

AAMA AND GAMA LEVELS IN URINE AS MARKERS OF ACRYLAMIDE EXPOSURE FROM THE DIET AND TOBACCO SMOKE

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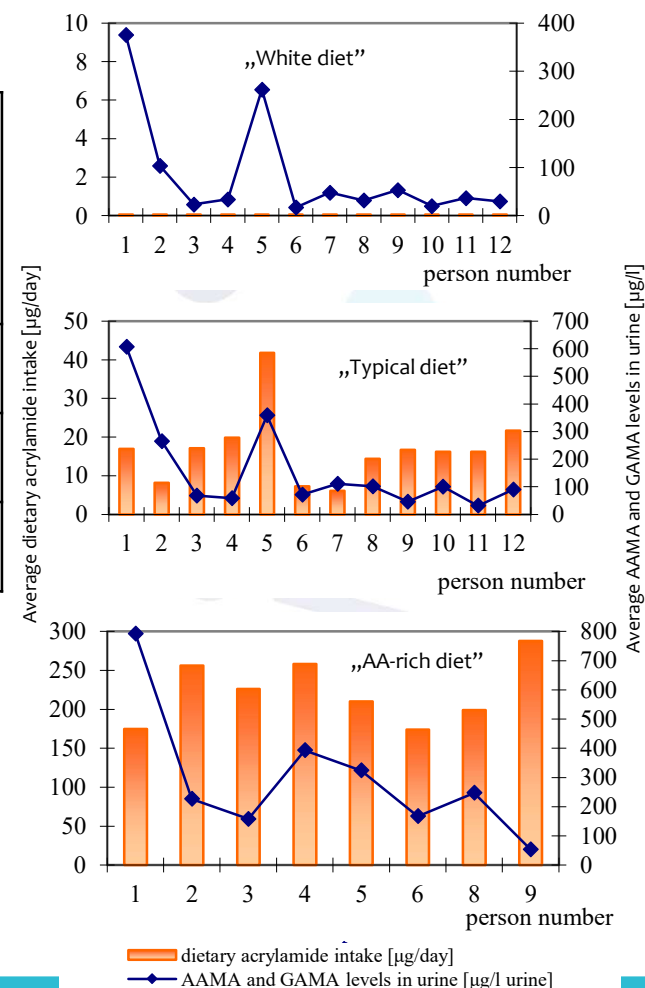
Average exposure to acrylamide estimated on the basis of AAMA and GAMA levels in urine of all volunteers, in relation to daily acrylamide intake and smoking habits.

Type of diets	AA intake [µg/day] (ME; range)	AAMA + GAMA [µg/g creatinine] (ME; range)	Acrylamide exposure estimated on the AAMA and GAMA levels in urine[µg/kg b.w./day] (Me; range)
TD	17,4 (0 ÷ 93)	68 (24 ÷ 406)	0,5 (0,1 ÷ 6,2)
WD	< LOQ (5 µg/kg)	44 (22 ÷ 167)	0,2 (0,1 ÷ 3,0)
AA-RD	218,2 (173,9 ÷ 287,9)	279 (198 ÷ 441)	1,5 (0,3 ÷ 5,3)

CONCLUSION

1. The median AAMA and GAMA levels in urine after the day of 'White diet' is significantly ($p < 0.05$) lower compared to AAMA and GAMA levels in urine after 'Typical diet' days and 'AA-rich diet' day.
2. We found significantly ($p < 0.05$) higher levels of acrylamide metabolites in the urine of smokers compared to non-smokers, regardless of the type of diet.
3. Our preliminary results confirm the possibility of using AAMA and GAMA determination in urine to assess exposure to acrylamide from the diet and tobacco smoke.

Acrylamide intake and excretion kinetics of AAMA and GAMA in urine depending on the type of diet and smoking habits.



INTRODUCTION Acrylamide (AA) is a 'probably carcinogenic to humans' monomer that can form in heated starchy food. Cigarette smoking appears to be the second most important source of exposure to AA. The main metabolite of AA which is responsible for its carcinogenic effect is glycidamide. Both of these compounds are conjugated with glutathione and further metabolised to form mercapturic acids: N-Acetyl-S-(2-carbamoyl-ethyl)-L-cysteine (AAMA) and N-Acetyl-S-(2-carbamoyl-2-hydroxyethyl)-L-cysteine (GAMA). **AIM** Assessment whether AAMA and GAMA levels in urine are suitable markers to estimate the exposure to acrylamide from food and tobacco smoke.

MATERIAL AND METHODS The study group consisted of 12 healthy volunteers (6 women and 6 men), three of whom smoked cigarettes (participants no. 1, 2 and 5). **Study design:** During the first 3 days of the study, participants were on their usual diet ('Typical diet', TD), followed by a 1-day diet without AA-sources foods ('White diet', WD) and smokers did not smoke. This was followed by another 3 days of their typical diet. On the last day of the experiment, participants consumed mainly food, which is a source of dietary AA ('AA-rich diet', AA-RD). Food consumption data from each day, over 24 hours was collected and participants provided the morning urine sample. AAMA and GAMA levels in urine was determined using validated LC-MS/MS method. The analysis was carried out using MRM technique (multiple reaction monitoring) in negative polarization. Ions of the tested compounds were monitored: m/z 233 $\rightarrow$ 104 (AAMA, CE: -22), m/z 236.9 $\rightarrow$ 108 (d4-AAMA, CE: -22), m/z 248.9 $\rightarrow$ 120.1 (GAMA, CE: -24) and m/z 252 $\rightarrow$ 119.9 (d3-GAMA, CE: -24) for the quantitative determination of the AAMA and GAMA content of urine, and m/z 233 $\rightarrow$ 58 (AAMA, CE: -54) and m/z 248.9 $\rightarrow$ 127.9 (GAMA, CE: -18) to verify the results obtained.