OCCURRENCE OF CARCINOGENIC HETEROCYCLIC AROMATIC AMINES IN FRIED CHICKEN BREASTS

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Abstract – The presence of heterocyclic aromatic amines (HAA) in fried chicken breasts was investigated. All samples contained HAAs including MeIQx, PhIP, and the β-carbolines Harman and Norharman. In particular, PhIP was found in high concentrations of 1.5 - 9.1 ng/g. MeIQx was detected at very low concentrations (n.d. - 1.1 ng/g). The concentrations of the co-mutagenic β-carbolines Harman and Norharman ranged from 0.8 to 2.3 ng/g. The content of the precursor glucose varied substantially from 30-250 mg/100 g based on dry matter. In contrast, the content of the precursor creatine in non-fried chicken breasts varied by only 8 %. A significant linear correlation existed between PhIP and the molar ratio of creatine/glucose (r=0.88, p<0.001). We thus conclude that with increased concentration of glucose in chicken breast, the formation of PhIP may be reduced.

Key Words – Heterocyclic Amines, precursor glucose, creatine, chicken, carcinogens.

I. INTRODUCTION

The variation in human diets and dietary content of food carcinogens may be an explanation for the observed deviation in cancer rates across the globe. Heterocyclic Aromatic Amines (HAA) are found in the crust of fried, broiled or cooked meat and fish. These compounds are likely formed from creatine/creatinine, amino acids, and carbohydrates in a series of Maillard reactions. Some HAA are known to be mutagenic in the Ames test and carcinogenic in long-term animal studies on rodents and non-human primates [1]. The International Agency on Cancer Research has classified several HAA as possible or probable human carcinogens and has suggested to reduce exposure to them [6]. In addition to the physical parameters such as heating time and temperature, and preparation methods [3], the quality of the raw material has an important impact on the formation of HAA. The objective of this study was to examine the occurrence of HAA in fried chicken breasts, a popular product of the food and food service industries. The generated data may be of use for the proper assessment of maximum human intake levels.

II. MATERIALS AND METHODS

Preparation of patties: Chicken breasts from a variety of different animals were cut into equally sized cubes (10 cm long x 14 cm wide x 1.3 cm thick). A double contact grill (Model Nevada, Neumärker, Hemer, Germany) was heated to a surface temperature of 230 °C. Chicken breasts were coated lightly with refined sunflower oil. Each chicken breast was covered with two pieces of tin foil and then fried for 2:30 min to a core temperature of 72 °C and to a surface temperature < 190 °C at the end of the frying process. Two breasts of each animal were fried and homogenized later to obtain a single sample.

Determination of HAA: The method includes the determination of polar and apolar HAA. The method of HPLC analysis with some modifications was based on the method described by Gross and Grüter [5]. The peaks of HAA, as well as Norharman and Harman, in samples were identified by comparing the retention times and UV-spectra with standards. The quantification was carried out with the method of standard addition.

Determination of creatine and glucose (residual glycogen): Creatine and glucose were determined enzymatically using a test kit following the instructions of the manufacturer (Roche diagnostics GmbH, Mannheim, Germany).

Determination of principal components: The main components of chicken breasts were determined according to the instructions of the official collection of methods of analysis [2]

III. RESULTS AND DISCUSSION

Results of our analysis showed that the raw material of 21 chicken breasts from various animals generally consisted of similar principal compounds with little variation in their concentrations including proteins, fats, minerals (ashes), and moisture (Table 1). Only the collagen content was noticeable different with a variation coefficient of 9%. Large differences were found in glucose content of the different chicken breasts, as signified by the large variation coefficient of 45%, and chicken breasts in general had very low glucose concentrations (Table 1). The total creatine content in raw meat hardly deviated between animals (CV=8%).

All fried breasts contained HAA including MeIQx, PhIP, Harman and Norharman. The mean concentrations of these compounds ranged from not detected to 9.1 ng/g (Fig. 1 and 2). Fried breasts of chicken had particularly high PhIP concentrations (Fig. 1), a result that is in line with that of other studies [4].

The formation is influenced by the precursor concentration and the molar ratio between creatine and glucose. A maximal effect on the formation of HAA in model systems was reported [7, 8], if the molar ratio of total creatine and glucose was close to 0.5. If the molar content of glucose increased, the formation of HAA subsequently decreased. However in our case, the concentration of creatine in samples was always higher than the glucose concentration, e.g. the molecular ratio of creatine/glucose in our 21 samples ranged between 9.6 and 64. A significant linear correlation only existed between PhIP and the molar ratio of creatine/glucose ($r=0.88$, $p<0.001$) (Fig. 3), and there increased glucose contents generally reduced the concentrations of PhIP. In contrast, MeIQx and β-carbolines did not significantly correlate to the ratio of creatine/glucose.

IV. CONCLUSION

The natural variation of HAA precursors in chicken breasts may explain the wide range of HAA concentrations in fried samples even if preparation procedures (time, temperature) were kept constant. The residual glycogen content in particular showed large variations. Therefore, a predictive assessment of human intake of HAA after consumption of a specific amount of fried chicken is difficult due to these variations. Risk assessments may instead have to focus on worst case assessments were contents are such that generation of HAA is maximum.

Table 1 Chemical composition of the raw material of chicken breasts

Analysis	Mean ± S.D. [g/100 g]	CV [%]
Protein	24.0 ± 0.25	1
Moisture	74.2 ± 0.58	0.8
Fat	1.57 ± 0.12	7.6
Minerals	1.18 ± 0.04	3.3
Collagen	0.51 ± 0.05	9.8
Glucose[1]	0.14 ± 0.06	45.2
Total Creatine[1]	1.74 ± 0.14	8

[1]based on dry matter

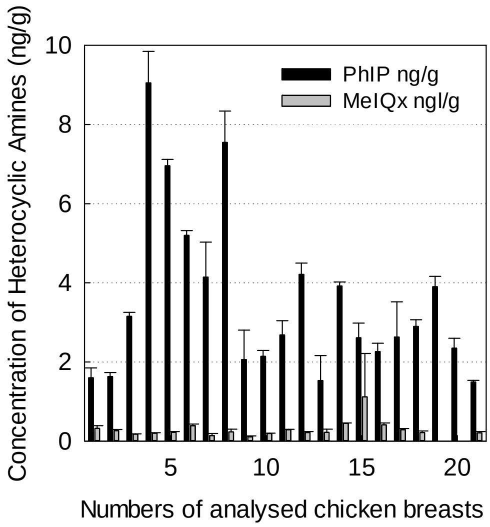

Figure 1. Concentrations of PhIP and MeIQx in fried chicken breasts

ABBREVIATIONS

HAA: Heterocyclic Aromatic Amines, MeIQx: 2-amino-3,8-dimethylimidazo[4,5-*f*]quinoxaline, 4,8-DiMeIQx: 2-amino-3,4,8-trimethylimidazo [4,5-*f*]quinoxaline, PhIP: 2-amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine, Norharman: 9*H*-pyrido[3,4-*b*]indole, Harman: 1-methyl-9*H*-pyrido [3,4-*b*]indole, SD: standard deviation, CV: Coefficient of variation

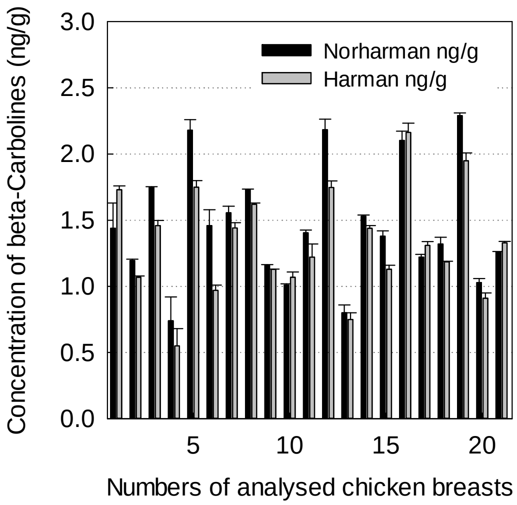

Figure 2. Concentrations of Harman and Norharman in fried chicken breasts

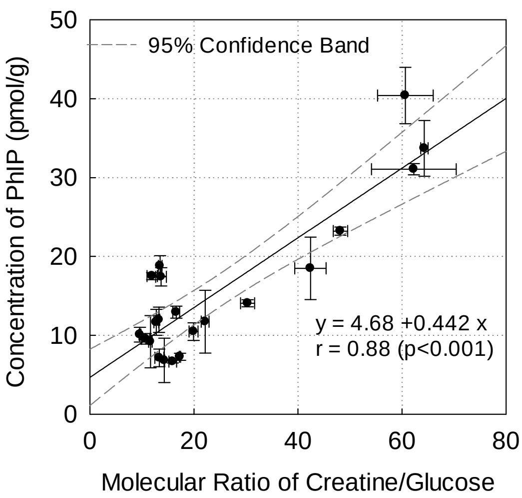

Figure 3. PhIP concentration in fried chicken breasts versus molar ratio of creatine and glucose (based on dry matter)

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REFERENCES

1. Adamson, R. H., Thorgeirsson, U. P., Synderwine, E. G., Thorgeirsson, S. S., Reeves, J., Dalgard, D. W., Takayama, S. & Sugimura, T. (1990). Carcinogenicity of 2-amino-3-methylimidazo[4,5-*f*]quinoline in non-human primates: induction of tumors in three macaques. Japanese Journal of Cancer Research 81, 10-14.
2. ANONYM (2005). Amtliche Sammlung von Untersuchungsverfahren nach § 64 LFGB, Beuth Verlag GmbH, Berlin Köln.
3. Arvidsson, P., Boekel M. A .J. S. van, Skog, K. & Jägerstad, M. (1997). Kinetics of formation of polar heterocyclic amines in a meat model system. Journal of Food Science 62, 911-916.
4. Busquets, R., Puignou, L., Galceran, M. T. & Skog, K. (2006). Effect of red wine marinades on the formation of Heterocyclic Amines in fried chicken breast. Journal of Agricultural and Food Chemistry 54, 8376-8384.
5. Gross, G.A. & Grüter, A. (1992). Quantitation of mutagenic/carcinogenic heterocyclic aromatic amines in food products. Journal of Chromatography 592, 271-278.
6. IARC (1993). IARC Monographs on the evaluation of carcinogenic risks to humans. No.56. Pp. 165-242. International Agency for Research on Cancer
7. Skog, K. & Jägerstad, M. (1990). Effect of monosaccharides and disaccharides in model systems. Mutation Research 230, 263-272.
8. Skog, K., Laser Reuterward, A. & Jägerstad, M. (1992). Inhibitory effect of carbohydrates on the formation of mutagens in fried beef patties Food and Chemical Toxicology 30(4), 289-297.