozyme. Reduced lysozyme levels have been found in the skin (10) and in saliva (11) of diabetic patients, whereas serum lysozyme levels were the same in patients and controls.

The present study showed decreased values of IgA in saliva of patients with AD, whereas the corresponding serum levels of IgA were the same in patients and controls. Both IgA and lysozyme are normally present in abundance in saliva, tears, urine, tracheobronchial secretions and gastric juice, and only in small amounts in serum, pleural and cerebrospinal fluid (8). Metzger et al. (12) demonstrated that IgA is present in human skin and secreted by normal human sweat and sebaceous glands. The authors postulate that IgA contributes as a humoral



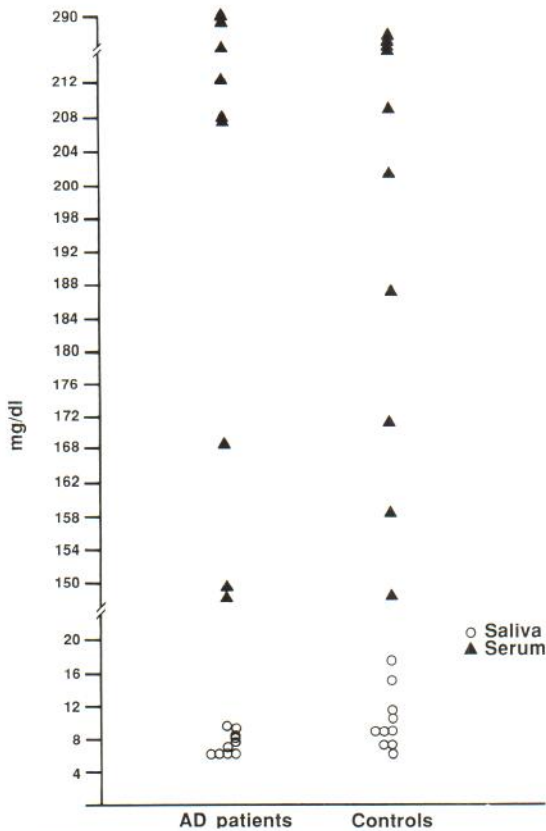


Fig. 2. IgA concentrations in patients with AD vs. controls.

component to the established cellular defence system of the skin. IgA deficiency has been found more frequent in atopic subjects (13), and one of us found a significantly lower serum concentration of IgA during scabies infection than after treatment (1).

The role of IgA as a protective factor in the inactivation of microorganisms is well known, and lysozyme synthesized in human epidermis is believed to protect skin against bacterial infections (14). Therefore, the decreased levels of both lysozyme and IgA in saliva may be one explanation for the well-known susceptibility of AD patients to many cutaneous bacterial, viral and fungal infections.

The present observations give no answer as to

whether the reduced lysozyme activity and reduced IgA values in saliva represent a specific immunological reaction in AD patients, or if they may be found in other skin diseases. Further investigations along these lines are in progress.

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