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Original Article

Vitamin K content of fast foods and snack foods in the US diet

Nathalie Weizmann[a], James W. Peterson[a], David Haytowitz[b], Pamela R. Pehrsson[b], Vincent P. de Jesus[b], Sarah L. Booth[a,*]

[a] *Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University, 711 Washington Street, Boston, MA 02111, USA*

[b] *Nutrient Data Laboratory, USDA, Agricultural Research Service, Beltsville, MD 20705, USA*

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Abstract

The predominant dietary form of vitamin K, phylloquinone (K1), is present in certain plant oils. During hydrogenation of these plant oils, K1 is converted to another form of vitamin K, dihydrophylloquinone (dK). The purpose of this study was to determine the K1 and dK content of fast foods and snack foods in the US food supply. Representative samples of key foods (109 fast foods and 23 snack foods) obtained from the National Food and Nutrient Analysis Program were analyzed for K1 and dK in duplicate by HPLC. Of the fast foods analyzed, including chicken products, hamburgers, burritos and nachos obtained from fast-food restaurants, the K1 and dK contents ranged from 0.4 to 23.7 and non-detectable (ND)—69.1 μg/100 g, respectively. Crackers and potato chips had wide ranges in K1 (1.4–24.3 μg/100 g) and dK content (ND—102 μg/100 g). When consumed frequently and in large amounts, fast foods and snack foods may be important contributors to vitamin K intake in the US diet. However, those fast foods and snack foods that contain high amounts of dK may not have equivalent contribution to vitamin K status compared to foods containing high amounts of K1 due to differences in biological activity.
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1. Introduction

The predominant dietary form of vitamin K, phylloquinone (K1), is present in green leafy vegetables (Booth et al., 1995) and certain plant oils, including soybean, canola and cottonseed (Peterson et al., 2002; Ferland and Sadowski, 1992). During the hydrogenation of these plant oils,

*Corresponding author. Tel.: +617-556-3231; fax: +617-556-3149.
E-mail address: sarah.booth@tufts.edu (S.L. Booth).

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K1 is converted to another form of vitamin K, dihydrophylloquinone (dK) (Davidson et al., 1996). Whereas the food composition data for K1 have expanded considerably in the past decade (Bolton-Smith et al., 2000; Booth et al., 1993, 1995; Piironen et al., 1997; Schurgers et al., 1999; Shearer et al., 1996), there are few data available on the dK concentrations of foods (Booth et al., 1996a). Furthermore, fast foods and snack foods, which are commonly consumed in the US diet, have not been adequately represented in previous food analysis studies.

In 1997, the National Food and Nutrient Analysis Program (NFNAP) initiated an integrated system for identifying foods and nutrients, food sampling, food preparation, sample preparation, and chemical analysis (Pehrsson et al., 2000). Approximately 1000 foods and ingredients were identified as important contributors of various nutrients in the US food supply (Haytowitz et al., 1996). Fast foods and snack foods were selected for sampling, and subsequently analyzed for K1 and dK, as reported here.

2. Materials and methods

The food samples used in this study were obtained from the USDA Nutrient Data Laboratories (NDL) as part of the NFNAP, which has been described in detail elsewhere (Pehrsson et al., 2000; Peterson et al., 2002). This food-sampling plan provides aliquots of composited, homogenized samples that are representative of Key Foods consumed in the United States. After purchase by agents at retail outlets around the US, the samples were shipped overnight to the Food Analysis Laboratory Control Center at Virginia Polytechnic Institute and State University in Blacksburg, Virginia for processing. The aliquots of the composited, homogenized samples were shipped frozen to the Vitamin K Laboratory at Tufts University, Boston, MA and stored at $-80^{\circ}C$ until they were assayed. All samples were assayed within 6 months of receipt.

The concentrations of two forms of vitamin K, K1 and dK, were determined using a reversed-phase, high-performance liquid chromatography (HPLC) procedure described elsewhere (Booth and Sadowski, 1997; Peterson et al., 2002). Samples were processed with an initial hexane–isopropyl alcohol extraction, followed by solid-phase extraction on silica columns. All samples were analyzed in duplicate, so reported values for individual samples represent the mean of the duplicates. If the coefficient of variation (CV) was greater than 15%, the assay was repeated, except in samples with K1 (or dK) concentrations of $< 5\,\mu g/100\,g$ of sample. A control of peach baby food was run in duplicate with each batch of food samples. Over a 12-month period, compilation of results gave a mean $\pm$ S.D. result of $3.7 \pm 0.3\,\mu g$ of phylloquinone/100 g of sample. As an additional measure of quality control, five aliquots of a snack food composite, that were unidentified at the time of analysis, gave a mean $\pm$ S.D. result of 9.3 ± 0.3 and $31.2 \pm 1.1\,\mu g/100\,g$ of K1 and dK, respectively. The HPLC method had a lower limit of detection of 14 pg per injection, which is equivalent to $0.006\,\mu g/100\,g$ of sample.

3. Results and discussion

The amounts of K1 and dK determined in the 109 samples of fast foods analyzed are presented in Table 1. The amounts of K1 for individual foods ranged from a low of $0.4\,\mu g/100\,g$ for a vanilla

Table 1
Phylloquinone (K1) and dihydrophylloquinone (dK) content of fast foods in the US food supply

Food		K1 (μg/100 g)			dK (μg/100 g)		
	n	Mean	s.d.[a]	Range	Mean	s.d.[a]	Range
Hamburgers							
Hamburger (2–4 oz)	17	6.4	2.4	1.1–10.1	0.1	0.4	ND-1.5
Hamburger with cheese (2–4 oz)	5	6.0	2.1	4.2–9.3	0.1	0.3	ND-0.7
Hamburger with sauce (>4 oz)	6	19.3	3.7	14.1–23.4	0.2	0.4	ND-1.0
Hamburger with cheese and sauce (>4 oz)	4	13.5	5.4	6.9–19.1	0.1	0.2	ND-0.3
Sandwiches							
Chicken sandwich	6	15.1	8.0	4.6–23.7	2.4	3.0	ND-7.7
Fish sandwich	2	13.7	—	4.9–22.5	4.6	—	ND-9.1
Chicken							
Chicken nuggets	8	7.5	3.9	2.3–13.6	13.9	6.2	4.7–23.4
Chicken tenders	4	6.3	0.6	5.7–7.2	32.6	2.4	29.6–35.3
French fries	12	11.2	4.5	5.3–17.0	42.8	18.1	14.5–61.1
Milkshake	2	0.4	—	0.4–0.4	ND	—	—
Burritos							
Burrito with bean	4	4.2	0.9	3.0–5.1	5.1	1.4	3.7–6.4
Burrito with beef	8	5.7	1.0	4.7–7.8	4.2	1.6	2.6–6.8
Burrito with chicken	4	5.3	0.5	4.8–5.9	3.8	1.0	2.6–4.7
Nachos	8	7.0	1.7	5.0–9.9	38.7	20.4	18.1–69.1
Tacos							
Taco	4	15.4	4.1	9.8–19.7	6.7	2.7	4.1–9.9
Taco with beef	8	16.0	5.4	9.3–22.4	4.5	1.0	3.5–5.9
Taco with chicken	4	8.8	1.6	7.7–11.1	4.4	0.6	3.8–5.2
Taco salad	4	10.7	2.7	7.7–14.3	10.0	2.8	6.4–13.0

[a] No s.d. reported for analyses of ⩽ 2 samples.

milkshake to a high of 23.7 μg/100 g for a chicken sandwich. The amounts of dK for individual foods ranged from non-detectable (ND) in most hamburgers and sandwiches to a high of 69.1 μg/100 g in nachos. Since potatoes contain very little vitamin K (Booth et al., 1995, 1993, 1996b), it is probable that the K1 and dK measured in French fries originated from those vitamin K-rich oils used to prepare them (Peterson et al., 2002). Likewise, foods of animal origin, such as hamburger meat, contain negligible amounts of K1 (Booth et al., 1995) so the K1 measured in the hamburgers was most likely from the plant oils used in the hamburger buns and/or the condiments used, such as iceberg lettuce and sauces.

Of those fast foods that have been previously analyzed for K1 and dK contents (Booth et al., 1995, 1996a), tacos and fish sandwiches had very similar contents compared with the current study. In contrast, hamburgers, French fries and chicken products contained more K1 and dK in the current study compared to the previous report. Given the wide ranges in nutrient content for individual foods in the current study, the differences in mean vitamin K content for individual foods compared to previous studies may simply reflect differences in sampling design. Alternatively during the 6–8-year span between studies, there may have been reformulation of these fast foods that influenced the K1 content. When fast foods were compared to frozen

Table 2
Phylloquinone (K1) and dihydrophylloquinone (dK) content of snack foods in the US food supply

Food		K1 (μg/100 g)			dK (μg/100 g)		
	n	Mean	S.D.[a]	Range	Mean	S.D.[a]	Range
Crackers:							
Saltine crackers	2	8.0	—	7.9–8.2	29.8	—	20.7–38.9
Cheese crackers with cheese	2	20.5	—	18.7–22.2	64.4	—	57.6–71.1
Cheese crackers with peanut butter	4	10.8	1.6	8.7–12.3	37.9	7.0	31.0–47.3
Chips							
Cheeto-type cheese snack chips	2	36.1	—	31.5–40.6	93.1	—	84.6–102
Corn chips	2	6.3	—	1.4–11.1	16.8	—	ND–33.6
Potato chips	2	22.0	—	19.8–24.3	ND	—	—
Olestra potato chips	2	347	—	329–366	ND	—	—
Tortilla chips	2	20.9	—	20.2–21.6	43.7	—	42.0–45.4
Olestra tortilla chips	2	180	—	169–192	ND	—	—
Pretzels	3	2.1	0.7	1.3–2.6	2.9	2.0	1.5–5.2

[a] No S.D. reported for analyses of $\leqslant$ 2 samples.

products purchased for home preparation as part of the NFNAP (Dumont et al, 2003), there were no consistent differences in K1 or dK concentrations.

The K1 and dK contents of the 23 samples of snack foods analyzed are presented in Table 2. Of those foods not containing Olestra, the amounts of K1 for individual foods ranged from a low of 1.3 μg/100 g for pretzels to a high of 40.6 μg/100 g for cheeto-type cheese snack chips. The amounts of dK for individual foods ranged from ND in corn chips and potato chips to a high of 102 μg/100 g in cheeto-type cheese snacks. The origin of the dK will be from the partially hydrogenated plant oils used in the manufacturing process (Peterson et al., 2002). Compared to previously published studies (Booth et al., 1995, 1996a), the K1 contents of the snack foods previously analyzed (saltine crackers, potato and corn chips and pretzels) were similar in the current study. In contrast, the dK content of all the snack foods reported in the current study were higher compared to previously published data, suggestive of a change in formulation of snack foods in the past 6–8 years (Booth et al., 1996a). Snack foods prepared with Olestra have high phylloquinone concentrations, but non-detectable amounts of dK. In Olestra-containing snack foods, K1 is added to offset any potential malabsorption of vitamin K associated with this lipophilic fat substitute.

Fast foods and snack foods are items that are often consumed in large quantities and as such, have the potential to have an important contribution to overall vitamin K intake. However, one study in young adults indicated that dK had less biological activity compared to an equivalent amount of the parent compound, K1 (Booth et al., 2001). Vitamin K-dependent reactions are related to both the length and the isomeric configuration of the side chain (Suttie et al., 1992). Hydrogenation of K1 to dK results in the saturation of a single double bond in the side chain, but conserves the napthoquinone ring, which is the active site for γ-carboxylation and influences the functionality of vitamin K-dependent proteins. Comparison of plasma K1 and dK concentrations following repletion suggests that dK is not as well absorbed compared to K1 (Booth et al., 2001).

Differences in absorption between the two forms of vitamin K may result in differences in their availability as cofactors for γ-carboxylation of vitamin K-dependent proteins. Carboxylation of hepatic proteins, which are responsible for coagulation, were partially conserved, whereas carboxylation in the extra-hepatic proteins, such as those in bone, was not conserved when dK was the exclusive form of vitamin K consumed after short-term K1 depletion among young adults (Booth et al., 2001). Therefore, despite its abundance in the fast foods and snack foods, dK may not have the equivalent contribution to the total intake of vitamin K, hence the current recommended daily intake of 90–120 μg/day for adults (Institute of Medicine, 2001), as compared to K1. More research is required though before K1 equivalents of dK can be determined.

In conclusion, fast foods and snack foods may be important contributors of K1 and dK in the US diet. However, differences in the biological activity among the different forms of vitamin K may complicate the interpretation of vitamin K intakes required for an adequate vitamin K status.

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Disclaimers

Any opinions, findings, conclusions or recommendations expressed in this publication are those of the authors, and do not necessarily reflect the view of the US Department of Agriculture.

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