Isoprostane Characteristics in Sick House Syndrome, Atopy and Asthma Patient

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Abstract

An imbalance between oxidants and antioxidants, in favor of oxidants leading to oxidative stress, is known to play an important role in the pathogenesis of various diseases. Isoprostanes are structurally stable isomers of the conventional enzymatically derived prostaglandins, which are produced *in vivo* primarily by a free radical catalyzed peroxidation of polyunsaturated fatty acids. In asthmatics, disease severity can occur from environmental exposure to air pollution. Some surveys suggested that air pollutants, especially diesel-exhaust particulates, could trigger allergic sensitization and development of atopic diseases. Sick house syndrome (SHS) presents healthy damage owing to the indoor environment of a building. The aim of this study was to examine isoprostane as a parameter for oxidative stress in environments related diseases such as sick house syndrome, atopy and asthma. We measured plasma and urinary levels of isoprostane from health volunteers, sick house syndrome, atopy and asthma patients. Plasma isoprostane concentrations in asthma and sick house syndrome group were significantly higher than in control. Urinary isoprostane levels were significantly higher in volunteers with sick house syndrome and asthma compared with health volunteers. These findings suggest that plasma and urinary isoprostane measurement may have useful clinical implications for investigating sick house syndrome and asthma. The interventions that decrease exposure to environmental reactive oxygen species might be beneficial in these diseases.

Introduction

Oxidative stress occurs the production of oxidants exceeds the capacity of the body's antioxidant defenses to detoxify them. An imbalance between oxidants and antioxidants, in favor of oxidants leading to oxidative stress, is known to play an important role in the pathogenesis of various diseases. Definitive evidence for this association is often lacking because of recognized shortcomings with methods previously available to assess oxidative stress status *in vivo*. Isoprostanes are structurally stable isomers of the conventional enzymatically derived prostaglandins, which are produced *in vivo* primarily by a free radical catalyzed peroxidation of polyunsaturated fatty acids. Isoprostane appear in the plasma and urine under normal conditions

and are elevated by oxidative stress. At least one of the isoprostane, 8-isoprostane (8-isoPGF$_{2\alpha}$), has been shown to have biological activity. It is a potent renal vasoconstrictor and has been implicated as a causative mediator of hepatorenal syndrome.

Asthma is a chronic inflammatory airway disease that affects children and adults of all ages. Although the pathogenesis of asthma remains incompletely defined, it has been shown to be a state of increased free radical formation. In asthmatics, oxidative stress occurs not only as a result of disease seriousness but also from environmental exposure to air pollution. Some surveys suggested that air pollutants, especially diesel-exhaust particulates, could trigger allergic sensitization and development of atopic diseases. In animal as well as in human experiments, diesel exhaust particulates are able to trigger an IgE-response. Epidemiological surveys also show that air pollutants trigger symptoms in patients. Sick house syndrome (SHS) presents healthy damage owing to the indoor environment of a building.

The aim of this study was to examine isoprostane as a parameter for oxidative stress in environments related diseases such as sick house syndrome, atopy and asthma. We measured plasma and urinary levels of isoprostane from health volunteers, sick house syndrome, atopy and asthma patients.

Materials and Methods

Plasma and urine sample were collected in the morning after 12hour starvation. Whole blood in lithium heparin was taken from each subject and immediately centrifuged at 1600g. Erythrocytes and plasma were separated and stored at -70°C until biochemical measurements taken. 8-isoprostane levels in plasma and urinary were measured by enzyme-linked immunosorbent assay. 50ul of plasma or urine was added in 96 well plate. Each sample was assayed at 3 dilutions. Each dilution was assayed in duplicate. We added 50ul of 8-isoprostane AChE tracer. 50ul of 8-isoprostane antiserum was added to each 96 well. The plate was covered with plastic film and incubated for 18 hours at room temperature. The contents in the 96 wells were removed and rinsed five times with wash buffer. We added 200ul of Ellman's Reagent to each well and covered the plate with plastic film. After 60-90 minutes incubation, we read the plate at a wavelength between 405 and 420 nm.

Results and Discussion

We measured plasma and urinary levels of isoprostane from health volunteers, sick house syndrome, atopy and asthma patients. Plasma isoprostane concentration in asthma and sick house syndrome group were significantly higher than in control group (Fig. 1.). No differences between the control and atopy group were observed for isoprostane.

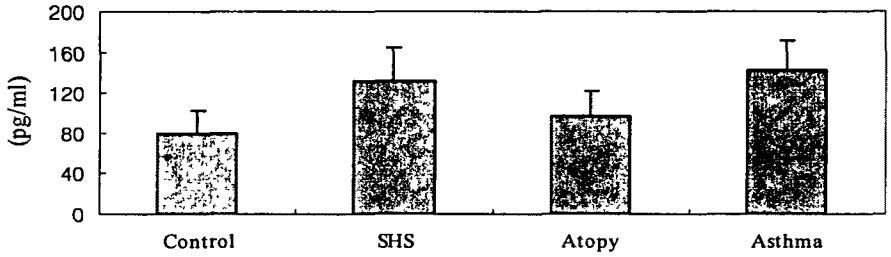

Fig. 1. Levels of isoprostane in plasma of sick house syndrome, atopy and asthma patients. Values are the mean ± standard deviation.

Urinary isoprostane levels were significantly higher in volunteers with sick house syndrome and asthma compared with healthy volunteers (Fig. 2). There were no differences between the control and atopy group. Oxidative stress is believed to play an important role in the pathophysiology of sick house syndrome and asthma.

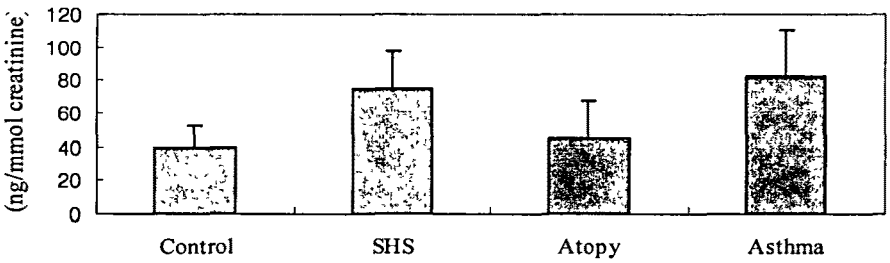

Fig. 2. Levels of isoprostane in urine of sick house syndrome, atopy and asthma patients. Values are the mean ± standard deviation.

Conclusion

Our study shows that plasma and urinary isoprostane concentrations are higher in sick house syndrome and asthma patients. These findings suggest that plasma and urinary isoprostane measurement may have useful clinical implications for investigating sick house syndrome and asthma. Therapeutic interventions that decrease exposure to environmental reactive oxygen species might be beneficial as adjunctive therapies in asthmatic and sick house syndrome patients.

Acknowledgment

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