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Heidi M. Moss
Utah State University

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KNOWLEDGE OF COUMADIN USE AND VITAMIN K INTERACTION IN ATRIAL FIBRILLATION PATIENTS

by

Heidi Michelle Moss

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of the requirements for the degree

of

DEPARTMENTAL HONORS

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Approved:

**Thesis/Project Advisor
Advisor Megan Bunch**

**Departmental Honors
Janet Anderson**

Director of Honors Program

Dr. Christie Fox

UTAH STATE UNIVERSITY

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Abstract

Background: Atrial fibrillation (AF) is the most commonly observed arrhythmia and is expected to increase to over 12 million in the next few decades. Patients with AF are at high risk of stroke due to the use of Coumadin in combination with stroke risk factors such as age >75 years, hypertension, diabetes, heart failure, and prior stroke or transient ischemic attack. Coumadin specifically targets the blood clotting cascade by inhibiting the regeneration of vitamin K needed for the activation of clotting factors. A 100 mcg increase in vitamin K intake over at least 4 days can reduce patient internationalized national ratio (INR) by 0.2 units. INR values outside of the recommended range of 2.0-3.5 increase the risk of intracranial bleeding and stroke. The objective of this study was to assess the understanding of the AF population related to their role in reducing the risks associated with Coumadin and the effects of proper understanding on stroke risk with a particular emphasis on patient understanding of the interaction between vitamin K and Coumadin.

Methods: Patients with known AF (n=75) who were receiving treatment from the Utah Heart Clinic and were currently taking Coumadin were asked to complete a one-time questionnaire of 52 questions related to Coumadin use and its' drug-nutrient interactions. Data collected was analyzed to identify any vitamin K and diet knowledge deficits related to nutrition and Coumadin use.

Results: Sixty-eight of the patients had at least one risk factor for stroke with hypertension, the most common stroke risk factor (58.7% of the population). Age > 75 years was the second most common, followed by heart failure. Only 63.9% of the patient population had some understanding of diet/vitamin K and Coumadin use.

Conclusion: This study demonstrates a lack of patient knowledge regarding the interaction of Coumadin and nutrition/vitamin K in patients with additional stroke risk factors.

Introduction

Atrial Fibrillation and Stroke

Atrial fibrillation (AF) is the most commonly observed arrhythmia in clinical practice. The number of people affected by AF is expected to increase from 5.6 to over 12 million in the next few decades (1,2). Both the Framingham study and a cohort from Olmsted County, Minnesota have shown age-adjusted increases in the prevalence and incidence of atrial fibrillation from the 1960's to 1989 (3-4). While age is a key risk factor for AF, other population demographics may also contribute to the increased prevalence of AF. The epidemiologic changes of AF are a global phenomena. The incidence and prevalence of AF in the Netherlands are similar to those in the US. AF admissions are also on the rise in China (5-6).

In patients with AF, systemic embolization can result from stasis in the left atrium and appendage, leading to stroke, significant morbidity, and/or mortality. Although absolute stroke risk varies among AF patients, stroke risk can be stratified based upon clinical and echocardiographic variables. Stroke risk factors, identified over the course of five AF prevention trials, include age >75 years, hypertension, diabetes, heart failure, and prior stroke or transient ischemic attack. There is a significant benefit obtained from the use of the anticoagulant medication Coumadin, as compared to aspirin, for the prevention of stroke in AF patients, particularly those at highest risk (i.e., those with more than one baseline risk factor) (7-11).

Risks of Coumadin Use

Although Coumadin is effective in reducing stroke, it carries with it the major concern of intracranial bleeding (12). The adjusted odds of developing intracranial bleeding (relative to an internationalized national ratio (INR) of 2.0 to 3.0) were 4.6 and 8.8 for INRs in the range of 3.5 to 3.9 and ≥ 4.0 , respectively (12). Elderly patients and those with prior cerebrovascular disease represent some of the highest risk populations for stroke. However, these groups are also at

highest risk for intracranial bleeding with Coumadin anticoagulation. Similarly, risk of thromboembolism increases significantly when the INR is subtherapeutic, less than 2.0 (13). Due to the risks of over- and under-coagulation, chronic use of Coumadin requires frequent INR/protime monitoring.

Coumadin anticoagulant Control and Education

To attenuate the risk of intracranial bleeding and stroke, good anticoagulation control becomes essential in the long-term management of patients with AF. However, maintenance doses of Coumadin vary significantly, ranging from less than 2 mg/day to ≥ 10 mg/day. The variability stems from many factors including: nutritional status, hepatic function, intestinal absorption, compliance, drug interactions, and genetic polymorphisms (14-16). Of these factors, understanding of drug interactions, nutrition, and compliance are those that can be positively influenced through intervention and education, improving patient outcomes.

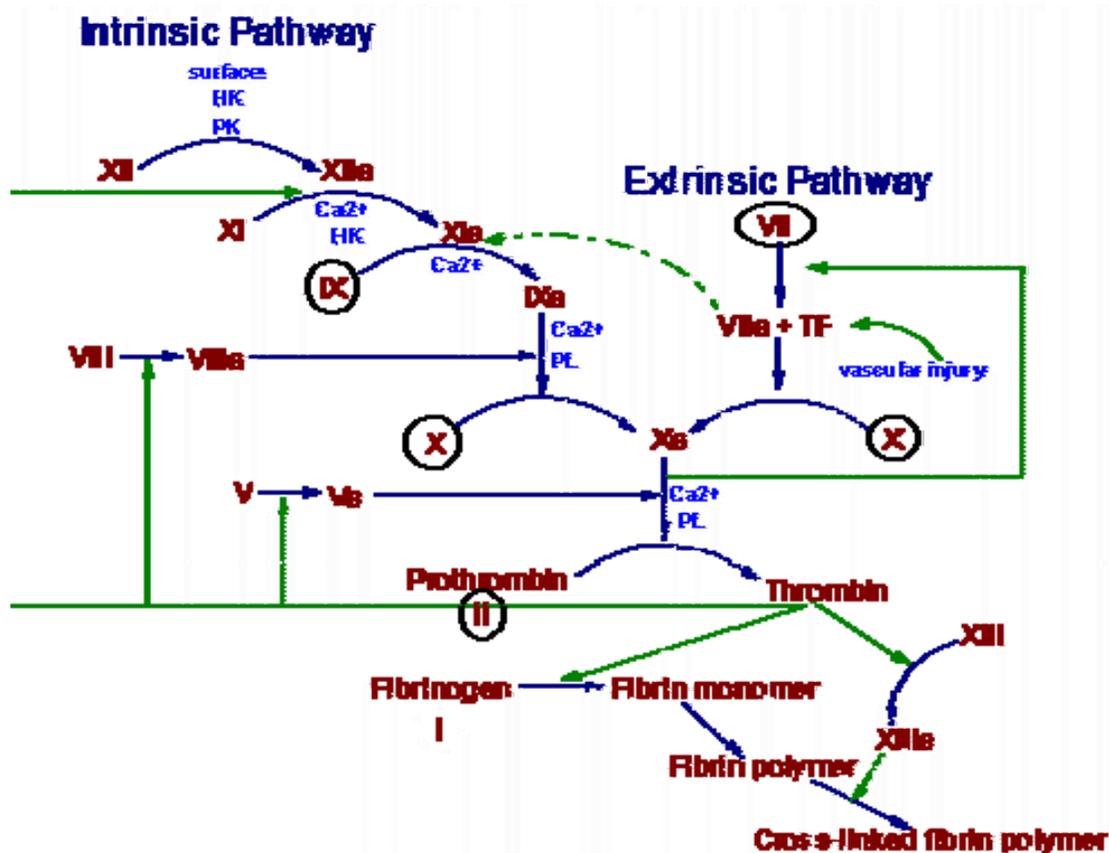
Coumadin and Drug Interactions

Many drugs interact with the metabolism of Coumadin. The number increases almost daily as new drugs enter the patient market. These drugs can lead to overanticoagulation, underanticoagulation, or increased bleeding risk by INR independent changes, such as altered platelet function or gastrointestinal bleeding. Interactions with medications in patients taking Coumadin are a widespread problem in clinical practice. For example, in one study, nearly one-third of patients taking Coumadin had also been prescribed a medication known to adversely interact with Coumadin (17). Consequently, patients must be instructed not to take any new medications, including herbal products/supplements or over-the-counter medications, without the knowledge of their attending physicians. Although education communicating these ideas is widely available and frequently used for patients with cardiac disorders (e.g., Long QT syndrome), it is not frequently used for patients taking Coumadin.

Vitamin K and its Role

Related to nutrition, vitamin K intake must be closely monitored, as it is a key nutrient that interacts with anticoagulation therapy. Vitamin K plays a key role in the blood clotting cascade. Vitamin K's principal action in blood clotting is to enable the generation of the active cofactor needed for carboxylation of glutamic acid, which interacts directly with clotting factors II, VII, IX, X, prothrombin, and specific clotting proteins (see Figure 1). Synthesis of glutamic acid allows for the binding of these factors and proteins to calcium, which is required for incorporation into the clotting cascade (18-20).

Figure 1: Clotting Cascade - Vitamin K affects clotting factors II, VII, IX, and X (21)

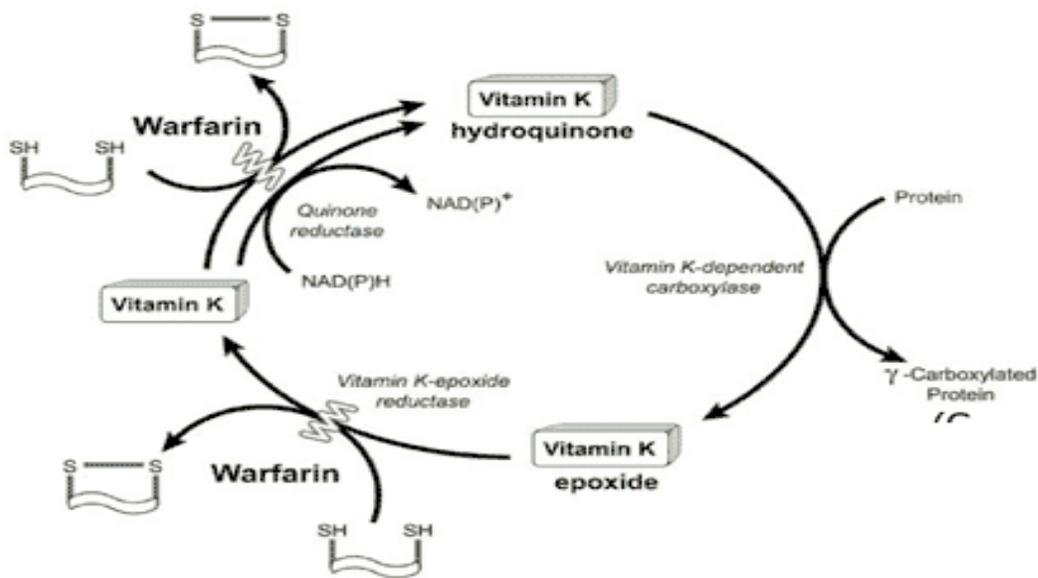


King, Michael W. Blood Coagulation: The Clotting Cascades. *Indiana University School of Medicine Medical Biochemistry*. November 19, 2008. <http://themedicalbiochemistrypage.org> (21)

Interaction Between Vitamin K and Coumadin

Coumadin specifically targets the blood clotting cascade by inhibiting the regeneration of vitamin K from vitamin K epoxide. Thus, the medication blocks the generation of the cofactor required in the carboxylation of glutamic acid and the ability of the clotting factors to bind to calcium and participate in the clotting cascade (18-20). Due to decreased regeneration of vitamin K, the formation of vitamin K dependent clotting factors is much more susceptible to dietary intake (19). Consequently, intake of vitamin K, either through foods or supplementation, can alter the effect of Coumadin.

Figure 2: Coumadin interaction with Vitamin K (22)



Higdon, Jane. The Vitamin K Cycle. *Linus Pauling Institute: Micronutrient Research for Optimum Health*. May 2004. <http://lpi.oregonstate.edu/infocenter/vitamins/vitaminK/kcycle.html> (22)

Vitamin K Sources and RDA

The primary sources of dietary vitamin K are dark green vegetables such as broccoli, spinach, collard greens, romaine lettuce, and vegetable oils. From diet alone, the mean intake of vitamin K is between 74 and 117 mcg/day in adults, which meets the current adequate intake (AI) requirement of 90 mcg/d for adult women and 120 mcg/day for adult men (23-24). For

those taking Coumadin, the National Institute of Health recommends keeping vitamin K intake constant with no more than one serving of foods high in vitamin K and three servings of foods moderately high in vitamin K per day (23). Research regarding vitamin K and Coumadin has reported intakes as low as 29 to 40 mcg/day (19-24). Vitamin K intake is highly correlated with serum vitamin K, and some studies have noted serum levels less than 1.5 mcg/L compared to a normal value, which is considered to be greater than 4.5 mcg/L, in patients taking Coumadin with unstable anticoagulation control (23).

Table 1: Sources of Vitamin K

Phylloquinone (Vitamin K) mcg/100g			
< 10	10-50	> 100	> 200
Milk	Asparagus	Cabbage	Broccoli
Butter	Celery	Lettuce	Kale
Eggs	Green beans	Brussels sprouts	Swiss chard
Cheese	Avocado	Mustard Greens	Turnip
Meats	Kiwi	Soybean oil	Watercress greens
Fish	Pumpkin (canned)	Canola oil	Collards
Corn	Peas		Spinach
Cauliflower	Peanut Butter		Salad greens
Grains	Lentils		
Fruits (most)	Kidney Beans		
Brewed tea	Pinto Beans		
Tomatoes	Soybeans		
	Coffee (brewed)		
	Olive oil		

Source: Groper SS, Smith JL, Groff JL. Advanced Nutrition and Human Metabolism. 4th ed. Wadsworth 2005 (25).

Table 2: Vitamin K Adult RDA Values

Vitamin K Adequate Intake (AI)	
Age/Gender	Vitamin K AI in mcg/d
Male > 19 years	120
Female > 19 years	90

Source: Groper SS, Smith JL, Groff JL. Advanced Nutrition and Human Metabolism. 4th ed. Wadsworth 2005 (25).

The Effect of Vitamin K Intake on INR

When assessing the interaction between vitamin K and Coumadin, it is important to monitor INR to assess the effect of dietary intake on the effectiveness of the Coumadin dose on blood clotting. The ideal INR range for AF patients taking Coumadin is 2.0-3.0 (23). Weekly changes of 714 mcg of dietary vitamin K intake have been found to significantly affect weekly INR values by one unit, equivalent to a weekly change in a Coumadin dose of 14.5 mg (18). Vitamin K intake of 150 mcg in women and 200 mcg in men is enough to significantly decrease INR. For every 100 mcg increase in vitamin K intake over at least 4 days, INR has been found to reduce by 0.2 units (18,23-24). Anticoagulant therapy with Coumadin is targeted at maintaining INR values between 2.0-3.0 to balance the risk between stroke and excessive bleeding. Intakes as low as 250 mcg can contribute to unstable anticoagulant therapy, and once vitamin K intake exceeds 500 mcg, it has been shown to reverse anticoagulation therapy (18).

Vitamin K and Unstable Anticoagulation

Patients with unstable coagulation compared to patients with stable INR values have consistently lower intakes of vitamin K. The impact of changes in vitamin K intake is amplified in patients with compromised vitamin k status and low serum values. In addition, it has been found that a patient's INR decreases with addition of a multivitamin supplement containing only

25-50 mcg of vitamin K were found to significantly lower dietary intake and serum levels of vitamin K (19,20). Although patients are cautioned about leafy vegetables that can contain very high levels of vitamin K (one-half cup of frozen spinach contains >500 micrograms of vitamin K), other high vitamin K products are less well-known, such as herbal supplements and multivitamins (26).

Vitamin K and Coumadin Dosing

Consumption of less than 500 mcg of vitamin K per week requires on average a Coumadin dose of 35 mg/wk compared to 38 mg/wk Coumadin for vitamin K intakes of 500-1000 mcg/wk and 37 mg/wk Coumadin for intakes greater 1,000 mcg/wk of vitamin K (18). An average Coumadin dose increases from 3.5 mg/day to 5.7 mg/day with vitamin K intakes below 250 mcg/d and above 250 mcg/d, respectively (24). Participants, whose vitamin K intake remained constant, manifested little or no change in Coumadin dosing after correcting for other drug and supplement interactions. This finding supports the recommendation to consume a consistent amount of vitamin K (18,23-24).

Patient Knowledge of Coumadin Side Effects

Coumadin is a medication that has multiple adverse effects, many of which patients may be unaware of. In addition to using frequent INR testing, the main overt sign of inappropriate anticoagulation therapy is excessive bleeding or bruising. A pre-test administered by Mazor et al. (27) showed that 5% of participants were not aware that it was important to contact the anticoagulation clinic if they had a cut that would not stop bleeding, 13% did not know to contact the clinic if they noticed unexplained bruising or blood in their urine or stools, and 35% of participants did not know that excessive Coumadin could cause a gastrointestinal bleed (27). As INR can measure inappropriate responses to anticoagulation therapy, it is imperative that patients understand the importance of frequent testing.

In assessing patient attitudes towards INR testing, 13% of the pre-test respondents did not believe that missing a lab appointment on occasion was a problem and 12% reported being able to tell if their Coumadin was at the right level by how they physically felt (27). Finally, 83% of participants were aware that too little Coumadin could cause a stroke, but only 21% were aware that too much Coumadin could cause a stroke (27). Patients must be aware of the overt signs of inadequate coagulation and the importance of INR testing to appropriately monitor anticoagulation therapy and reduce risk of excessive bleeding and stroke.

Patient Compliance

Compliance to medications of any type is essential to receive study-validated benefits. In a prior study of elderly patients, nonadherence to Coumadin was estimated at 21%; the patients sampled missed pills more often rather than took too many (28). One important additional finding was that patients often perceived that they were more adherent than they actually were throughout the study. It is likely that patients not enrolled in a study will actually have much higher rates of nonadherence and will be at risk for either bleeding or thromboembolism as a result. Even if vitamin K education and knowledge in relation to Coumadin use is adequate it may not translate into appropriate INR values.

Research Objective

INR values outside of the recommended range of 2.0-3.5 increase the risk of intracranial bleeding and stroke (11). The relationship between vitamin K intake and changes in INR leading to over- and under-anticoagulation has been established (19-20,23-24). Although medical knowledge is not yet advanced enough to remediate many of the primary causes of variability, intervention and education focusing on proper nutrition and vitamin K intake may improve patient outcomes (14-16).

To date, few, if any, studies have measured patient understanding of Coumadin use and its implications in stroke and intracranial bleeding risk. The objective of this study was to assess the understanding of the AF population related to their role in reducing the risks associated with Coumadin and the effects of proper understanding on stroke risk with a particular emphasis on patient understanding of the interaction between vitamin K and Coumadin.

Research Design and Methods

This study was conducted at Intermountain Medical Center (Murray, Utah) in collaboration with Utah State University (Logan, Utah). Patients with known AF (n=75) who were receiving treatment from the Utah Heart Clinic and were currently taking Coumadin were asked to complete a one-time questionnaire of 52 questions related to Coumadin use and its interactions. Participation was voluntary and no identifying information was obtained. The questionnaire was completed under the supervision of a registered dietitian or student dietitian. The study was approved by the Utah State University Institutional Review Board (protocol #2187).

The data collected was analyzed to identify any vitamin K, vitamin supplement, and any knowledge deficits related to nutrition and Coumadin use. The data was stratified by stroke risk.

Results

The mean age of the patient population was 69.2 (n=75). The data was stratified by gender to assess differences between male and female. The patients were categorized according to stroke risk, based on the CHADS₂ Score. The CHADS₂ score is used to estimate risk of stroke in AF patients (7-11).

Sixty-eight of the patients had at least one risk factor for stroke (see Table 1). The seven remaining patients with no stroke risk factors were taking Coumadin because they had recently undergone a cardiac ablation.

Hypertension was the most common stroke risk factor (58.7% of the population). Age > 75 years was the second most common, followed by heart failure.

Table 1. Stroke Risk Characteristics

Item	M+F n(%)	M n(%)	F n(%)
n	75	47	28
Age	69.2	66.7	72.3
Stroke Risk			
1. Hypertension	44(58.7)	28(59.6)	16(57.1)
2. Heart failure	25(33.3)	14(29.8)	11(39.3)
3. Age > 75	26(34.7)	12(25.5)	14(50.0)
4. Diabetes	12(16.0)	7(14.9)	5(17.9)
5. TIA	19(25.3)	11(23.4)	8(28.6)
7. TIA on Coumadin	6(8.0)	3(6.6)	3(10.7)

To assess general knowledge concerning Coumadin use, the data was categorized by response to certain questions in the questionnaire. Questions pertaining to diet in regards to Coumadin use were assessed. The following questions were used to assess knowledge: “Do you think getting enough vitamin K is important?”; “What do you think Vitamin K does for us?”; “Do you know how to interpret a supplement facts label?”; “Can changing your diet change your Coumadin dose?”; “Are you aware that you get vitamin K from the foods you eat?”; “Do you know how to interpret a nutrition facts label?”; and “Is it important to watch how much vitamin K you get each day when you are on Coumadin?” If the patient answered yes to one of the aforementioned questions, it was deemed that the patient had some knowledge related to

Coumadin use. Table 2 represents the percent of the patient population who had some understanding of diet and Coumadin use.

Table 2. Coumadin Knowledge

Item	M+F	M	F
Dietary influence (%)	63.9	65.1	61.8

Discussion

Seventy-five patients participated in this study. Of these, 68 had at least one stroke risk factor. Because only seven participants did not have at least one stroke risk, data was not analyzed by stroke risk versus non-stroke risk as originally intended. In the future, data will be stratified based on the number of stroke risks based on the CHADS₂ score.

This study found that 63.9% of the population had some knowledge related to diet and Coumadin use. Because the data was categorized by response to certain questions, the results do not identify any differences in knowledge regarding the differences in questions asked. In the future, the questions will be weighted by importance to assess the degree of knowledge of diet and Coumadin use.

Changes in INR are observed with short-term vitamin K intake; however, INR values and Coumadin dosing are not affected over an extended period of time if vitamin K intake returns to levels normally consumed by participants (23-24). Schurgers et al. (23) found that meals with a large amount of vitamin K rich foods significantly decreased INR by 0.3-0.6 units. However this effect was not maintained past three to seven days (23). Khan et al. (24) demonstrated a change of 0.2 units in INR with 100 mcg/d increases in vitamin K over a 4 and 7-day period. However, when INR levels were measured over 28 days following changes in vitamin K intake there was

no significant change in INR compared to baseline (24). Once Coumadin dosing has been established and INR levels stabilize, most patients have their INR tested monthly. Short-term changes in vitamin K intake that affect INR levels may not be noticed in monthly lab values due to the stabilization of INR over time. This could be a concern with patients who are not aware of the interaction of vitamin K and have a great variability in dietary vitamin K intake.

Patients taking Coumadin are instructed to maintain a consistent intake of vitamin K (23). This can easily be misconstrued to mean that foods containing vitamin K should be avoided. In addition, if patients are unaware of what foods contain vitamin K, it impacts their ability to maintain consistency in vitamin K intake and may lead to avoidance of foods, which contain little or no vitamin K (20). Misinterpretation of vitamin K recommendations may be the cause of low dietary intake and serum vitamin K levels found in patients with unstable control (20). In patients with low vitamin K intake and serum levels, use of a multivitamin containing 25 mcg of vitamin K has been found to decrease INR by a median of 0.51. For those patients with normal serum vitamin K levels who consume the RDA of vitamin K, use of a multivitamin containing 25 mcg of vitamin K has been found to have no significant change in INR (23).

Chronic low dosing of vitamin K may attenuate INR changes in response to small variations in vitamin K intake in those patients with low serum values. Sorano et al. (29) demonstrated an improvement of anticoagulation therapy when patients consume at least 20-40 mcg/day of vitamin K. The effectiveness of chronic supplementation of vitamin K needs to be investigated further to determine if this is feasible and safe. Furthermore, changes in patient education regarding Coumadin may help improve patients understanding of appropriate intake of vitamin K and in what products it can be found.

Couris et al. (30) developed a brief, self-assessment instrument (K-Card) to determine daily variations in dietary vitamin K intake that could be validated and used in the assessment of patients

receiving oral Coumadin anticoagulant therapy. The K-card included foods that previous studies have shown provide 5 mg vitamin K per serving, foods with lower vitamin K contents that are consumed in larger quantities, and common supplements and herbal products. The items listed were color coded into categories such as vegetables, meat/poultry/fish, mixed dishes, fats/oils/salad dressings, snacks, desserts, beverages, and dietary supplements (30). The mean dietary vitamin K intake estimated using the K-Cards was 138.8 +/- 15.7 mg compared to diet record averages of 136.0 +/- 15.8 (30). Self-assessment measures such as the K-Card can accurately assess vitamin K status and may be helpful in increasing patient understanding of what foods contain vitamin K.

This study showed that over 30% of patients were unaware of the effects of nutrition and vitamin K on Coumadin therapy and stroke risk. While 63.9% of patients had some knowledge of vitamin K and its interaction with Coumadin, they may have only answered one question regarding nutrition and vitamin K correctly. The percent of patients who have some knowledge regarding nutrition and Coumadin may be overstated, as it does not account for the level of knowledge the patient had about this relationship or the level of awareness the patient had pertaining to what foods and products contained vitamin K. In short, many patients are still lacking the necessary education regarding Coumadin to maintain adequate INR values and minimize the risk of stroke and intracranial bleeding.

In a randomized clinical trial conducted by Khan et al. (31), 125 patients over the age of 65 were divided into three groups: patients who received usual clinical care, patients who received additional education regarding Coumadin, and patients who received additional Coumadin education in combination with education on how to self-monitor INR. Coumadin education was done in a two-hour session given to patients in groups of 2-3 that covered information regarding atrial fibrillation, clinical benefits and risk of Coumadin use, and factors that affect INR with emphasis on drug interactions and diet (31). The INR standard deviation

decreased by 0.24 in the combination group, 0.26 in the Coumadin education group, and only 0.16 in the control group meaning that those who received additional Coumadin education beyond usual care had a decreased variability in INR (31).

Although personalized patient education is effective in increasing patient knowledge, it is costly and time consuming for healthcare employees. Mazor et al. (27) compared the effectiveness of videos depicting dialogue between a physician and patient regarding Coumadin use and its interactions to a control group who received standard care. Coumadin-related knowledge and belief in the importance of lab testing improved between pre and post-test for those patients who viewed the educational video, either with statistical evidence, narrative evidence, or a combination of both, compared to the control group who received usual care and not the educational video (27). Additional attention toward administration of appropriate education can lead to increased patient knowledge.

Stone et al. (32) compared the use of personalized patient education with the use of educational videotapes. Both groups scored significantly higher on post education questionnaires designed to assess knowledge. In addition, this study found no difference in knowledge improvement between patients who received Coumadin education through personalized teaching or videotape (32). A follow-up questionnaire showed that patients in each group rated the effectiveness and importance of the education equally (32).

One-on-one teaching by physicians and nurses is time consuming. In the current era of both physician and nursing shortages, one-on-one teaching is not feasible for diseases that are highly prevalent in the community, such as AF. An alternative is group teaching through audiovisual presentations (videotapes, DVDs, or internet-based). Audiovisual presentations have been shown to be effective tools if the presentations are appropriate for the audience, brief and focused, and deliver the necessary information (33-35). Patients can view these presentations in

their homes as frequently as needed. One additional concern to using these educational methods to increase knowledge is how to ensure knowledge is translated to action and improved patient results.

Conclusion

This study demonstrates a lack of patient knowledge regarding the interaction of Coumadin and vitamin K in patients with additional stroke risk factors. Inadequate education regarding vitamin K and its interaction with Coumadin can lead to an increased risk of stroke and intracranial bleeding (12,13,18). Education of the drug-nutrient interaction of Coumadin can reduce patient risk when coupled with compliance and can be effectively administered through audiovisual mediums. Further research is warranted to quantify the effect of lack of patient knowledge on morbidity and mortality and appropriate educational interventions (31).

References

1. Go, AS et al. Prevalence of diagnosed atrial fibrillation in adults: National implications for rhythm management and stroke prevention: the anticoagulation and risk factors in atrial fibrillation (ATRIA) study. *JAMA* 2001;285:2370-2375
2. Miyasaka, Y et al. Secular trends in incidence of atrial fibrillation in Olmstead county, Minnesota, 1980 to 2000, and implications on the projections for future prevalence. *Circulation* 2006;114:119-125
3. Tsang, TS et al. The prevalence of atrial fibrillation of incident stroke cases and matched population controls in Rochester, Minnesota. *J of Amer College of Cardiology* 2003;42:93-100
4. Wolf, PA et al. Secular trends in the prevalence of atrial fibrillation: The Framingham study. *American Heart Journal* 1996;131:790-795
5. Heeringa, J et al. Prevalence, incidence, and lifetime risk of atrial fibrillation: the Rotterdam study. *European Heart Journal* 2006;27:949-953
6. Wen-Hang, QI. Retrospective investigation of hospitalized patients with atrial fibrillation in mainland China. *International J of Cardiology* 2005;105:283-287.
7. The Boston Area Anticoagulation Trial for Atrial Fibrillation Investigators. The effect of low-dose warfarin on the risk of stroke in patients with nonrheumatic atrial fibrillation. The Boston Area Anticoagulation Trial for Atrial Fibrillation Investigators. *N Engl J Med* 1990;323:1505-11
8. Stroke Prevention in Atrial Fibrillation Study. Final results. *Circulation* 1991;84:527-39.
9. Connolly SJ et al. Canadian Atrial Fibrillation Anticoagulation (CAFA) Study. *J Am Coll Cardiol* 1991;18:349-55.
10. Ezekowitz MD et al. Warfarin in the prevention of stroke associated with nonrheumatic atrial fibrillation. Veterans Affairs Stroke Prevention in Nonrheumatic Atrial Fibrillation Investigators. *N Engl J Med* 1992;327:1406-12.
11. Petersen P et al. Placebo-controlled, randomized trial of warfarin and aspirin for prevention of thromboembolic complications in chronic AF. The Copenhagen AFASAK study. *Lancet* 1989 Jan 28;1(8631):175-9.
12. Fang MC et al. Death and disability from warfarin-associated intracranial and extracranial hemorrhages. *Am J Med.* 2007;120:700-5.
13. Oake N et al. Frequency of adverse events in patients with poor anticoagulation: a meta-analysis. *CMAJ.* 2007;176:1589-94
14. Hallak HO et al. High clearance of (S)-warfarin in a warfarin-resistant subject. *Br J Clin Pharmacol.* 1993;35:327-30.
15. Hulse ML. Warfarin resistance: diagnosis and therapeutic alternatives. *Pharmacotherapy.* 1996;16:1009-17.
16. Anderson JL et al. Randomized trial of genotype-guided versus standard warfarin dosing in patients initiating oral anticoagulation. *Circulation.* 2007;116:2563-70.
17. Juurlink DN. Drug interactions with warfarin: what clinicians need to know. *CMAJ.* 2007;177:369-71.
18. Couris R et al. Dietary vitamin K variability affects international normalized ratio (INR) coagulation indices. *Int J Vitam Nutr Res.* 2006;76:65-74.

19. Sconce E et al. Patients with unstable control have a poorer dietary intake of vitamin K compared to patients with stable control of anticoagulation. *Thromb Haemost.* 2005 May;93(5):872-5.
20. Kurnik D et al. Over-the-counter vitamin K-containing multivitamin supplements disrupt warfarin anticoagulation in vitamin K-depleted patients. *Thromb Haemost.* 2004 Nov;92(5):1018-24.
21. King, MW. Blood Coagulation: The Clotting Cascades. *Indiana University School of Medicine Medical Biochemistry.* November 19, 2008. <http://themedicalbiochemistrypage.org>
22. Higdon, J. The Vitamin K Cycle. *Linus Pauling Institute: Micronutrient Research for Optimum Health.* May 2004. <http://lpi.oregonstate.edu/infocenter/vitamins/vitaminK/kcycle.html>
23. Johnson MA. Influence of vitamin K on anticoagulant therapy depends on vitamin K status and the source and chemical forms of vitamin K. *Nutrition Reviews.* 2005;63:91-100.
24. Khan T et al. Dietary vitamin K influences intra-individual variability in anticoagulant response to warfarin. *British J of Haematology.* 2004;124:348-354.
25. Groper SS et al. *Advanced Nutrition and Human Metabolism.* 4th ed. Wadsworth 2005.
26. Juurlink DN. Drug interactions with warfarin: what clinicians need to know. *CMAJ.* 2007;177(4): 369-71.
27. Parker CS et al. Adherence to warfarin assessed by electronic pill caps, clinician assessment, and patient reports: results from the INRANGE study. *J Gen Intern Med.* 2007;22(9): 1254-9.
28. Mazor KM et al. Patient education about anticoagulant medication: Is narrative evidence or statistical evidence more effective? *Patient Education and Counseling* 2007;69:145–157
29. Sorano GG et al. Controlled vitamin K content diet for improving the management of poorly controlled anticoagulated patients: a clinical practice proposal. *Haemostasis* 1993;23:77-82
30. Couris RR et al. Development of a self-assessment instrument to determine daily intake and variability of dietary vitamin K. *J Am Coll Nutr.* 2000 Nov-Dec;19(6):801-7.
31. Stone S et al. Comparison between videotape and personalized patient education for anticoagulant therapy. *J Fam Pract* 1989, 29:55-57.
32. Khan TI et al. The value of education and self-monitoring in the management of warfarin therapy in older patients with unstable control of anticoagulation. *Br J Haematol* 2004, 126:557-564.
33. Freda MC et al. Are they watching? Are they learning? Prenatal video education in the waiting room. *J Perinat Ed* 1994;3:20-28.
34. O'Donnell L et al. Reducing AIDS and other STDs among inner city Hispanics: The use of qualitative research in the development of video-based patient education. *AIDS Educ Prev* 1994;6:140-153.
35. Leaffer T et al. The Internet: An underutilized tool in patient education. *Comput Nurs* 2000;18:47-52.

Age _____

Please circle:

Gender

Male

Female

Education Level

Less than the 8th Grade

8-12th Grade

High School Graduate

College Graduate

Advanced Degree

Stroke Risk Factors

1. High blood pressure: Yes/No
2. Heart failure: Yes/No
3. Age greater than 75 years: Yes/No
4. Diabetes: Yes/No
5. Prior stroke or mini-stroke (TIA): Yes/No
7. Prior stroke or mini-stroke when on Coumadin: Yes/No

If you had a stroke on Coumadin, was your blood level:

- a. Too low
- b. Normal
- c. Too high
- d. Not sure

Other Cardiac Problems

1. Have you had a prior heart attack: Yes/No
2. Have you had a stent or bypass surgery: Yes/No
3. Do you have any problems with your heart valves: Yes/No
If yes, was the problem:
 - a. Narrow
 - b. Leaky
 - c. Not sure

4. Have you had surgery for your heart valves: Yes/No

Please answer the following questions:

1. Have you ever experienced bleeding in your urine or stools? Yes/No
2. Have you ever received a blood transfusion because of bleeding? Yes/No
3. Have you fallen in the past year? Yes/No
4. If you have fallen in the past year, how many times? _____
5. How long have you been on Coumadin?
 - a. Less than 1 year
 - b. 1 year – 5 years
 - c. 5 years – 10 years
 - d. Greater than 10 years
 - e. Not sure
6. Do you take your Coumadin as prescribed by your doctor? Yes/No
7. Do you ever skip your Coumadin dose? Yes/No
8. Do you ever double up your Coumadin dose? Yes/No
9. Do you ever not refill your Coumadin because of cost? Yes/No
10. What is the most common reason why you may not take your Coumadin dose?
 - a. Cost
 - b. Forgetting

- c. Mixing up medications
 - d. Lack of desire
 - e. Illness
 - f. None of the above
11. Have you gained weight after starting Coumadin? Yes/No
12. If yes, approximately how much weight have you gained? _____
13. If yes, why do you think you gained the weight? (Circle all that apply)
- a. Changed diet and avoided vegetables
 - b. Exercised less
 - c. Ate more at each meal
 - d. Craved new foods that were less healthy
14. Have you lost weight after starting Coumadin? Yes/No
15. If yes, approximately how much weight have you lost? _____
16. If yes, why do you think you lost the weight? (Circle all that apply)
- a. Changed diet and avoided many foods
 - b. Illness
 - c. Ate less at each meal
 - d. Stopped drinking alcohol
17. What is considered a normal INR (blood Coumadin level)?
- a. Less than 1
 - b. 2-3
 - c. 4-5
 - d. Greater than 5
 - e. Not sure
18. Do you know what your current INR (blood Coumadin level) is? Yes/No
19. Approximately how often do you get your INR (blood Coumadin level) checked?
- a. Once a week

- b. Twice a month
- c. Once a month
- d. Twice a year
- e. Once a year
- d. Never
- f. Not sure

20. Do you ever not get your INR (blood Coumadin level) checked because of cost? Yes/No

21. Are you aware that your other medications can interact with Coumadin? Yes/No

22. Do you ask your pharmacist before starting a new medication if it interacts with Coumadin? Yes/No

23. Do you ever take over-the-counter pain medications? Yes/No

24. If yes, which ones? (Circle all that apply)

Excedrin® Tylenol® (Acetaminophen) Aleve® (Naproxen)

Advil® (Ibuprofen) Motrin® (Ibuprofen) Aspirin

25. Do you ask your doctor before using over-the-counter pain medications? Yes/No

26. Do you ever take over-the-counter stomach remedies? Yes/No

27. If yes, which ones? (Circle all that apply)

Tagamet HB® (Cimetidine) Pepto Bismol® (Bismuth Subsalicylate)

Laxatives Stool Softeners

Alka-Seltzer®

28. Do you ask your doctor before using over-the-counter stomach remedies? Yes/No

29. Do you take vitamin supplements? Yes/No

30. If yes, which ones (Circle all that apply)

Multivitamin (Dose:)

Vitamin A (Dose:)

Vitamin E (Dose:)

Vitamin D (Dose:)

Vitamin C (Dose:)

31. Are you aware that vitamin supplements can interact with Coumadin? Yes/No
32. Do you ask your doctor before using a vitamin supplement if it interacts with Coumadin? Yes/No
33. Do you think getting enough Vitamin K is important?
- a. Yes
 - b. No
 - c. Not sure
34. What do you think Vitamin K does for us? (Circle all that apply)
- a. Improves eye sight
 - b. Strengthens bones
 - c. Improves the texture and softness of skin
 - d. Helps to form clots
 - e. It is an anti-oxidant to help the body
35. Do you take any herbal or natural medications or supplements? Yes/No
36. If yes, which ones? (Circle all that apply)
- | | | |
|--|--------------|----------------------|
| Garlic | Ginger | Glucosamine |
| Ginko Biloba | CoEnzyme Q10 | Green Tea |
| St. John's Wort | Flaxseed | Melatonin |
| Papaya Extract | Ginseng | Soy Protein Products |
| Fish oil supplements that contain EPA or DHA | | |
37. Are you aware that natural medications or supplements can interact with Coumadin? Yes/No
38. Do you ask your doctor before using a natural medication or supplement if it interacts with Coumadin? Yes/No
39. Do you know how to interpret a supplement facts label on natural medications or supplements? Yes/No
40. How often do you drink alcoholic beverages?
- a. Every day
 - b. 4-6 days a week
 - c. 2-3 days a week

- d. Once a week
- e. 2-3 times a month
- d. Once a month
- e. Less than once a month
- f. Never

41. How often do use tobacco products?

- a. Every day
- b. 4-6 days a week
- c. 2-3 days a week
- d. Once a week
- e. 2-3 times a month
- d. Once a month
- e. Less than once a month
- f. Never

42. Can changing your diet change your Coumadin dose?

- a. Yes
- b. No
- c. Not sure

43. How often do drink grapefruit juice or eat grapefruit?

- a. Every day
- b. 4-6 days a week
- c. 2-3 days a week
- d. Once a week
- e. 2-3 times a month
- d. Once a month
- e. Less than once a month
- f. Never

44. Are you aware that grapefruit and grapefruit juice interact with Coumadin?

- a. Yes
- b. No
- c. Not sure

45. Are you aware that you get Vitamin K from the foods you eat?

- a. Yes
- b. No
- c. Not sure

46. How much Vitamin K do the following foods contain? Please circle the amount:

Cooked broccoli (1 cup)	0-9 mcg	10-29 mcg	30-89 mcg	99-1200 mcg
Vegetable Oil (1 Tbsp)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Canned tuna in oil (3 oz)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Iceberg lettuce (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Cooked spinach (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Coleslaw (3/4 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Red grapes (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Green leaf lettuce (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Walnuts (14 halves)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Grapefruit juice (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Red wine (3.5 fl oz)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Olive Oil (1 tbsp)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg

Cooked asparagus (4 spears)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Raw celery (1 stalk)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Vanilla ice cream (1/2 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Avocado (3 oz)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
75% lean ground beef (3 oz)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Roasted chicken (1 drumstick)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Raw pineapple (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Cooked salmon (1/2 fillet)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Swiss cheese (1 oz)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
2% milk (1 cup)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Hard-boiled egg (1 large)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg
Chunky peanut butter (1 Tbsp)	0-9 mcg	10-24 mcg	25-99 mcg	100-499 mcg

47. Do you know how to interpret a nutrition facts label on food products? Yes/No

48. How many meals do you eat each day?

- a. One
- b. Two
- c. Three
- d. Four
- e. Five
- f. Less than one

g. More than 5

49. How many meals do you eat each day with Vitamin K?

a. One

b. Two

c. Three

d. Four

e. Five

f. Less than one

g. More than 5

h. Not sure

50. Is it important to watch how much Vitamin K you get each day when you are on Coumadin?

a. Yes

b. No

c. Not sure

51. Do you believe that taking Coumadin negatively influences your quality of life? Yes/No

52. If yes, why do you think Coumadin negatively influences your quality of life? (Circle all that apply)

a. Frequent blood draws

b. Don't get to eat your favorite foods

c. Diet is too restrictive

d. No longer drink alcohol or only occasionally

e. Worry about bleeding

f. Feel unwell or experience side effects on the medication