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요중 AAMA에 의한 한국 성인 아크릴아마이드(AA)의 하루섭취량 추정 및 기여 식습관에 대한 분석

Estimation of the Daily Human Intake of Acrylamide (AA) Based on Urinary N-acetyl-S-(2-carbamoylethyl)-cysteine (AAMA) and the Contribution of Dietary Habits in South Korean Adults

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ABSTRACT

Objectives: This study estimated the adult Korean daily intake of acrylamide (AA) and investigated its relationship with demographic, lifestyle and dietary habits by using urinary concentrations of N-acetyl-S-(2-carbamoylethyl)-cysteine (AAMA).

Methods: Human data (n=1870) was collected in a nationwide cross-sectional biomonitoring program representing the population (18–69 years) residing in South Korea. Urinary AAMA was analyzed with a LC-MS/MS system. Daily intakes of AA were estimated using mass daily AAMA, which was calculated through urinary AAMA concentration and daily creatinine excretion. Statistical analysis was performed with SAS procedures for calculating geometric means, confidence intervals and the exponentiated beta coefficient of multiple linear regressions.

Results: Daily intake of AA was estimated at 0.475 μg/kg body weight (BW) per day (95% confidence interval (CI): 0.447-0.503). In the case of current smokers, AA intake was 0.957 μg/kg BW per day (95% CI: 0.847-1.067), which was significantly higher than that of former smokers and never smoked ($p<0.0001$). The strong affecting factors were age (95% CI: 0.68-1.14; $p=0.0180$), education level (95% CI: 1.05-1.42; $p=0.0163$), body mass index (BMI) (95% CI: 1.00-1.82; $p<0.0001$), and smoking status (95% CI: 0.97-3.05; $p<0.0001$). Korean dietary habits increasing AA intake were coffee ($p=0.0005$), cup noodles ($p=0.0010$) and canned foods ($p=0.0005$). Meanwhile, foods decreasing AA intake were fresh fruit ($p=0.0076$), cooked beef ($p=0.0335$) and cooked pork ($p=0.0147$).

Conclusion: The Korean daily intake of AA in adults was estimated to be similar with those found in developed countries. The factors increasing daily AA intake were coffee, cup noodles and canned foods, and decreasing factors were fresh fruit, cooked beef and cooked pork.

Key words: Acrylamide (AA), daily intake, dietary habits, urinary N-acetyl-S-(2-carbamoylethyl)-cysteine (AAMA)

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I. Introduction

Human exposure to acrylamide (AA) is significantly dependent on the consumption of carbohydrate-rich foods that are prepared at temperatures above 120°C and low moisture.[1,2] AA can be found in various fried, deep-fried and oven-baked foods. It is a concern in foods which are regularly consumed through many years, such as chips (French fries), crisps and bread, as well as biscuits, crackers and breakfast cereals.[1] AA is neuro-toxic,[3] clastogenic ,[4] and mutagenic in somatic and germ cells in rodents[4,5] and is considered as a probable carcinogen in humans.[6] AA is readily and rapidly absorbed following oral ingestion and either reacts with glutathione to form N-acetyl-S-(2-carbamoylethyl)-cysteine (AAMA) or is metabolized via CYP2E1 to for glycidamide.[7] AAMA is the predominant metabolite excreted in urine. Fuhr et al.[8] noted that, overall, 60.3% of the human dose was recovered in the urine, and unchanged AA and AAMA accounted for urinary excretion of 4.4% and 50.0% of the dose, respectively. The apparent terminal elimination half-lives for unchanged AA and AAMA were 2.4 h and 17.4 h, respectively.[8] Good biomarker could identify exposure sources and highly exposed subpopulations. Urinary AAMA is used as an excellent AA biomarker of many national-scale biomonitoring programs for assessing AA exposure.

Dietary intake of AA is difficult to assess because exact information on food processing procedures, especially for home-made meals, is rarely available. A duplicate diet study of adults at Switzerland which was performed in 2002, reported a mean daily intake of AA was 0.277 μg/kg body weight (BW) per day.[9] However, the range of AA found in food was broad and the determinants of the variability unknown. The foods that have been analyzed to data represent only a portion of the total diet and do not include foods representative of those consumed in developing countries.[10] Nonetheless, because food contributes a significant proportion of the total exposure based on estimates of biomarker, it is likely that there are other important sources as well. WHO[10] estimated that the average intake of AA for the general population, based on available biomarker data, was a ranged of 0.3 to 0.8 μg/kg BW per day in developed country. Human biomonitoring data could be used for interpreting environmental and food exposure estimates using the biomonitoring equivalent (BE) value, which refer to the concentration or range of chemicals or its metabolites in a biological medium (blood, urine, or another medium) consistent with an existing health-based exposure guidance value.[11] BEs can be estimated by using available human or animal pharmacokinetic data, and then AAMA can be used as a BE dossier of AA.[11] The scientific basis and derivation of BE values for AA based on existing exposure guidance values of AAMA for acrylamide. Hay & Aylward[11] reported the daily mass of AAMA excreted in urine had a functional relationship with the daily intake of AA, and proposed a screening tool for evaluation of biomonitoring data in context of risk assessment for AA. Ji et al[12] also estimated daily AA intake based the urinary AAMA concentrations, and reported the estimated median (95th percentile) values of daily AA intake was 1.04 (2.47) μg/kg BW per day in South Korean children. Diet has been considered as one of the major contributors among the non-occupational persons.[1] Strong positive correlation was found between urinary AA and intake of starch and coffee among Norwegian adults.[13]

The aim of this study is to estimate the daily human intake of AA in South Korean adults, with using a specific biomarker, in this case urinary AAMA, and to investigate their relationships with demographic, lifestyle and dietary habits in a selection of the South Korean general population. The data of urinary AAMA have been supplied from Korean nationwide biomonitoring program which was reported in a previous paper.[14,15]

II. Materials and Methods

1. Study population

Human biological sampling and data collections in this study were conducted between July and October of 2009, in the form of a cross-sectional survey representing the adult population (18–69 years) residing in South Korea.[14,15] Participants were recruited from 100 census blocks selected by a stratified two-stage cluster random-sampling design, based on the National Census Registry. Approximately 2063 individuals completed the interviews and provided urine samples, and 1870 individual data (90.6%) was reasonable for this study. The Korean Food and Drug Administration supervised this study, while the Asan Medical Center Institutional Review Board approved the study protocol in accordance with the ethical principles for medical research involving human subjects, as defined by the Helsinki Declaration. Study participants provided written, informed consent.[14,15]

2. Data collection

Demographic data, including gender, age, education, income, smoking status, and current residence, were gathered through face-to-face interviews, which was included the survey of dietary habits with the self-developed questionnaires. Height and weight were measured while the subjects were wearing light clothing and no shoes. Spot urine samples were collected at different times throughout the day, because of the easy of collection of large population samples, and the persistent characteristics of AAMA with a urinary half–life of approximately 17.4 hr.[8,11] The variability of volume of spot urine samples were adjusted with urinary creatinine concentrations.[16] Urinary metabolites were analyzed with a LC-MS/MS system consisting of a HPLC system (Varian, Palo Alto, CA, USA) and triple-quadrupole tandem mass spectrometry coupled with electrospray ionization (Varian, Palo Alto, CA, USA). The LOD and LOQ for AAMA was 2.5 ng/ml and 10 ng/ml. Individuals whose urinary concentration fell below the LOD were assigned a value of LOD/2.[17] The process of analysis is explained in detail at the papers of Lee et al.[14,15]

3. Estimation of the daily intake of acrylamide

Daily intakes of AA based on urinary AAMA was estimated by using following equation:[11]

$$\text{AA daily dose} = \text{AAMA urine} * \left[\frac{MW_{AA}}{MW_{AAMA}}\right] * 2 \qquad (1)$$

where AA daily dose denotes the daily intake of acrylamide (mg); AAMA urine is the mass of AAMA excreted in urine per day (mg); and MWAAMA and MWAA are the molecular weights of AAMA and AA (234.1 and 71.1), respectively. The mass of daily AAMA was estimated with urinary concentration of AAMA adjusted for creatinine, and with daily creatinine excretion (DCE) which estimated with the following equation as a function of height, weight and age.[11,12,18]

$$\text{Male DCE} = 1.93(140 - A) * BW^{1.5} * h^{0.5} \qquad (2)$$

$$\text{Female DCE} = 1.64(140 - A) * BW^{1.5} * h^{0.5} \qquad (3)$$

Where DCE is daily creatinine excretion in μg, A is age in years, BW is bodyweight in kg, and h is height in cm

This equation (1) is based on a study in which the excretion of AAMA was measured in human volunteers receiving a single oral dose of AA at a dose of either 12.4 μg/kg or 13 μg/kg.[7,8,11] The most abundant metabolite, AAMA is specific to acrylamide exposure. Under steady-state exposure conditions consistent with chronic exposure at the reference dose (RfD) of AA, the daily elimination of AAMA on a molar basis should be equal to approximately 50% of the intake associated with the RfD of AA.[7,8,11]

4. Statistical analysis

Sample weights were post-stratified to the 2005

South Korea Census result of the population. Geometric means (GM) and confidence intervals (CI) were calculated based on the Taylor series linearization method. Multiple linear regressions of the log-urinary metabolites' concentrations on the predictor variables were fitted using the SAS procedures PROC SURVEYREG (SAS, version 9.3, Cary, NC, USA). The SURVEYREG procedure performs linear regression analyses for complex survey sample designs, including designs with stratification, clustering, and unequal weighting. Because of the skewed distributions, we calculated crude geometric mean by taking the antilog of the mean of the natural log-transformed values. We fit multivariate logistic regression of the predictor variables to find the influence of the dietary habits. The exponentiated beta coefficient as odds ratio represents the proportional changes in the arithmetic mean associated with each level of the explanatory variables, relative to a referent level, adjusting for the other variables in the model.[15,19]

III. Results

1. Estimated daily human intake of AA

The geometric mean of daily intakes of AA was estimated to be 0.475 μg/kg BW per day in the South Korean population 18-69 years of age (Table 1). When applied the simultaneous adjustment of covariates in a log-linear multiple regression, the significant different subgroups were gender (p= 0.0127), age (p=0.0180), education level (p=0.0163), body mass index (BMI) (p<0.0001), and smoking status (p<0.0001). Men and women who had never smoked showed the similar values of daily intakes, such as 0.380 and 0.352 μg/kg BW per day, respectively. But estimates of current smokers was 0.957 μg/kg BW per day, which was significantly higher than the value of former smokers (p<0.001) and who never smoked (p<0.0001). Smoking subgroups who showed higher estimated value were men, 60-69 years old, lower education (≤high school), lower household income, and lower BMI (<18.5) (Table 2).

2. Dietary habits affected the daily intake of AA

Korean dietary habits which significantly increased the daily intake of AA, were coffee (p=0.0005), cup-noodles (p=0.0010) and canned-foods (p=0.0005). Subgroups who drank more than four cups of coffee per day, and who ate canned-foods 4-6 times per week, showed the high estimated values, such as 0.743 and 0.782 μg/kg BW per day, respectively. In addition, Korean foods which significantly decreased the daily intake of AA, were fresh fruit (p=0.0076), cooked-beefs (p=0.0335) and cooked-pork (p=0.0147). Subgroups who ate fresh fruit 4-6 times per week, and who ate cooked-beefs and cooked-pork above 4 times per month, showed the low estimated values, such as 0.405, 0.420, and 0.340 μg/kg BW per day, respectively. When applying the simultaneous adjustment of covariates in a log-linear multiple regression, the strong affecting factor was fruit drinks which was boiled extracts in plastic bags (p=0.0046) (Table 3).

IV. Discussion

Biomonitoring Equivalents (BEs) are defined as the concentration of chemical or its metabolite in a biological medium (blood, urine or other medium) that is consistent which an existing health-based exposure guideline.[11] Urinary AAMA could be a good BE of daily intake of AA, because urinary AAMA is the predominant metabolite of AA,[7,8] and accounts for approximately 50% of an oral dose of AA with a urinary half-life of approximately 17 hr.[11]

Our estimated geometric mean of daily AA intake in Korean adults (18-69 years) was 0.475 μg/kg BW per day, which was less than 1.04 μg/kg BW per day of Korean children reported by Ji et al.[12] This comparison was similar with earlier studies[20,21] that the average dietary AA intake estimate for

Table 1. Estimated daily human intakes of acrylamide based on urinary AAMA concentrations in the South Korea

	N	GM (μg/kg BW/day) (95% CI)	Adjusted proportional change (95% CI) [a]
Total	1870	0.475 (0.447-0.503)	
Gender			
Men	803	0.615 (0.569-0.661)	0.81 (0.70-0.95)
Women	1067	0.366 (0.335-0.396)	1.00 (reference)
p [b]		<0.0001	0.0127
Age(years)			
18-29	247	0.464 (0.396-0.533)	0.80 (0.68-0.94)
30-39	412	0.493 (0.439-0.547)	0.94 (0.81-1.09)
40-49	456	0.491 (0.446-0.537)	0.99 (0.88-1.12)
50-59	433	0.454 (0.403-0.504)	1.00 (0.87-1.14)
60-69	322	0.454 (0.399-0.509)	1.00 (reference)
p [c]		0.6125	0.0180
Education			
<high school	532	0.463 (0.413-0.512)	1.22 (1.05-1.42)
High school	707	0.511 (0.474-0.548)	1.14 (1.03-1.25)
>high school	631	0.453 (0.413-0.494)	1.00 (reference)
p [c]		0.102	0.0163
Household income (US$/month)			
<880	400	0.501 (0.452-0.550)	1.16 (0.97-1.39)
880-2649	896	0.490 (0.446-0.534)	1.09 (0.91-1.30)
2650-4410	423	0.446 (0.407-0.485)	1.10 (0.92-1.31)
>4410	151	0.430 (0.352-0.508)	1.00 (reference)
p [c]		0.1451	0.3149
BMI			
<18.5	60	0.564 (0.450-0.678)	1.54 (1.31-1.82)
18.5-22.9	811	0.470 (0.427-0.512)	1.22 (1.11-1.35)
23.0-24.9	451	0.460 (0.406-0.513)	1.10 (1.00-1.22)
≥25.0	548	0.483 (0.444-0.532)	1.00 (reference)
p [c]		0.3926	<0.0001
Smoking status			
Current	405	0.957 (0.847-1.067)	2.56 (2.15-3.05)
Former	229	0.422 (0.371-0.473)	1.15 (0.97-1.37)
Never	1236	0.358 (0.336-0.381)	1.00 (reference)
p [c]		<0.0001	<0.0001
Current residence			
Rural	450	0.458 (0.395-0.521)	1.04 (0.94-1.16)
Urban	1420	0.478 (0.445-0.512)	1.00 (reference)
p [b]		0.6117	0.4425

[a] The exponentiated ß-coefficient from a log-linear multiple regression that included all covariates in the table; [b] *p* determined by a survey t-test; [c] *p* determined by a linear trend test. GM, geometric mean; BW, body weight; CI, confidence interval; BMI, body mass index; AAMA, N-acetyl-S-(2-carbamoylethyl)-cysteine

Table 2. Estimated daily human intakes (μg/kg BW/day) of acrylamide by smoking status in the South Korea

	Never smoking		Former smoking		Current smoking	
	N	GM (95%CI)	N	GM (95% CI)	N	GM (95% CI)
All (N=1870)	1236	0.358 (0.336-0.381)	229	0.422 (0.371-0.473)	405	0.957 (0.847-1.067)
Gender						
Men	220	0.380 (0.332-0.428)	214	0.406 (0.358-0.454)	369	0.990 (0.886-1.094)
Women	1016	0.352 (0.325-0.379)	15	0.675 (0.339-1.011)	36	0.646 (0.264-1.028)
Age (years)						
18-29	145	0.349 (0.297-0.401)	23	0.460 (0.356-0.565)	79	0.840 (0.592-1.089)
30-39	283	0.358 (0.321-0.395)	37	0.431 (0.285-0.577)	92	0.986 (0.799-1.173)
40-49	315	0.361 (0.323-0.400)	43	0.386 (0.309-0.462)	98	1.043 (0.861-1.224)
50-59	289	0.367 (0.318-0.416)	73	0.422 (0.330-0.515)	71	0.974 (0.747-1.201)
60-69	204	0.363 (0.304-0.421)	53	0.432 (0.321-0.545)	65	1.018 (0.795-1.241)
Education						
<high school	388	0.380 (0.334-0.426)	57	0.426 (0.348-0.504)	87	1.089 (0.866-1.311)
High school	452	0.344 (0.310-0.379)	93	0.464 (0.382-0.546)	162	1.140 (1.000-1.281)
>high school	396	0.358 (0.328-0.389)	79	0.385 (0.319-0.451)	156	0.801 (0.616-0.986)
Household income(US$/month)						
<880	271	0.373 (0.331-0.414)	40	0.401 (0.279-0.523)	89	1.154 (0.949-1.359)
880-2649	577	0.357 (0.326-0.388)	116	0.490 (0.399-0.582)	203	0.965 (0.769-1.162)
2650-4410	291	0.340 (0.311-0.368)	55	0.380 (0.312-0.448)	77	0.991 (0.796-1.186)
>4410	97	0.394 (0.318-0.470)	18	0.240 (0.166-0.313)	36	0.628 (0.396-0.860)
BMI						
<18.5	47	0.466 (0.364-0.568)	3	0.533 (0.292-0.774)	10	1.514 (0.722-2.306)
18.5-22.9	568	0.372 (0.342-0.401)	88	0.422 (0.335-0.509)	155	0.918 (0.699-1.137)
23.0-24.9	294	0.342 (0.299-0.384)	55	0.442 (0.343-0.540)	102	0.910 (0.757-1.063)
≥25.0	327	0.328 (0.292-0.365)	83	0.405 (0.340-0.470)	138	1.011 (0.880-1.141)
Current residence						
Rural	299	0.360 (0.312-0.407)	62	0.404 (0.314-0.495)	89	1.055 (0.902-1.208)
Urban	937	0.358 (0.331-0.385)	167	0.426 (0.367-0.486)	316	0.942 (0.818-1.066)

GM, geometric mean; BW, body weight; CI, confidence interval; BMI, body mass index.

children and adolescent was about 2-fold higher than that of adults which was estimated to be between 0.05 and 0.5 μg/kg bw/day, because of causing the diets difference. Sirot et al.[20] reported that dietary AA exposure among the children was 1.6-fold higher than that of the adult (0.43 μg/kg BW/day) in French population.

Korean adults estimate was posited at the lower range value of the CONTAM panel report, in which the mean dietary AA exposures was estimated at 0.4 to 1.9 μg/kg BW per day[2] and was similar with values of several developed countries. Following a Joint FAO/WHO consultation,[10] the average intakes of AA for the European general population was estimated to be the range of 0.3-0.8 μg/kg BW per day. In Sweden, median dietary intakes of AA were estimated to be approximately 0.38 μg/kg BW per day for 1,211 individuals between 18 and 74 years old,[22] while they varied from 0.41 to 0.42 μg/kg BW per day for men and women of between 16

Table 3. Estimated daily intakes of acrylamide by dietary habits in the South Korea

Dietary habits	N	GM (μg/kg BW/day) (95% CI)	Adjusted proportional change (95% CI) [a]
Total	1870	0.475 (0.447-0.503)	
Fresh fruits			
<1/month	363	0.525 (0.455-0.594)	1.00 (0.88-1.12)
1-3/month	307	0.515 (0.462-0.568)	1.00 (0.88-1.14)
1-3/week	534	0.498 (0.448-0.548)	1.00 (0.90-1.11)
4-6/week	240	0.405 (0.347-0.463)	0.95 (0.86-1.06)
≥1/day	426	0.419 (0.378-0.459)	1.00 (reference)
p [b]		0.0076	0.8949
Coffee			
<1/month	346	0.442 (0.388-0.497)	0.79 (0.65-0.96)
1-3month	145	0.479 (0.407-0.550)	0.83 (0.69-0.99)
1-3/week	203	0.434 (0.383-0.485)	0.76 (0.62-0.92)
4-6/week	161	0.443 (0.369-0.517)	0.75 (0.60-0.94)
1-3/day	889	0.472 (0.432-0.511)	0.78 (0.65-0.92)
≥4/day	126	0.743 (0.608-0.878)	1.00 (reference)
p [b]	N	0.0005	0.131
Fruit drinks (boiled extracts in plastic bags)			
<1/month	1267	0.462 (0.433-0.491)	0.71 (0.59-0.85)
1-3/month	215	0.453 (0.389-0.517)	0.69 (0.56-0.86)
1-3/week	135	0.538 (0.437-0.639)	0.79 (0.64-0.98)
4-6/week	45	0.505 (0.325-0.685)	0.86 (0.63-1.16)
≥1/day	108	0.632 (0.481-0.784)	1.00 (reference)
p [b]		0.0902	0.0046
Cup noodles			
<1/month	1247	0.431 (0.399-0.463)	0.96 (0.83-1.11)
1-3/month	426	0.520 (0.461-0.580)	1.01 (0.91-1.13)
≥4/month	197	0.590 (0.502-0.678)	1.00 (reference)
p [b]		0.0010	0.6853
Canned foods			
<1/month	902	0.443 (0.412-0.474)	1.00 (0.76-1.32)
1-3/month	582	0.453 (0.409-0.498)	0.94 (0.72-1.24)
1-3/week	266	0.470 (0.408-0.532)	0.97 (0.72-1.31)
4-6/week	67	0.782 (0.595-0.969)	1.09 (0.82-1.45)
≥1/day	53	0.693 (0.465-0.920)	1.00 (reference)
p [b]		0.0005	0.399
Beef, cooked			
<1/month	982	0.498 (0.464-0.532)	1.09 (0.99-1.43)
1-3/month	546	0.461 (0.399-0.522)	1.23 (1.01-1.50)
≥4/month	342	0.420 (0.367-0.472)	1.00 (reference)
p [b]		0.0335	0.1086

Table 3. Estimated daily intakes of acrylamide by dietary habits in the South Korea

Dietary habits	N	GM (μg/kg BW/day) (95% CI)	Adjusted proportional change (95% CI) [a]
Pork, cooked			
<1/month	1434	0.481 (0.449-0.513)	1.19 (0.99-1.43)
1-3/month	334	0.487 (0.428-0.546)	1.23 (1.01-1.50)
≥4/month	102	0.340 (0.266-0.415)	1.00 (reference)
p [b]		0.0147	0.137
Seafood			
<1/month	1040	0.466 (0.429-0.503)	1.13 (0.97-1.30)
1-3/month	281	0.449 (0.376-0.522)	1.08 (0.91-1.29)
1-3/week	377	0.515 (0.459-0.572)	1.25 (1.07-1.47)
4-6/week	77	0.607 (0.473-0.742)	1.20 (0.98-1.47)
≥1/day	95	0.388 (0.289-0.487)	1.00 (reference)
p [b]		0.0742	0.0693

[a] The exponentiated ß-coefficient from a log-linear multiple regression that included all covariates in the table; [b] *p* determined by a linear trend test; GM, geometric mean; BW, body weight; CI, confidence interval.

and 79 years old in Norway.[23)]

The no-observed-adverse-effect level (NOAEL) of AA neurotoxicity for the most sensitive effect (microscopic nerve change) in rats was 0.5 mg/kg b.w. per day.[24)] Based on a life-long experiment, an oral reference dose (RfD) of 0.5 μg/kg BW per day calculated by dividing the NOAEL with margins of exposure (MOEs) of 1,000.[25)] The CONTAM panel performed benchmark dose (BMD) analyses on data for neurotoxicity and on the tumor incidence induced by AA in experimental animals, and selected BMDL10 values of 0.43 mg/kg BW per day for peripheral neuropathy in rats.[1)] So an oral reference dose (RfD) of AA calculated to 0.43 μg/kg BW per day with MOEs. And also this value is similar with the estimated Korean daily AA intake of 0.475 μg/kg BW per day.

Gender is looked like an important factor affecting daily intake levels of AA when simply comparing the estimated values of between men and women. Actually in our study, the estimate of South Korean men was significantly higher as 0.615 μg/kg BW per day than 0.366 μg/kg BW per day of women ($p<0.0001$). But the estimated values of men and women who had never smoking, were showed to be similar, such as 0.380 and 0.352 μg/kg BW per day, respectively (Table 2). For Norway, a similar trend value was showed between men and women, linked to food and coffee in a limited Norwegian exposure assessment study.[23)]

Smoking has been previously shown to be significantly correlated with a high intake of AA.[13,26,27)] In our study, daily AA intake of current smokers was the highest, as 0.957 μg/kg BW per day, and the value of former smokers was also significantly higher than non-smokers ($p<0.001$). Bjellaas, et al[13)] reported that the estimated dietary intake of smokers was high in Norwegians, which was explained due to variations in AA metabolism caused by differences in the levels and activities of CYP2E1.[28)] Human CYP2E1 could be inhibited by nicotine and cotinine,[29)] possibly resulting in the prolonged elevation of AA blood concentrations and a shift towards a greater proportion of AA excreted as AAMA.[11)] The rates of smoking persons in South Korea were reported to be 43.1% and 5.7% for men and women (≥19 years), respectively.[30)]

Foods importantly contribute to the estimated daily intake of AA levels among a variety of different countries. Although dietary patterns vary within and between countries, foods contributing consistently 80-90% of the total AA intake are potatoes, coffee, bread, biscuits/cakes and to a lesser extent breakfast cereals, crackers, rusks and other chips in Europe.[1] The CONTAM panel also reported that AA in certain foods could be formed during the preparation at temperature above 120°C and low moisture, especially in foods containing asparagine and reducing sugars.[2] Korean foods in our study, which contributed to increasing the daily AA intake, were cup-noodles, canned-foods and sea-food. This results will be causing the modern dietary transition in South Korea, with which after 1988, the advertising contents of food focused on eating-out, ready-cooked foods, such as fast foods, instant foods and franchise.[31] These fast foods in South Korea may contain much AA formed from ammonia and acrolein, which was occurred by heat decomposition of triglycerides present in frying oil, in the absence of asparagine.[32]

Coffee contains considerable levels of AA. The CONTAM panel reported AA was found at the highest level in coffee substitutes (dry) as average medium bound levels of 1499 μg/kg and in coffee (dry) as average medium of 522 μg/kg, although lower levels are expected in coffee beverages and coffee substitutes beverage as consumed by the population, due to dilution effects.[2] The high amounts of AA can form early in the roasting process (reaching peak levels of 2000ppb), and the brewing of coffee extracts AA from the matrix fairly efficiently.[33] Recently, coffee consumption levels in South Korea showed a very increasing trend. Korean was reported to drink 14.3 and 9.6 cups of coffee per week for man and women, respectively.[30] Their total import of coffee was reported to be 13,700 ton in 2012, which was very increased as much as five times compared to 2,650 ton in 2006.[34] In this study, the estimated daily intake of AA was shown to be higher, as much as 0.743 μg/kg BW per day among those who drink four more cups per day as compared to the groups who consume less coffee. Coffee was reported to contribute up to 30-40% of the total daily intake of AA, whereas potato crisps were responsible for almost 20% of the intake in adults in Norway and Sweden.[1,23] No practical solutions currently exist for reducing AA levels in coffee. Time and degree of roasting have been reported among the main factors affecting the levels of AA in coffee. So any change of the processing conditions (e.g., temperature, roasting duration) will considerably change the AA levels of final products.[35] But the exact relative contribution and their causality are not clear with our results, because of the limits of the cross-sectional study.

Many countries have been constructed a big data about some hazard materials such as AA in human and food, by performing the nationwide monitoring survey. Europe had collected the AA levels in food,[36] and built the comprehensive European consumption database for exposure assessment.[37]

USA and Canada also have accumulated a big data which was included many chemicals by federal monitoring program such as National Health and Nutrition Examination Survey (NHANES),[38] and human biomonitoring of environmental chemicals in Canada.[39] In case of Korea, National Institute of Environmental Research has also accumulated a big database with national monitoring results such as Korean National Environmental Health Survey (KoNEHS),[40] but not include many chemicals including AA. For exactly estimating the human exposure assessment, many kinds of hazard chemicals required to be included within Korean nationwide monitoring program.

V. Conclusion

Korean adult daily intake of AA was estimated to be 0.475 μg/kg BW per day (95% CI: 0.447-0.503) based on the nationwide biomonitoring data of AAMA. After simultaneously adjusting covariates in a log-linear multiple regression, the strong affecting factors of daily AA intakes were age (95% CI: 0.68-0.94; p=0.0180), education level (95% CI: 1.05-1.42; p=0.0163), BMI (95% CI: 1.00-1.82) and smoking status (95% CI: 0.97-3.05; p<0.0001). Korean dietary habits which significantly increased the daily intake of AA were coffee (p=0.0005), cup-noodles (p=0.0010) and canned-foods (p=0.0005). Meanwhile, Korean foods which significantly decreased the daily intake of AA were fresh fruit (p=0.0076), cooked-beef (p=0.0335) and cooked-pork (p=0.0147).

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