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Microwave radiation from cellular phones increases allergen-specific IgE production

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Key words: allergen-specific IgE production; atopic eczema dermatitis syndrome; cellular phone; microwave; Th1 and Th2 cytokine.

Exposure to microwave radiation caused neurological symptoms in humans, while it caused chromosomal instability in lymphocytes (1, 2). Microwave radiation from cellular phones enhanced allergic responses in patients with atopic eczema dermatitis syndrome (AEDS), while it failed to do so in normal subjects (3). Here, the effects of microwave radiation from cellular phones on allergen-specific IgE production were studied.

After obtaining informed written consent, 30 mild AEDS patients [mean SCORAD index 9 (95% CI: 8–10)] (15 women and 15 men, mean 28 years, range 22–32 years) were studied (4). AEDS patients were treated with application of mixture of pinetar (Yoshida

Pharmaceutical Co. Ltd, Tokyo, Japan) and zinc oxide ointment, but without oral medication. All of AEDS patients were positive for latex, but not to Japanese cedar pollen (JCP), by skin prick test and serum allergen-specific IgE. The serum levels of latex-specific IgE and JCP-specific IgE in AEDS patients were 27.4 and <0.34 IU/ml, respectively.

Randomly assigned 15 AEDS patients first viewed a weather information video, while putting cellular phones to the right ear for 30 min with transmitting but without sound (microwave radiation study). The model of cellular phone used was C5001T (Toshiba, Tokyo, Japan; specific absorption rate = 1.62). After 2 weeks, they viewed a weather information video while putting same cellular phones to the right ear for 30 min, but phone was turned off (control study). On the other hand, other 15 patients first conducted the control study, and after 2 weeks they conducted the microwave radiation study. None of the subjects were aware whether cellular phones had transmitting or not. Just before and immediately after each study, blood was sampled by medical staff who were blinded to the study.

Mononuclear cells from blood were separated, and they were cultured ($4 \times 10^4/0.2$ ml/well) with medium, and 0.1 ng/ml of latex or JCP (TORII PHARM, Co., Ltd, Tokyo, Japan) was added to each culture individually. After 14 days of culture, each allergen-specific IgE production was determined by commercial ELISA, while after 5 days of culture, cytokine production was determined by ELISA (R&D Systems, Inc., Minneapolis, MN) (4). Statistical analysis was performed ANOVA with Bonferroni's correction. This study was approved by the Ethical Committee of Ujitakeda Hospital.

Nonexposure to microwave radiation (control study) had no effect on allergen-specific IgE production, while exposure to microwave radiation (microwave radiation study) significantly increased allergen-specific IgE production (Table 1). This increase was selective to allergen to which AEDS patients were allergic, as exposure to microwave radiation increased

Microwave radiation from cellular phones increases allergen-specific IgE production in patients with AEDS.

Table 1. Effect of exposure to microwave radiation on production of allergen-specific IgE and cytokine in patients with AEDS

Stimulus	<i>In vitro</i> production			
	Control study		Microwave radiation study	
	Before	After	Before	After
(a) Latex				
Latex-specific IgE	4.6 (3.9–5.3)	4.5 (3.8–5.2)	4.3 (3.7–4.9)	7.3 (6.4–8.2)*
IFN- γ	358 (334–380)	370 (351–389)	339 (314–354)	193 (183–203)**
IL-12	299 (281–317)	308 (288–328)	285 (269–301)	151 (137–165)**
IL-4	937 (861–1013)	919 (839–999)	926 (830–1022)	1431 (1308–1534)*
IL-13	343 (316–360)	321 (290–352)	336 (307–3369)	510 (483–537)*
(b) Japanese cedar pollen				
JCP-specific IgE	<0.3	<0.3	<0.3	<0.3
IFN- γ	43 (35–51)	46 (39–53)	49 (41–57)	45 (38–52)
IL-12	41 (32–50)	42 (33–52)	43 (33–53)	44 (33–55)
IL-4	51 (43–59)	54 (44–64)	58 (48–68)	56 (47–65)
IL-13	40 (32–48)	41 (33–49)	38 (30–46)	36 (28–34)

Patients with AEDS were either not exposed (control study) or exposed to microwave radiation (microwave radiation study). Before and after study, mononuclear cells were stimulated with latex (a) or Japanese cedar pollen (JCP) (b), and production of latex-specific IgE (ng/ml) (a) or JCP-specific IgE (ng/ml) (b), or various cytokines (pg/ml) (a, b) was measured. Values are median (95% CI).

*Significant decrease ($P < 0.01$) compared with before.

**Significant increase ($P < 0.01$) compared with before.

latex-specific IgE production, while it failed to induce JCP-specific IgE production.

The amount of allergen-specific IgE production was dependent on balance of Th1 cytokine (IFN- γ and IL-12) and Th2 cytokine (IL-4, IL-13) (4). Thus, the effect of microwave radiation on cytokine production was studied. Latex induced IFN- γ , IL-12, IL-4 and IL-13 production in AEDS patients, while JCP failed to do so. Exposure to microwave radiation significantly decreased latex-induced IFN- γ and IL-12 production, while it significantly increased latex-induced IL-4 and IL-13 production. In contrast, microwave radiation had no effect on production of these cytokines by JCP. We also studied the effect of microwave radiation on normal subjects. However, peripheral blood mononuclear cells from normal donors, who were exposed to microwave radiation, failed to induce latex-specific IgE production (<0.3 ng/ml) by latex or JCP-specific IgE production (<0.3 ng/ml) by JCP. It also failed to modulate production of

IFN- γ , IL-12, IL-4 or IL-13 stimulated by latex or JCP (data not shown).

Collectively, these findings may have some implication for the study of immunological effect of microwave radiation from cellular phones on allergic diseases. However, further study is necessary to elucidate the exact mechanisms.

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Occupational allergy to raw beef due to cross-reactivity with dog epithelium

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Key words: contact urticaria; cross-reactivity; dog allergy; meat allergy; occupational allergy; serum albumin.

The association between allergy to epithelia and mammalian meat has been previously reported (1, 2). Most of the authors describe serum albumin to be responsible for this cross-reactivity (3, 4).

A dog-allergic cook showed cutaneous and respiratory symptoms when exposed to raw beef.

We present the case of a 28-year-old asthmatic man sensitized to dog epithelium, who developed wheezing symptoms when exposed to raw beef. He worked as a cook and tolerated the ingestion of cooked beef. He also presented contact urticaria after contacting with raw beef. Symptoms appeared mostly during defrosting procedures. The patient avoided any contact with dog presenting symptoms only when in contact with beef.

Skin-prick tests to common allergens were positive to dog and cat danders and negative to pollens (grasses, weeds and trees), house dust mites (*Dermatophagoides pteronyssinus*, *Lepidoglyphus*,