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Linking Peanut Allergenicity to the Processes of Maturation, Curing, and Roasting

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The processes of peanut maturation, curing, and roasting are known to have an important role in peanut flavors. One of these processes (i.e., roasting) has been found to have an effect on allergenicity. To determine if the other processes (i.e., maturation and curing) affect allergenicity, mature and immature roasted peanuts and peanuts cured at different temperatures (35−77 °C) were, respectively, tested for IgE binding and advanced glycation end adducts (AGEs). Peanuts with and without stress proteins, which are associated with peanut maturation and curing, were also tested. Results showed that mature roasted peanuts exhibited a higher IgE binding and AGEs level than immature roasted peanuts. Curing temperatures between 35 and 60 °C gave no difference in the profiles. However, a higher curing temperature (i.e., 77 °C) exhibited a profile of higher levels of AGEs and IgE binding. These levels were higher in peanuts with stress proteins than without stress proteins. Roasting increased stress protein level and IgE binding. From these results, the processes of maturation and curing, in conjunction with roasting, may be associated with allergenicity, suggesting that these processes may lead to changes in the allergenic properties of peanuts.

KEYWORDS: Peanuts; allergenicity; IgE binding; AGE adducts; Maillard reaction; ELISA; blot assays; stress proteins; antibodies to dehydrin; maturity; curing; roasting; food processing

INTRODUCTION

It is known that in order to achieve the highest grades of peanuts with desirable flavor, the processes of maturation, curing (a drying process), and roasting must be carefully monitored (*1*, *2*). Previous studies show that roasting increases the allergenic property of peanuts (*3*−*7*). However, it is not known whether peanut maturity and curing have an effect on allergenicity. During maturation and curing, metabolites such as carbohydrates (*8*) and proteins (*9*−*11*) increase significantly. These metabolites react in a Maillard reaction during roasting to form flavor compounds including advanced glycation end products (AGEs) (*12*, *13*). All of these are important because immature peanuts or those cured improperly at excessively high temperatures (>35 °C) tend to develop fruity fermented off-flavors after roasting (*1*). As AGEs are associated with allergenicity (*5*−*7*), it is postulated that peanut maturity and curing may affect allergenicity in addition to flavors.

Another reason for the above postulation was based on the study of roasting. During roasting, AGE adducts (i.e., protein-bound AGEs) are known to increase (*5*−*7*). This increase in AGE adducts is probably more pronounced in mature peanuts than in the immature peanuts because, as indicated above, metabolites such as sugars and proteins, especially the major peanut allergen Ara h1 (*14*), increase during maturation and, consequently, after roasting, may help increase the level of AGE adducts in mature peanuts. If so, allergenicity may increase (*5*−*7*). It is, therefore, postulated that peanut maturity may have an effect on allergenicity. Similarly, curing is thought to be associated with allergenicity because curing is a drying process and, depending on the conditions (e.g., at undesirable temperatures), may produce different levels of AGE adducts and thereby different degrees of allergenicity. In this study, we focused on the curing temperatures and their effects on AGE adducts and allergenicity.

In addition, during peanut maturation and curing, a new class of proteins, namely, stress proteins or dehydrin-like proteins, are produced (*10*). Dehydrin is a stress protein with highly conserved lysine-rich sequences and is commonly found in plants under stress conditions such as heat, dehydration, or lack of oxygen (*15*). Dehydrin-like or stress proteins are formed because peanut maturation and curing are a condition of stress caused mainly by physiological dehydration or water loss and lack of oxygen (*11*). These proteins are thought to confer stress tolerance, maintain homeostasis, and protect cells from anaerobic

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or stress damage. In mammalian immune systems, stress proteins may serve as carriers of peptides or antigens to be transferred to the T-cells for immune responses and therapies (*16*, *17*). In plants, a major allergen has been identified as a stress-related protein (*18*). Peanut stress proteins (<28 kDa) may not have allergenic properties (preliminary studies). However, they are thought to be related to peanut allergenicity because they are liable to form AGE adducts due to their dehydrin-like property (i.e., lysine-rich sequences (*15*), which react with sugars) and thus potentially may enhance the allergenic property of peanuts. In addition, peanut stress proteins are involved in the processes of peanut maturation and curing (*10*), which, as indicated above, are postulated to have an effect on allergenicity.

In this study, levels of AGE adducts and IgE binding (a measure of allergenicity) were examined in mature and immature peanuts and peanuts cured at different temperatures. In addition, peanuts with and without stress proteins were compared in terms of IgE binding. The objectives were to determine the effect of peanut maturity and curing in conjunction with roasting on AGE adducts and IgE binding and if stress proteins are related to IgE binding.

MATERIALS AND METHODS

Apparatus. A CERES 900C microtiter plate reader was purchased from Bio-Tek Instruments, Inc. (Winooski, VT).

Reagents and Materials. Ethylene glycol-bis(β-aminoethyl ether)-*N*,*N*,*N*′,*N*′-tetraacetic acid (EGTA), bovine serum albumin (BSA), microtiter plate (Corning), anti-rabbit IgG alkaline phosphatase conjugate, ρ-nitrophenyl phosphate, rabbit anti-human IgE−peroxidase conjugate, *o*-phenylenediamine, Tween 20, and phosphate-buffered saline (PBS) were purchased from Sigma Co. (St. Louis, MO). Immobilon-P membrane was obtained from Millipore Corp. (Bedford, MA). The immunblot assay kit (NBT/BCIP substrate) was purchased from BioRad Laboratories, Inc. (Hercules, CA). Rabbit antiserum to dehydrin, a plant stress protein, was purchased from Stressgen Biotechnologies (Victoria, BC). Human sera from three patients with peanut allergy were obtained from the University of Arkansas, Children's Hospital (Little Rock, AR), and a pooled serum was used. A superblock blocking buffer and bicinchoninic acid (BCA)−protein assay kit were purchased from Pierce Chemical Co. (Rockford, IL). Raw and roasted peanut seeds (Florunner, NC 9, Georgia Green, and SunOleic) were obtained from the National Peanut Research Laboratory, Dawson, GA, and USDA-ARS, North Carolina State University, Raleigh, NC.

Determination of Peanut Maturity. Maturity was determined by commercial kernel size category (e.g., no. one and jumbo (i.e., immature and mature)) or the visual hull-scrape color method (*19*) in which peanuts were sorted by pod color such as yellow and black (i.e., immature and mature). Although kernel size is not an objective measure of maturity, research has shown that the percentage of mature kernels in a sample generally increases with increasing size (*20*).

Curing. Peanuts were windrow-dried for at least 3 days, followed by drying with forced air heated to temperatures ranging from 35 to 77 °C.

Production of Antibodies to AGEs. Antibodies against AGEs were produced in rabbits as previously described (*6*), using a reaction mixture of BSA and glucose (after incubation for 90 days at 37 °C) as an immunogen.

Preparation of Peanut Protein Extracts. Extracts were prepared as previously described from defatted meals of raw and roasted peanuts (*6*). Briefly, meals (40 mg) were stirred in 0.3 mL of 0.02 M sodium phosphate, pH 8.5, 10 mM EGTA for 20 min at 4 °C, followed by centrifugation at 8500*g* for 10 min. The resultant supernatants (extracts) were used for enzyme-linked immunosorbent assays (ELISA). The concentration of proteins in the extract was determined using the BCA assay.

Assay of AGE Adducts in Raw and Roasted Peanuts. An indirect ELISA ($n = 3$) was performed according to the previous method (*6*). Briefly, a diluted (1:100) rabbit antiserum against AGEs (100 μL per well) was added to a microtiter plate coated with a peanut protein extract (30 μg/mL). After it was incubated for 30 min at 25 °C and washed, an anti-rabbit IgG alkaline phosphatase conjugate (1:8000) (100 μL) was added, incubated for 30 min at 25 °C, and washed. A substrate solution (ρ-nitrophenyl phosphate in 10% diethanolamine, pH 9.8, 0.5 mM $MgCl_2$;1 mg/mL) (100 μL) was then added and incubated for 30 min at 37 °C. Absorbance was read at 405 nm, using the Ceres 900C BioTek microplate reader.

Assay of Stress Proteins. Peanut stress proteins were identified in blot assays as previously described (*10*), using antibodies against dehydrin, a plant stress protein. Briefly, proteins (15 μg) from a peanut extract were applied to sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS−PAGE) and transferred to an Immobilon-P membrane. Stress proteins on the membrane were then detected with a rabbit antiserum against dehydrin (1:500), followed by addition of an anti-rabbit IgG alkaline phosphatase conjugate (1:10 000) and a substrate from BioRad Immune-blot assay kit. For determination of stress proteins in ELISA ($n = 3$), the method was similar to the one described above for AGEs except that antibodies against dehydrin (1:500) instead of AGEs were used.

Assay of IgE Binding to Peanuts. An indirect or a competitive inhibition ELISA was carried out ($n = 3$) as previously described (*6*, *7*). In the indirect ELISA, a diluted pooled serum (containing IgE antibodies) (100 μL, 1:20) from peanut allergic individuals was added to a plate coated with a peanut protein extract. In the competitive inhibition ELISA, a diluted pooled serum (IgE, 1:20) (50 μL) was incubated in a protein-coated plate with a peanut protein extract (served as an inhibitor) (50 μL) at a protein concentration ranging from 0.1 to 1000 μg/mL. In both ELISA, after incubation for 45 min at 25 °C and wash, a rabbit anti-human IgE peroxidase conjugate (1:500) (100 μL) was added, incubated for 30 min at 25 °C, and washed. A substrate solution (100 μL) of *o*-phenylenediamine (0.5 mg/mL), 0.03% hydrogen peroxide in 0.1 M citrate buffer, pH 5.5, was then added. After it was incubated for 30 min at 37 °C, the reaction was stopped with 50 μL of 4 N sulfuric acid. The absorbance was read at 490 nm. In the competitive ELISA, the absorbance value of a sample containing IgE antibodies and a peanut extract (inhibitor) was represented by B, while B_o represented the absorbance value of a control containing IgE only.

Statistical Analyses. Values are means of three determinations and are represented as mean ± SD. All statistical analyses were performed using Student's *t*-test at 95% confidence.

RESULTS AND DISCUSSIONS

Maturity vs AGEs and IgE Binding. We hypothesized that levels of AGE adducts are different between mature and immature peanuts because they (peanuts) have different amounts of proteins and sugars. These protein−sugar components react in a Maillard reaction to form AGE adducts during roasting. In this case, mature peanuts may have a higher level of AGE adducts than immature peanuts because of a higher content of proteins and sugars in the former (*8*−*11*). Using polyclonal antibodies against AGEs, adducts of AGEs can be detected (*6*, *7*). Data (**Figure 1a**) show that after curing and roasting, mature peanuts (classified as black or jumbo) exhibited approximately 20% higher levels of AGE adducts than immature peanuts (classified as yellow or one). No difference in AGE level was found between mature and immature raw peanuts (data not shown). This indicates that only when roasted, mature and immature peanuts have different levels of AGE adducts.

As AGE adducts are related to allergenicity (*5*−*7*), the above mature and immature roasted peanuts were further analyzed for IgE binding. Data (**Figure 1b**) show that mature roasted peanuts exhibited a higher IgE binding (lower curve) than immature roasted peanuts. No difference in IgE binding was found between mature and immature raw peanuts (data not shown). This demonstrates that the maturity state of peanuts, when roasted, can have an effect on IgE binding. Like previous studies

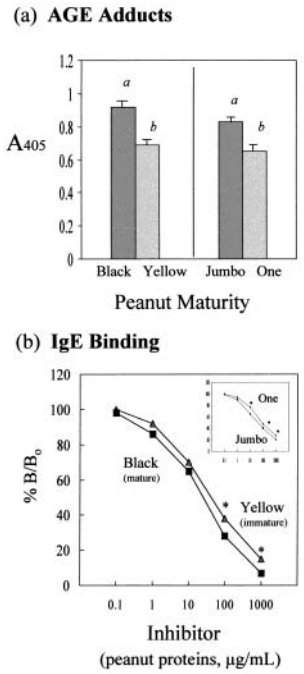

Figure 1. Effect of peanut maturity on (**a**) AGE adducts and (**b**) IgE binding (including inset figures). Yellow or one = immature; black or jumbo = mature. (**a**) AGE adducts from mature and immature roasted peanuts (*Florunner*) were determined in an indirect ELISA. Briefly, a rabbit antiserum against AGE (BSA–glucose reaction product) (1:100) was added to a plate coated with a peanut protein extract, followed by detection using an anti-rabbit IgG alkaline phosphatase conjugate (1:8000) and a substrate solution of *p*-nitrophenyl phosphate. Values are mean ± SD, $n = 3$. A value with a different letter is significantly different at 95% confidence. (**b**) IgE binding was determined in a competitive ELISA. Briefly, a pooled serum (IgE antibodies; $1{:}40_{final}$) from peanut allergic individuals was incubated in a protein-coated plate with a peanut protein extract (as inhibitor) at the concentration indicated. Detection was carried out using a rabbit anti-human IgE–peroxidase conjugate (1:500) and a substrate solution of *o*-phenylenediamine. Values are means of three determinations. The asterisk (*) denotes a marked difference between yellow and black or between jumbo and one.

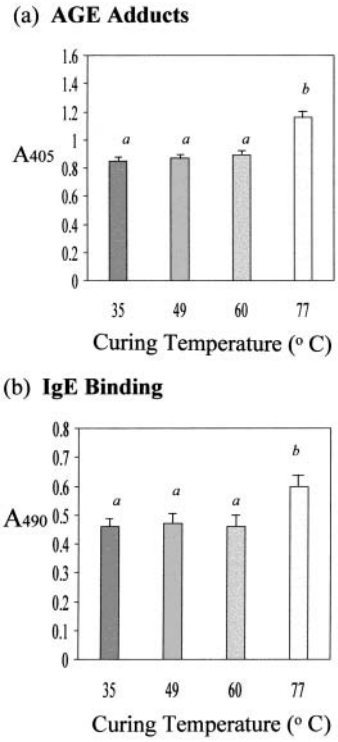

Figure 2. Effect of curing temperatures on (**a**) AGE adducts and (**b**) IgE binding. Peanuts (Georgia Green) were cured at the temperatures indicated and then roasted prior to assays. (**a**) AGE adducts were determined as described in **Figure 1**, using plates coated with protein extracts from peanuts cured at the temperatures indicated. (**b**) IgE binding was determined in an indirect ELISA. Briefly, a pooled serum (IgE antibodies; $1{:}40_{final}$) from peanut allergic individuals was added to a plate coated with a protein extract from peanuts cured at the temperature indicated. Detection was carried out using a rabbit anti-human IgE–peroxidase conjugate (1:500) and a substrate solution of *o*-phenylenediamine. Values are mean ± SD, $n = 3$. Values with the same letters are not significantly different at 95% confidence.

(*5*−*7*), this study shows a link between AGE adducts and allergenicity. For example, when AGE adducts increase (**Figure 1a**; mature roasted peanuts), peanut allergenicity increases as well (**Figure 1b**; mature roasted peanuts).

Curing Temperatures vs AGEs and IgE Binding. Curing is a drying process usually involving 3−7 days of windrow drying, followed by drying with forced heated air for 20−24 h until peanut kernels have reached a moisture content safe for storage. The curing process reduces the kernel moisture content from 40% to approximately 8−10% wet basis, and also, AGE adducts are detected. Previously, several drying air temperatures (35, 49, 60, and 77 °C) were tested to determine their effect on drying rate, milling quality, and seed germination (*21*). However, it is not known whether these temperatures have an effect on AGE adducts and/or IgE binding (i.e., allergenicity). To determine the effects of curing temperatures, peanuts were cured at 35−77 °C, roasted, and tested for the presence of AGE adducts and IgE binding. Results show that peanuts of different curing temperatures, except 77 °C, were not different in levels of AGE adducts (**Figure 2a**) or IgE binding (**Figure 2b**). However, at 77 °C, a higher level of AGEs and IgE binding was observed. It was concluded that curing temperatures between 35 and 60 °C do not give a difference in profiles of AGEs and IgE binding. However, curing at a higher temperature such as 77 °C gives a profile of higher IgE binding and AGEs level. This suggests that curing at higher temperatures can have an adverse effect on the allergenic property of peanuts. It should be noted that curing is not just a drying process but also a stress condition associated with metabolic changes (*10*, *11*). This condition produces stress proteins (see below), which may also link curing to allergenicity. Under normal conditions, peanuts are cured with air heated no greater than 35 °C, and therefore, should not affect levels of AGEs or IgE binding.

Stress Proteins vs IgE Binding. We previously reported that stress proteins are induced during peanut maturation or curing (*10*, *11*). To determine the relationship between stress proteins and allergenicity, immature peanuts that were cured and not cured were used. The difference between cured and noncured is in the induction or expression of stress proteins. This induction occurs in immature peanuts during maturation or curing; once mature or cured, these peanuts exhibit stress proteins. As shown in **Figure 3a**, two major stress proteins (between 20 and 28 kDa) were detected primarily in peanuts that had been cured. By contrast, stress proteins were absent in noncured peanuts (i.e., before curing). When analyzed for IgE binding, peanuts with stress proteins (i.e., after curing) gave a higher IgE binding than peanuts without stress proteins (i.e., before curing) (**Figure 3b**). The reason for this probably is because peanuts with stress proteins may contain proteins with lysine-rich sequences due

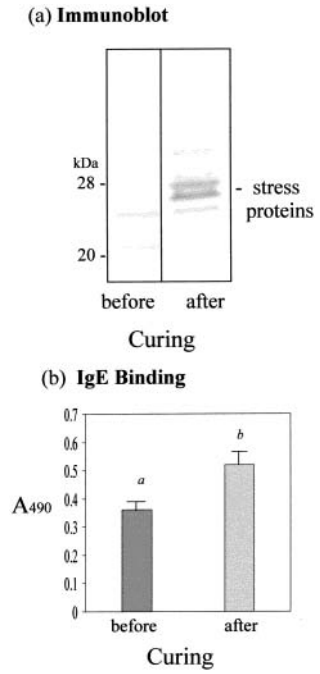

Figure 3. Relationship of stress proteins to IgE binding. (**a**) Immunoblot assay of stress proteins and (**b**) IgE binding of proteins from immature raw peanuts (Florunner) (cured or not cured). (**a**) Blots were carried out by subjecting peanut protein extracts to SDS–PAGE and transferring proteins to an Immobilon P-membrane. Stress proteins were then detected using a rabbit antiserum against dehydrin (1:500), an anti-rabbit IgG alkaline phosphatase conjugate (1:10 000), and a NBT/BCIP substrate kit from BioRad. (**b**) IgE binding was determined in an indirect ELISA as described in **Figure 2**. Values are mean ± SD, $n = 3$. A value with a different letter is significantly different at 95% confidence.

to their dehydrin-like property (*15*), and these lysine residues may react with the sugars to form AGE adducts and, consequently, increase IgE binding due to increased AGE adducts (*5*−*7*).

Further evidence for the association of stress proteins with allergenicity is based on the study of raw and roasted peanuts. Previously, we reported that roasted peanuts have a higher IgE binding than raw peanuts (*3*−*7*). To determine if raw and roasted peanuts are also different in levels of stress proteins, several peanut varieties that have previously been tested for IgE binding (e.g., Florunner, NC 9, Georgia Green, and SunOleic) were analyzed for stress proteins. Data (**Figure 4**) show that while levels of stress proteins were approximately the same among all peanut varieties (roasted or raw), roasted peanuts exhibited a much higher level of stress proteins than raw peanuts. This increase in stress proteins probably would lead to an increase of AGEs due to the lysine-rich sequences in these proteins as described above. The consequent increase of AGEs may in turn lead to an increase in IgE binding. Overall, it appears that AGE adducts and stress proteins may be potential markers for changes in IgE binding. Alcohol dehydrogenase, an enzyme induced during peanut maturation and curing (*9*, *11*), has also been identified as a potential marker for changes in peanut allergenicity because under certain conditions, it increases or decreases with IgE binding (*22*).

Conclusions. The goal of this study was to determine if maturity and curing, in conjunction with roasting, affect peanut allergenicity or IgE binding. As the processes of maturation and curing are associated with stress or the induction of stress proteins, we also examined the relationship of stress proteins

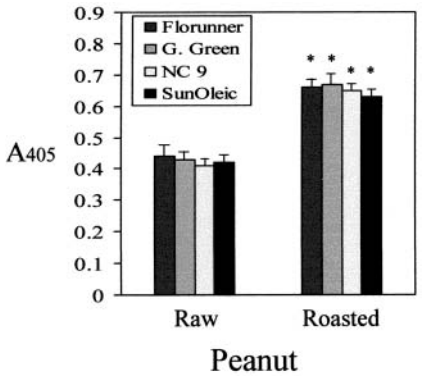

Figure 4. Effect of roasting on stress proteins. Stress proteins from raw and roasted peanuts of different varieties were determined in an indirect ELISA. Briefly, a rabbit antiserum against dehydrin (1:500) was added to a plate coated with a protein extract from the variety indicated. Detection was carried out using an anti-rabbit IgG alkaline phosphatase conjugate (1:10 000) and a substrate solution of *p*-nitrophenyl phosphate. Values are mean ± SD, $n = 3$. The asterisk (*) denotes a marked difference between roasted and raw.

to IgE binding. In addition, levels of AGE adducts, which may occur in the above processes and may be linked to IgE binding, were determined. Results showed that mature peanuts (classified as black or jumbo), when cured and roasted, exhibited higher IgE binding and increased level of AGE adducts than did immature peanuts (classified as yellow or one). Therefore, peanut maturity may be associated with IgE binding. Curing using drying air temperatures between 35 and 60 °C did not give a difference in the profile of IgE binding and AGE levels. However, curing at a higher temperature (e.g., 77 °C) appeared to result in higher IgE binding and AGE levels. This suggests that curing, if handled improperly (e.g., at high and undesirable temperatures), may affect the allergenic properties of peanuts. Curing using recommended temperatures (<35 °C) has no detrimental effect on peanut allergenicity.

The relationship between allergenicity and stress proteins was also investigated. Peanuts with stress proteins exhibited a higher IgE binding than those without stress proteins. This increase in IgE binding probably was due to the AGE adducts (*5*−*7*) formed in the former, which contains lysine-rich sequences liable to reaction with the sugars. In addition, stress proteins increased during roasting. This increase coincided with the increase of AGE adducts and IgE binding (*5*−*7*). It was, therefore, thought that stress proteins may be related to allergenicity.

Overall, we concluded that the processes of maturation, curing, and roasting may be associated with peanut allergenicity. Of the three processes discussed, only curing and roasting can be controlled. The degree of maturation is somewhat controlled by the growers' choice of when to harvest. To ensure that allergenicity does not change significantly, proper monitoring of these processes is necessary. End products such as AGE adducts and stress proteins may be used as markers for predicting changes in IgE binding. However, further research is needed to prove their usefulness.

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