Exposure and Biomarkers
White Paper

I. Background

Until recently, the presence of acrylamide in food was not anticipated because it is not generally used in food processing and further it is a highly reactive molecule. As a result of studies conducted in Sweden (Tareke et al., 2002), a number of governments have analyzed foodstuffs for acrylamide. Although there has been no comprehensive evaluation of the US (or other) food supplies, acrylamide has been reported to be present in a variety of foods. The levels are generally highest in foods that have been subjected to dry heat processing such as frying, grilling or baking. Lower levels have been found in other heat-processed foods. Monitoring is on-going by many organizations and the results are being posted to their websites. These sites may contain additional information of interest to the workshop participants.

www.fao.org/es/esn/jecfa/acrylmidehttp://www.cfsan.fda.gov/~lrd/pestadd.html (http://www.slv.se/HeadMenu/livsmedelsverket.asp; http://www.bag.admin.ch/verbrau/aktuell/f/Acrylamidgehalt_liste_2_F.pdf; http://www.snt.no/nyt/tema/akrylamid/analyse_eng.html; and http://www.foodstandards.gov.uk/multimedia/pdfs/acrylamideback.pdf).

The bioavailability of acrylamide in food is uncertain. However, there is some evidence that the population at large has detectible levels of hemoglobin-acrylamide adducts, and Hb-adducts of the glycidamide epoxide metabolite, in blood (Tareke et al., 2002). There are many questions that need to be answered about this finding- how widespread are these adducts in the population, and how do they vary with age, ethnicity, diet, etc. These adducts may be useful as biomarkers of exposure. To date, there is no firm evidence as to the source of the acrylamide that has been measured in blood.

The mechanism(s) of formation of acrylamide in food will be addressed by another Breakout Group (Mechanisms of Formation of Acrylamide in Foods). Preliminary observations that will affect the Surveillance, Exposure and Biomarkers WG discussions are summarized here in order to help frame the workshop discussions. The available data appear to show that both temperature and duration of heat processing are contributing factors in the formation of acrylamide in foodstuffs. Recent reports in the literature suggest that the presence of asparagines and reducing sugars may be a factor that would limit the foods affected to those containing asparagines and reducing sugars. To date, acrylamide has not been demonstrated in foods that have been processed at temperatures below approximately 120. However, data are extremely limited in this regard. Water content also appears to be an important factor as foods with high water contents have had the lowest acrylamide levels.

There are likely to be multiple sources of exposure to acrylamide. This paper addresses only issues surrounding the potential for exposure as a result of residues in foods. Nonetheless other potential sources of exposure will need to be considered in order to fully understand the significance of food residues and the effectiveness of any proposed recommendations.

Until recently the main concern for exposure to acrylamide came from occupational exposure, from exposure to residual acrylamide in polyacrylamide used in a variety of products, and from exposure in water. Occupational exposure to acrylamide is thought to arise from two main routes: inhalation exposure either to dust or to vapor, and dermal exposure.

II. Acrylamide Levels in Foods

The amount of information about residues of acrylamide is limited. Table 1 summarizes the available public data, which are extremely limited. The mean (arithmetic and geometric) and median are presented along with the number of samples and the range of values reported. Specifically, small numbers of samples have been analyzed and these represent only a limited number of foodstuffs. The foodstuffs analyzed to date represent only a subset of the types of processing and cooking methods that foods undergo before consumption. These values should be regarded as range finding estimates as the samples that have been analyzed are not representative of the food supply. To date no systematic, statistically designed data collection has been reported either in the United States or elsewhere. The samples have not been selected by processing characteristics (e.g. amount of time or temperature). Also, there has been no review of the quality of the available data or confirmation of findings. The level of detail available for samples varies widely (some samples were reported by broad category, other by the specific food item). For some categories there are only 1 or 2 samples; even the category with the most reported samples were represented by fewer than 100 analyses. FDA has undertaken the analysis of many more foods and has recently presented summary information for the available results. Additional information will be available at the conference including data for cereals that were collected in Europe.

The food categories analyzed to date include staple foodstuffs and therefore represent significant components of virtually all consumers' diets (Tables 2, 3). These categories contribute more than a third of the caloric intake for US consumers (Table 4).

A. Variation of acrylamide levels in foods: across source materials, processing types/methods

The data that are currently available were generally not collected in a way that allows analysis by processing/cooking method. Several governments have reported some limited studies in which some foodstuffs were cooked for a longer period of time. Based on those studies it appears that prolonged cooking increases acrylamide levels, in some cases dramatically. The UK cooked potato chips for an extended period of time and found the residues of acrylamide increased from 3,500 ppb to 12,800 ppb. Many of the food samples that were collected from the marketplace contained measurable amounts of acrylamide. As can be seen from Table 1, residue values vary among product categories and within categories. However, inadequate information is available to adequately characterize the variation or to allow reliable prediction of residues by processing procedure.

B. Reports to date by food, by processing technique/impact of heat/time, etc.

Some limited controlled cooking studies have been conducted by RIKILT (Netherlands) and by the UK government. Acrylamide levels increased with both time and temperature during frying. Additional research is underway including studies by the US FDA.

III. Food Consumption Patterns and Levels of Exposure

The FAO/WHO consultation concluded that the toxicological effect of relevance to the potential exposures from residues in foods is carcinogenicity since other effects would only occur if exposure were much higher. Therefore, exposure assessment should be conducted to reflect lifetime exposure in order to capture the higher exposure during childhood as well as the exposures during adult years. Lifetime can be accomplished for US consumers by utilizing the USDA Continuing Survey of Food Intake for Individuals (CSFII). The CSFII is a statistically representative sample of the dietary patterns of all age groups. The most recent USDA CSFII conducted in 1994-96,98 was analyzed to provide an overview of consumption patterns and to conduct a range-finding exposure assessment for acrylamide.

A. Characteristics of consumption of foods analyzed and reported to contain acrylamide

Table 2 provides descriptive information about consumption patterns for US consumers for foods of interest for the present discussions. The categories of foods that have been reported to contain acrylamide are consumed by a significant portion of the US population. The serving sizes range from 40 grams to 448 ml depending upon the food category. The total daily intake of these foods also varies.

Table 3 provides information about the contributions of broad food categories to calorie, protein, fiber and fat intake. These categories provide most of the caloric intake as well as most of the protein and much of the fat. Table 4 provides further detail about the contribution of food categories that have been analyzed and reported to contain acrylamide. The categories identified in Table 4 are more detailed than those presented in Table 3. The categories listed in Table 4 contribute almost 1/3 of the daily calories (626 calories of the daily intake of 1956 calories from all foods). These categories represent about 20% of the protein intake.

B. Exposure to acrylamide

Exposure assessments, using different acrylamide concentration data were provided by several countries and the IARC for use by the FAO/WHO consultation. The assessments included food consumption data from Australia, The Netherlands, USA, Norway and the IARC EPIC study that included food consumption data for 10 different European countries. Most of the analyses relied on data from Sweden for the levels of acrylamide in foods.

Exposure assessments using Monte Carlo statistical techniques were conducted using the available food consumption data for two populations (Netherlands, US) in order to provide an

estimate of likely short-term intakes based on the acrylamide residue data provided by Sweden. Although the matching of the residue data to foods consumed and the modeling methods varied slightly, the estimated exposures were similar. The resulting exposure estimates ranged from 0.8 µg/kg bw/day for the average consumer, to 3 µg/kg bw/day for the 95th percentile consumer, and 6.0 µg/kg bw/day for the 98th percentile consumer.

The FAO/WHO Consultation considered whether estimates of exposure over longer periods of time, including chronic or lifetime exposures, could be assessed given the present state of knowledge for acrylamide. It was agreed that the small and un-representative sample sets for acrylamide occurrence in foods limited the degree to which extrapolations could be made for subsets of populations based on either biologic (e.g., gender, age, ethnic background) or food consumption differences. Nonetheless, the data do allow bounding statements for the typical or median exposures that occur through food for Western European, Australian and North American diets. The general agreement of the several methods used to estimate exposure using well described food consumption data from Australia, US, Norway, The Netherlands, Sweden and from the IARC (European EPIC) indicate a lower bound estimate of typical exposures in the range of 0.3 to 0.8 µg/kg bw/day depending upon whether the average of median exposure is estimated and which age groups were evaluated.

Within a population, the FAO/WHO consultation speculated that children would have exposures that are 2-3 times those of adult consumers when expressed on a body weight basis. However, in comparing these results to the toxicological endpoint for carcinogenicity, the estimates must be adjusted to reflect the short time period of this exposure. Therefore, a weighted US population average seems most appropriate.

Although there is inadequate data to reliably estimate exposure for high consumers[1], their exposure could be several times the mean exposure.

Based on the available data, food appears to contribute a significant proportion of total exposure (based on the limited data, it seems likely that there are other important sources as well).

The foods for which residue data were submitted does not always match the foods reported as consumed. This could be a source of uncertainty in the resulting exposure estimates. The currently available information does not permit empirical inferences to be drawn on the detailed differences in ingredients or food processing that influence the level of acrylamide present in food as consumed, with the exception of limited information on the effects of duration and temperature of cooking. This information strongly suggests that the duration of cooking at high temperatures is positively, but not necessarily linearly, correlated with the level of acrylamide found in the food.

IV. **Biomarkers of Exposure: Adducts (Hemoglobin and DNA) -**

[1] High consumers would be individuals who consume larger quantities of foods containing acrylamide on a regular basis

A limited number of studies have been conducted on hemoglobin adducts derived from acrylamide and glycidamide in rodents exposed to acrylamide under defined conditions, and in humans exposed to acrylamide in the workplace or via cigarette smoking. Initial studies of the pharmacokinetics of acrylamide in rats indicated that radioactivity derived from ^{14}C acrylamide was extensively bound to hemoglobin in rats (Miller et al., 1982). Initial adduct studies were conducted with analysis of acrylamide bound to cysteine residues to form carboxamidoethylcysteine by Bailey et al. (1986). Analysis was conducted by acid hydrolysis of globin, followed by separation and derivatization of the formed carboxyethylcysteine, and analysis by GC/MS. A non-linear (sublinear) dose response was observed. Calleman et al. (1990) expanded on these observations, by measuring the acrylamide adduct at the cysteine residue, and also a cysteine adduct derived from glycidamide, a metabolite of acrylamide. Bergmark et al. (1991) evaluated the dose response for these adducts in rats dosed with 0-100 mg/kg by i.p injection, and reported a linear dose response for the acrylamide adduct, and a non-linear dose response for the glycidamide adduct. This data suggested saturation of the oxidation of acrylamide to glycidamide. By evaluating the dose rate effect of administering the same cumulative dose over a short or long duration, they suggested that the conversion of acrylamide to glycidamide occurs at a higher percentage at lower administered doses of acrylamide. A detailed pharmacokinetic model for acrylamide and glycidamide based on the hemoglobin adduct data was produced, which indicated that the oxidation of acrylamide to glycidamide was a saturable process, and results in a higher percentage of acrylamide oxidized at lower exposures (Calleman et al., 1992)

In a study of occupational exposure in China in a factory manufacturing acrylamide and polyacrylamide from acrylonitrile, extensive adduct levels derived from acrylamide, glycidamide and acrylonitrile were detected in exposed workers (Bergmark et al., 1993). Since acid hydrolysis of acrylamide and acrylonitrile adducts at cysteine residues produce identical products for analysis, a new method for analysis was used, which consisted of cleaving the adduct formed at the N-terminal valine residue with the "modified Edman degradation" and analysis by GC/MS. Extremely high levels of adducts were found in the exposed workers, with levels of the acrylamide-valine adduct ranging from 0.3-34 nmol/g globin. A clear distinction could be made between adduct levels in the exposed workers, and those in unexposed individuals. The glycidamide-valine adduct was reported to be 1.6-32 nmol/g. A more extensive analysis of the data and of the neurotoxicity associated with acrylamide exposure was reported by Calleman et al. (1994). A number of difficulties are apparent in relating the extent of exposure with adduct formation. Limited air sample data were obtained over the few months preceding the sampling for analysis of hemoglobin adducts. High variability of adduct levels between exposed workers suggested that inhalation exposure was not the predominant route. The contribution of dermal exposure to total exposure could not be estimated.

Bergmark (1997) carried out a study of acrylamide-valine in smokers, non-smokers, and laboratory workers who used acrylamide in the preparation of polyacrylamide gels. Acrylamide-valine adducts in the PAGE workers (mean 54 pmol/g) were significantly increased compared to nonsmoking controls (mean 31 pmol/g). The acrylamide adducts in smokers (mean 116 pmol/g) correlated with the number of cigarettes smoked per day. The high level of adduct in smokers was interpreted as confirmation of acrylamide in cigarette smoke. The unexpectedly high levels in nonsmoking controls was noted, with the source not known.

Perez et al. (1999) developed a more sensitive method for analysis of AAVal and GAVal using GC with tandem mass spectrometry. A background of 30 pmol/g was found in individuals without any known exposure to acrylamide. AAVal levels in 11 occupationally exposed workers were in the range 27-1854 pmol/g globin. Recorded levels of GAVal were 3-12% of those of AAVal.

Hagmar et al. (2001) reported the elevated level of AAVal in tunnel workers who had been exposed to high levels of acrylamide and N-methylolacrylamide in a grouting material. Adducts were detected in the "normal background range" from 0.020-0.070 nmol/g in 46 of the 210 workers examined. In the remaining 163, adducts ranged up to 17.7 nmol/g globin. Adducts above 0.5 nmol/g were associated with a higher level peripheral nervous system effect. No measurement of GAVal was reported.

There are a number of difficulties encountered in relating the hemoglobin adducts from acrylamide with the extent of exposure to acrylamide: the long duration of the red cell in circulation requires estimation of exposure over 120 days, exposure in occupational settings can occur via dermal and inhalation exposures, while exposure monitoring is generally only conducted via air sampling. The extent of acrylamide adduct formation for a given exposure will depend on factors including the dose, its rate and extent of absorption, and the rate of its removal by metabolism to glycidamide and to glutathione conjugates (Calleman (1996)). Adducts from glycidamide will depend on factors including the rate of metabolism of acrylamide to glycidamide, and the rate of removal of glycidamide by excretion and by further metabolism. Since these factors can vary between species, a simple extrapolation from data obtained in rodents will not necessarily yield an accurate representation of the behavior of acrylamide and glycidamide kinetics in people. Thus information to relate quantitatively biomarkers of exposure to acrylamide exposure is necessary.

DNA adducts from acrylamide have received very little attention. Adducts formed by direct reaction of acrylamide with DNA in vitro have been reported (Solomon et al., 1985). The direct reaction of acrylamide with DNA in vivo as a significant mode of action is thought to be unlikely, since this occurs so slowly, in comparison with its reaction with other macromolecules, and its metabolism and elimination. While several studies have been conducted in which radiolabeled acrylamide was administered to rodents to evaluate covalent binding to DNA, only one study has involved the measurement of a specific adduct formed in DNA. Glycidamide is a reactive epoxide metabolite that is mutagenic and is thought to mediate the carcinogenic effects of acrylamide. Glycidamide reacts with guanine to form 7-(2-carbamoyl-2-hydroxyethyl)guanine. A single study has evaluated the formation of this adduct in tissues from rats and mice administered a single dose of acrylamide (Segerback et al., 1995). Analysis was conducted by administration of ^{14}C acrylamide, and quantitation of radioactivity released from DNA that comigrated with a cold standard of 7-(2-carbamoyl-2-hydroxyethyl)guanine. No studies have been conducted that relate the level of DNA adducts with GAVal or with AAVal in rodents exposed to acrylamide.

V. Research Needs in the Area of Exposure Assessment Identified by WHO Consultation

a. Systematic examination of the relation between acrylamide levels and processing/cooking conditions
b. Hypothesis-driven model studies to elucidate sources, mechanism(s) of formation and fate of acrylamide in heated foodstuffs
c. Optimization of formulation, processing and cooking conditions to minimize and possibly eliminate acrylamide levels in foods prepared industrially and at home
d. Extend the range of foods investigated to include staple foods from different regions and diets
e. Consistent system for collecting and describing the available data
f. Biomarkers of exposure are needed to provide the most direct means of evaluating exposures to acrylamide from food and other sources. Biomarkers need to be evaluated and calibrated and their correlation with dietary intakes investigated
g. Investigate sources of exposure to humans to acrylamide to better define the relative contribution of food, smoking and other sources including the potential for endogenous formation of acrylamide
h. Investigate quantitative risk assessment models on the basis of scientific merit and uncertainty of estimates (exposure side of risk assessment)

Further investigation using different food groups according to cooking methods could be considered once more specific information is available on the relationship between cooking/processing methods and the presence/formation of acrylamide in foods.

VI. **Questions for the Breakout Group**

The FAO/WHO consultation concluded that the known food exposures did not appear to explain the levels of acrylamide-hemoglobin adducts observed. The consultation felt that tobacco smoking could provide substantial exposure to acrylamide. In contract, "exposure evaluations that exist for cosmetics, food packaging, water treatment sources," suggest that exposure levels from these known potential sources did not contribute significantly.

What factors should be considered in developing an exposure analysis for use by risk assessors? For example, should population subgroups (e.g. infants and children) be considered? Or should a population average be used? Are data available worldwide to permit such analyses? If not, what data should be developed to permit a valid exposure analysis of acrylamide intake in food?

Can the formation of hemoglobin-acrylamide adducts be considered as a biomarker for food exposure? What experiments are necessary to establish this relationship? What other circulating molecules should be investigated for adduct formation (e.g. albumin)? What characteristics of such adducts define them as indicators of exposure vs. protective mechanisms vs. toxicologically relevant?

What are the important measures to be derived from exposure assessment: the amount of acrylamide in diet, the amount of acrylamide from other sources, the total amount of acrylamide exposure, the amount of acrylamide absorbed, or the metabolic fate of the absorbed acrylamide?

Can other markers of exposure be used in assessing exposure to acrylamide, in assessing the amount of acrylamide absorbed, and in assessing the metabolism of acrylamide to glycidamide?

VII. References

Bailey, E., Farmer, P.B., and Shuker, D.E. (1987). Estimation of exposure to alkylating carcinogens by the GC-MS determination of adducts to hemoglobin and nucleic acid bases in urine. Arch Toxicol 60, 187-91.

Bergmark, E. (1997). Hemoglobin adducts of acrylamide and acrylonitrile in laboratory workers, smokers and nonsmokers. Chem Res Toxicol 10, 78-84.

Bergmark, E., Calleman, C.J., and Costa, L.G. (1991). Formation of hemoglobin adducts of acrylamide and its epoxide metabolite glycidamide in the rat. Toxicol Appl Pharmacol 111, 352-63.

Bergmark, E., Calleman, C.J., He, F., and Costa, L.G. (1993). Determination of hemoglobin adducts in humans occupationally exposed to acrylamide. Toxicol Appl Pharmacol 120, 45-54.

Calleman, C.J., Bergmark, E., and Costa, L.G. (1990). Acrylamide is metabolized to glycidamide in the rat: evidence from hemoglobin adduct formation. Chem Res Toxicol 3, 406-12.

Calleman, C.J., Bergmark, E., Stern, L.G., and Costa, L.G. (1993). A nonlinear dosimetric model for hemoglobin adduct formation by the neurotoxic agent acrylamide and its genotoxic metabolite glycidamide. Environ Health Perspect 99, 221-3.

Calleman, C.J., Stern, L.G., Bergmark, E., and Costa, L.G. (1992). Linear versus nonlinear models for hemoglobin adduct formation by acrylamide and its metabolite glycidamide: implications for risk estimation. Cancer Epidemiol Biomarkers Prev 1, 361-8.

Calleman, C.J., Wu, Y., He, F., Tian, G., Bergmark, E., Zhang, S., Deng, H., Wang, Y., Crofton, K.M., Fennell, T., and Costa, L.G. (1994). Relationships between biomarkers of exposure and neurological effects in a group of workers exposed to acrylamide. Toxicol Appl Pharmacol 126, 361-71.

Calleman, C.J. (1996). The metabolism and pharmacokinetics of acrylamide: implications for mechanisms of toxicity and human risk estimation. Drug Metab Rev 28, 527-90.

Food and Agriculture Organization of the United Nations and the World Health Organization (FAO/WHO). 2002a. Dietary exposure assessment of acrylamide in the Netherlands. FAO/WHO Consultation on the health implications of acrylamide in food Geneva, 25-27 June.

Food and Agriculture Organization of the United Nations and the World Health Organization (FAO/WHO). 2002b. Acrylamide dietary exposure assessment report. Geneva, 25-27 June.

Food Standard Agency (FSA). 2002a. Packaging gets the acrylamide all-clear. UK. http://www.foodstandards.gov.uk/multimedia/pdfs/acrylamideback.pdf

Food Standard Agency (FSA). 2002b. Food Standard Agency study of acrylamide in food background information & research findings press briefing 17.05.02.

Hagmar, L., Tornqvist, M., Nordander, C., Rosen, I., Bruze, M., Kautiainen, A., Magnusson, A. L., Malmberg, B., Aprea, P., Granath, F., and Axmon, A. (2001). Health effects of occupational exposure to acrylamide using hemoglobin adducts as biomarkers of internal dose. Scand J Work Environ Health 27, 219-26.

Institute of Science in Society (ISIS). 2002. Acrylamide in cooked foods: the glyphosate connection. http://www.i-sis.org.uk/acrylamide.php

Knonings EJM. 2002. Resultaten van de analyse op acrylamid ein producten op de nederlandse markt.

Miller, M.J., Carter, D.E., and Sipes, I.G. (1982). Pharmacokinetics of acrylamide in Fisher-344 rats. Toxicol Appl Pharmacol 63, 36-44.

NAS (1993). Pesticides in the Diets of Infants and Children", Committee on Pesticides in the Diets of Infants and Children, Board on Agriculture and Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council,National Academy Press, Washington DC, 1993)

Norwegian Study (Norwegian Food Control Authority):
http://www.snt.no/nyt/tema/akrylamid/analyse_eng.html

Segerback, D., Calleman, C.J., Schroeder, J.L., Costa, L.G., and Faustman, E.M. (1995). Formation of N-7-(2-carbamoyl-2-hydroxyethyl)guanine in DNA of the mouse and the rat following intraperitoneal administration of [14C]acrylamide. Carcinogenesis 16, 1161-5.

Solomon, J.J., Fedyk, J., Mukai, F., and Segal, A. (1985). Direct alkylation of 2'-deoxynucleosides and DNA following in vitro reaction with acrylamide. Cancer Res 45, 3465-70.

STN. (date not available). Results of acrylamide in thirty Norwegian food samples.
http://www.snt.no/nytt/tema/Akrylamid/analyse_eng.html

Swish Office of Public Health:
Results in French: http://www.bag.admin.ch/verbrau/aktuell/f/Acrylamidgehalt_liste_2_F.pdf

Swedish Study (J. Agric. Food Chem 2002) (National Food Administration):
http://www.slv.se/HeadMenu/livsmedelsverket.asp

Tareke, E., Törnqvist, M. (date not available). Acrylamide: not only in the Halland Ridge (Hallandsåsen) but also in fried hamburgers! Institute for Environmental Chemistry, University of Stockholm.

Tareke, E., Rydberg, P., Karlsson, P., Eriksson, S., Törnqvist, M. 2002. Analysis of acrylamide, a carcinogen formatted in heated foodstuffs. J. Agric. Food Chem 50:4998-5006.

The K.S. Crump Group, Inc. 1999. Consideration of the potency classification of acrylamide based on the incidence of Tunica Vaginalis Mesotheliomas (TVMs) in male Fischer 344 rats.

Table 1. Acrylamide levels in different foods and food product groups from Netherlands, Norway, Sweden, Switzerland, the United Kingdom and the United States of America

	Arithmetic Mean (ug/kg)	Geometric Mean (ug/kg)	Median (ug/kg)	Minimum-Maximum	Number of Samples	Data sources					
						CSPI	Norway	Netherlands	Sweden	Switzerland	UK
Bread	36	35	30	12-162	78		X	X	X	X	
French Fries	887	293	320	<60-12,000	67	X	X	X	X	X	X
Potato Chips	1241	967	1115	<60-3,100	60	X	X	X	X	X	X
Biscuit/Cookie	399	132	100	22.5-1,035	44		X	X	X	X	
Crisp Bread	1082	233	176	<30-4,000	28			X	X		X
Cereal	216	163	189	<30-1,346	22	X	X		X	X	X
Corn Chips	742	116	162	10.6-385	6	X			X		
Pop Corn	416	416	416	416	1				X		
Battered Fried Products	37	41	39	30-64	4				X		
Fried Potato	170	145	183	61-230	4		X			X	
Coffee	250	249	255	200-310	4					X	

Table 2. Consumption information for all food groups examined

Food Category	Percentage of US Population Consuming Food	NLEA Serving Size	Mean Consumption Per Eating Occasion	Mean Per Capita Consumption (g/day) 2-day Ave	Mean Per User[1] Consumption (g/day) 2-day Ave
Fries	28%	70 g	93.4	16.1	56.7
Potato Chips	18%	30 g	41.9	4.5	25.5
Cereal	41%	15g, 30g, 55g	49.1	15.9	38.5
Yeast Breads and Rolls	88%	50g	65.9	74.2	83.9
Baked Potato	8%	140 g	128.5	4.63	69.1
Salty Snacks	30%	30 g	55.1	11.4	38.2
Cookies	31%	30 g	39.2	9.0	28.8
Fried Potato	4%	70 g	113.4	2.4	60.6
Fried Breaded Meat	27%	85 g	110.1	19.9	74.9
Crackers	24%	30 g	26.4	4.3	18.1
Quick Breads	36%	50g	78.6	20.8	57.0
Fried Fruits & Veggies	3%	85 g	110.5	2.0	62.2
Cakes	19%	55g, 80g, 125g	89.1	10.7	55.2
Fried pastry	8%	55 g	76.9	3.8	45.4
Bars	4%	40 g	40.7	1.1	27.2
Nuts Seeds Butters	4%	2 tbsp (~32 g)	54.4	1.4	31.8
Coffee	45%	240 ml	448.1	249.3	561.5
Total: All Foods	**99%**				

[1] User defined as having consumed on at least 1 day of the survey.

Table 3. Nutrient intakes from broad CSFII food categories: All foods

Food Group (1 digit level)	Calories	Contribution To Average Total Daily Calorie Intake	Protein (g/day)	Contribution To Average Total Daily Protein Intake	Fat (g/day)	Contribution To Average Total Daily Fat Intake	Fiber (g/day)	Contribution To Average Total Daily Fiber Intake
Meat, Poultry Fish and Mixtures	385.4	20%	33.1	45%	21.3	29%	1.1	8%
Grain Products	677.3	35%	0.1	0%	20.4	28%	6.4	43%
Sugars, Sweets, and Beverages	262.9	13%	1.0	1%	1.2	2%	0.3	2%
Sugars and Sweets	66.24	3%	0.4	1%	0.1	0.1%	0.05	0.3%
NonAlcoholic Beverages	153.2	8%	0.4	1%	1.1	2%	0.2	1%
Alcoholic Beverages	43.42	2%	0.2	0.3%	0.009	0.01%	0.096	1%
Milk	224.2	11%	11.5	16%	10.7	15%	0.2	1%
Vegetables	174.8	9%	4.1	6%	7.4	10%	3.6	24%
Fruits	90.64	5%	1.0	1%	0.5	1%	1.7	11%
Fats, Oils, and Salad Dressing	60.7	3%	0.1	0%	6.4	9%	0.01	0.1%
Dry beans, peas, other legumes, nuts and seeds	48.34	2%	2.2	3%	2.6	4%	1.3	9%
Eggs	31.87	2%	2.066	3%	2.3	3%	0.02	0.1%
All CSFII Foods	**1956**		**74**		**73**		**15**	

Table 4. Nutrient analyses for foods analyzed for acrylamide categories 2-day average per capita

Food Category	Calories	Contribution To Average Total Daily Intake	Protein (g/day)	Contribution To Average Total Daily Intake	Fat (g/day)	Contribution To Average Total Daily Intake	Fiber (g/day)	Contribution To Average Total Daily Intake
Fries	41	7%	0.5	3%	2.1	9%	0.5	8%
Potato Chips	22	4%	0.3	2%	1.4	6%	0.2	3%
Cereal	59	9%	1.3	7%	0.6	2%	1.0	18%
Yeast Breads and Rolls	201	32%	7.6	40%	5.4	24%	2.1	36%
Baked Potato	6	1%	0.1	1%	0.1	1%	0.1	2%
Salty Snacks	46	7%	1.0	5%	2.2	1%	0.5	9%
Cookies	40	6%	0.5	3%	1.6	7%	0.2	4%
Fried Potato	5	1%	0.1	0.4%	0.3	1%	0.1	1%
Fried Breaded Meat	50	8%	4.4	23%	2.7	12%	0.1	1%
Crackers	20	3%	0.4	2%	0.7	3%	0.1	2%
Quick Breads	60	10%	1.5	8%	2.2	10%	0.5	8%
Fried Fruits Veggies	4	1%	0.1	0.3%	0.3	1%	0.04	1%
Cakes	38	6%	0.4	2%	1.4	6%	0.1	2%
Fried pastry	15	2%	0.2	1%	0.9	4%	0.1	1%
Bars	4	1%	0.1	0.5%	0.1	1%	0.04	1%
Nuts Seeds Butters	8	1%	0.3	2%	0.7	3%	0.1	2%
Coffee	6	1%	0.3	2%	0.05	0.2%	0.001	0.02%
Total: All Swedish Foods	626		19		23		6	