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Nursing Students' Knowledge of Alcohol - Interactive Medications

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Abstract

In 2018, nearly 57% of American adults reported drinking alcohol in the past month, and 41.5% of current drinkers reported taking alcohol-interactive (AI) medications. Consuming alcohol and medications concurrently may result in adverse effects. The purpose of this study was to determine the effectiveness of a one-hour lecture about AI medications in a class of undergraduate nursing students (N = 48) at a small Midwestern university. The Jarvis Nursing Knowledge of Alcohol-Interactive Medications survey was distributed on August 27, 2018, and again on November 19, 2018. A significant increase was found between pretest and posttest on correct identification of mechanism (27.47 ± 14.18 vs. 37.33 ± 16.60; t =3.15; p < .02). A significant increase was also found between pretest and posttest scores on the ability to discriminate a medication as AI or Non-AI (68.5% ± 6.3 vs 71.5% ± 6.5; t= 2.5; p <. 02). While scores increased significantly, students failed to consistently recognize the correct medication-alcohol interaction. A one-hour lecture emphasizing AI medications in the pre-licensure program enhanced students' knowledge; however, future research is needed to determine retention of AI medication knowledge.

Nursing Students' Knowledge of Alcohol - Interactive Medications

Alcohol is one of the most popular and potentially harmful drugs in the United States. More than 88,000 people die from alcohol related causes each year, making it the third highest cause of preventable death (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 2018). Of people aged 18 years or older, 86.4% reported drinking alcohol in their lifetime; 70.1% reported drinking in the past year; and 56.0% reported drinking in the past month (NIAAA, 2018). In 2015, 26.9 % of people ages 18 years or older reported binge drinking in the past month and 7% reported heavy alcohol use in the past month. Binge drinking is a pattern of drinking that brings blood alcohol concentration (BAC) levels above 0.08 g/dL. Binge drinking is defined as four drinks for women and five drinks for men in about two hours. Heavy drinking is defined as binge drinking on five or more days in the past month (NIAAA, 2018).

Alcohol-interactive (AI) medications are any medication with which alcohol interacts on a pharmacokinetic (movement through the body) or pharmacodynamic (interaction in cells) level to produce additive or antagonistic effects. Over a 12-year period (1999–2010), an average of 41.5% of current drinkers 20 years and older reported taking at least one AI prescription medication (Breslow et al., 2015). From 2011-2014, 48.9% of adults over 18 years of age used at least one prescription medication in the last 30 days (Centers for Disease Control [CDC], 2016). People age 65 years and older have a higher prevalence of taking multiple medications, and current drinkers, age 65 and older, have a higher prevalence of taking at least one AI prescription medication (Breslow et al., 2015; National Center for Health Statistics, 2014). In the U.S. people aged 65 years or older ,90.6% reported taking a prescription drug in the last 30 days (CDC, 2016). Due to age related changes, people aged 65 years and older metabolize alcohol more

slowly and have less body water compared with younger adults, the bioavailability of alcohol increases when consuming the same amount of alcohol as a younger adult (Weathermon & Crabb, 1999). Age-related changes in absorption, distribution, and metabolism of alcohol and medications, place people aged 65 and older at greater risk for adverse effects of combining alcohol and medications (Moore et al., 2007).

Central nervous system (CNS) agents are among the most common prescription medications used across all age groups in the United States. Through pharmacokinetic and pharmacodynamic interactions, alcohol interferes with the absorption, distribution, metabolism, and excretion of medications and can have additive or antagonistic effects, particularly in the CNS (Tanaka, 2003). Within the class of CNS agents, pain relievers were involved in 28.4% of the alcohol associated drug interaction emergency department (ED) visits, mainly opioids at 17.4% (Castle, Dong, Haughwout, & White, 2016). Anti-anxiety drugs, sedatives, and sleep medications were involved in 24.7% of the alcohol associated drug interaction ED visits, mainly benzodiazepines at 16.7% (Castle et al., 2016). Within the class of psychotherapeutic agents, antidepressants and antipsychotics were involved in 8.6% and 6.1%, respectively, of alcohol associated drug interaction ED visits (Castle et al., 2016)

Education on AI medications is a necessity for nurses, as evidenced by the rise of prescription medications and increased use of alcohol in the U.S. Nurses need to be educated on AI medication because a vital responsibility of nurses is to be vigilant about health risks and to provide health education. In undergraduate nursing schools only 56% of schools offer one to five hours of didactic alcohol education, and 30% of schools offer one to five clinical hours of

alcohol education (Mollica, Hyman, & Mann, 2011). The education that was identified by (Mollica, Hyman, & Mann, 2011) focused on alcohol withdrawal, abuse, and addiction.

As well as undergraduate students lacking AI medication knowledge, professional registered nurses also lack AI medication knowledge. In 2016, professional nurses working at a Midwestern Magnet-designated hospital correctly identified AI medication only 56.3% of the time (Serafico & Jarvis, 2017). A knowledge gap of AI medications was identified in practicing nurses; and therefore important that there is adequate education on AI medication content in undergraduate programs. Including AI medication teaching into the curriculum of undergraduate nursing programs will help future nurses with the critical knowledge they need to deliver safe care to patients. Therefore, the aims of this study are to:

1. Assess nursing students' baseline level of knowledge of AI medications.
2. Determine if a single lecture in the sophomore year pathophysiology and pharmacology course is effective in increasing knowledge on AI medication content.
3. Determine to what extent the Jarvis Nursing Knowledge of AI Medications (JaNKam) survey is reliable when administered to nursing students.

Background

Literature Review

From 2000-2018 no published studies addressing the level of undergraduate nursing students' knowledge on alcohol physiology or alcohol interactive medications were found. Literature was published regarding undergraduate nursing students' attitudes towards both alcohol abuse and caring for patients admitted for alcohol abuse, nursing students' knowledge on alcoholism, and nursing students' knowledge on intoxication.

Metabolism of Alcohol

Alcohol is absorbed primarily in two places in the human body: the stomach and the small intestines. Of those two locations, 20% of alcohol is absorbed by the stomach and 80% is absorbed by the intestines (Burcham & Rosenthal, 2018). Meals high in fat, carbohydrates, or protein are all equally effective in slowing stomach emptying (Cederbaum, 2012). The small intestine's alcohol absorption is independent of the presence of food (Burcham & Rosenthal, 2018). The small intestine, however, is dependent on gastric emptying time and as such the rate of alcohol absorption is limited by the presence or absence of food in the stomach. Milk is particularly effective at slowing gastric emptying, thus delaying alcohol absorption. Alcohol has minimal nutritional value, with a caloric value of 7kcal per gram (Cederbaum, 2012). Alcohol is different from other nutrients in that it is not stored, instead remaining in the body until excreted. (Cederbaum, 2012). Additionally, alcohol metabolism is not heavily influenced by hormones (insulin, glucagon, leptin, catecholamine and thyroid hormone), which is dissimilar to hormonal effects on other nutrients (Cederbaum, 2012).

Alcohol is a unique substance, in that many membranes are permeable to alcohol. Alcohol is both lipid and water soluble and thus is distributed to all body tissues and fluids quickly after drinking. The blood brain barrier is permeable to alcohol, which allows the drug to gain easy access to the CNS and exert its effects. The CNS levels rise rapidly because the brain receives a large proportion of the blood flow. Alcohol is a teratogen because the placenta is permeable to alcohol, which can lead to fetal abnormalities if alcohol is ingested during pregnancy.

Several factors, including alcohol usage, sex, and age alter the rate of alcohol metabolism. Alcohol is metabolized at a constant rate of 15 ml (0.5 oz) per hour, regardless of how much alcohol is ingested. The metabolism of alcohol is unique because despite rising plasma drug levels, the rate of the drug's breakdown is relatively unchanged. The breakdown rate equates to one standard drink an hour. A standard drink is defined as one standard can/bottle of beer, one 5 oz glass of wine, or one standard shot (30 mL of spirits). When used on a regular basis, alcohol induces liver drug metabolizing enzymes, which increase the rate of metabolism. Due to the hepatic enzyme induction, chronic drinkers can metabolize the drug faster than people who drink moderately (Cederbaum, 2012).

The activity of the metabolizing enzyme alcohol dehydrogenase (ADH) in the stomach differs between males and females. Women have a lesser degree of stomach ADH activity, thus decreasing the first pass metabolism (Weathermon & Crabb, 1999). The enzyme ADH catalyzes the oxidation of alcohol to aldehydes. The decreased stomach ADH results in decreased first pass effect and increased bioavailability, which partly explains why women achieve a higher BAC than men after the same quantity of drinks (Burcham & Rosenthal, 2018). The first pass effect is defined as the amount of alcohol that is metabolized by ADH before reaching systemic circulation. Furthermore, women have a smaller percentage of body water (45%) compared to men (about 55%) (Burcham & Rosenthal, 2018). This differing amount of water in fluid compartments means less dilution of alcohol, causing an increased concentration in the tissues and fluids in women, thereby increasing alcohol's effects (Burcham & Rosenthal, 2018). Age also affects alcohol metabolism. For example, the fetal liver eliminates alcohol poorly which in turn leads to fetal alcohol syndrome (Cederbaum, 2012). There is also a small decline in alcohol

elimination with older adults, potentially due to decreased liver mass, or decreased water content (Cederbaum, 2012).

The process of metabolism occurs in both the stomach and liver, and starts with the conversion of alcohol to acetaldehyde by ADH. Once acetaldehyde is formed, it is converted to acetate by aldehyde dehydrogenase (ALDH). Much of the acetate produced circulates to peripheral tissues where it is activated into acetyl CoA (Cederbaum, 2012). After acetate is activated into acetyl CoA, it then enters the citric acid cycle. Acetyl CoA is a molecule that participates in the citric acid cycle of energy production by delivering an acetyl group.

Alcohol is metabolized primarily by the enzyme alcohol dehydrogenase. The enzyme ADH functions by oxidizing endogenous (alcohols produced by metabolic processes) and exogenous (alcohol that is consumed) alcohols. The highest amount of ADH is found in the liver, with lesser amounts present in the gastrointestinal (GI) tract, kidneys, and nasal mucosa. As much as 90% of alcohol consumed is removed from the body via oxidation by the liver, with the remaining 10% of alcohol being directly removed via excretion in breath, sweat, and urine (Cederbaum, 2012). Alcohol oxidation is primarily limited by the maximum capacity of ADH.

Alcohol is metabolized to a lesser extent by the cytochrome P450 family of enzymes in the liver. Cytochrome P450s are a family of enzymes responsible for oxidation of steroids, fatty acids. The CYP2E1 is a P450 enzyme that has the highest activity for oxidizing alcohol to acetaldehyde (Cederbaum, 2012). As alcohol concentrations increase, CYP2E1enzymes increase their involvement in oxidation (Cederbaum, 2012). Importantly, some medications compete for metabolism by CYP2E1 enzymes and thus when consumed with alcohol, the metabolism of those medications is inhibited. The inhibition of the medication’s metabolism increases their half

life, which then results in the drinker experiencing increased sensitivity to the drugs and adverse effects of the medication. The CYP2E1 enzymes are increased in chronic drinkers, and thus, when alcohol is not present, medications metabolized by these enzymes will have decreased half-lives and as a result have less therapeutic effectiveness.

AI Medications

Alcohol is a CNS depressant. When alcohol is consumed in combination with other CNS depressants (eg., barbiturates, benzodiazepines, opioids), the effects are additive and the CNS depression is potentiated. Barbiturates increase cytochrome P450 activity in the liver and accelerate alcohol elimination from the blood (Weathermon & Crabb, 1999). An adverse effect of opioids is the respiratory depression from the action of these medications as Mu receptor agonists (Burchum & Rosenthal, 2018). Opioid medications act at opioid receptors in the medulla to suppress the cough reflex which may lead to the accumulation of secretions in the airway (Burchum & Rosenthal, 2018). Respiratory depression and suppression of the cough reflex is increased by concurrent use of other CNS depressants such as alcohol (Burchum & Rosenthal, 2018). Alcohol enhances the effects in the CNS of antihistamines such as drowsiness, sedation, and decreased motor skills (Weathermon & Crabb, 1999). The combination of alcohol and benzodiazepines alter the psychomotor skills associated with driving, primarily due to a potentiated effect on reaction and coordination. Short-acting benzodiazepines without active metabolites appear to cause less impairment than longer-acting benzodiazepines (Chan & Anderson, 2014).

Alcohol interacts with Histamine 2 (H2) receptor blockers (drugs that decrease gastric acid) by decreasing the first pass metabolism. The presence of H2 receptor blockers such as

cimetidine (Tagamet) or ranitidine (Zantac) inhibit stomach ADH activity (Cederbaum, 2012). Since stomach ADH activity is inhibited, blood concentrations of alcohol increase.

Some antibiotics such as isoniazid (Nydrazid, Landiazid) and erythromycin (Erythrocin) interact with alcohol. Erythromycin may increase alcohol absorption in the small intestines and therefore increase blood alcohol concentrations. Isoniazid can cause liver damage which can be exacerbated by alcohol consumption as well.

Alcohol and NSAIDS (non-steroidal anti-inflammatory drugs) can injure the gastrointestinal (GI) mucosa. Alcohol in combination with NSAIDS, including ibuprofen (Advil), can cause a significant increase in gastric bleeding. Aspirin (Bayer) has been shown to inhibit stomach ADH, therefore decreasing the first pass effect and increasing blood concentrations of alcohol (Cederbaum, 2012). Aspirin (Bayer) increases gastric emptying which leads to faster absorption by the small intestine (Weathermon & Crabb, 1999). Aspirin (Bayer) , indomethacin (Indocin) , and ibuprofen (Advil) and cause enhanced bleeding when taken in conjunction with alcohol (Weathermon & Crabb, 1999). Concurrent alcohol intake at any concentration increases the risk of upper gastrointestinal bleeding associated with aspirin. The likely explanation for this interaction includes the reversal of the inhibition of vascular prostaglandin synthesis by alcohol which promotes bleeding (Chan & Anderson, 2014).

Alcohol also interacts with anticoagulants, particularly warfarin. Warfarin (Coumadin) acts as an anticoagulant by decreasing production of clotting factors VII, IX, X, and prothrombin (Burchum & Rosenthal, 2018). Alcohol acts as an anticoagulant by decreasing platelet aggregation, decreasing levels of fibrinogen, and increasing levels of tissue plasminogen activator (clot dissolving enzyme) (Burchum & Rosenthal, 2018). The synergistic interaction

between warfarin (Coumadin) and alcohol results in higher levels of anticoagulation and thus creates a higher risk of hemorrhage. When alcohol is used on a regular basis, it induces hepatic drug metabolizing enzymes, thereby increasing the rate of its own metabolism and that of other drugs. Warfarin (Coumadin) is inactivated in the liver mainly by CYP2C9, and the 2C9 isoenzyme of cytochrome P450 (Burchum & Rosenthal, 2018). A person who chronically uses alcohol will have decreased anticoagulation effects of warfarin (Coumadin) due to the increased metabolism.

Acetaminophen (Tylenol) and alcohol interact and pose the risk of potentially fatal liver damage. There is evidence that two to four standard drinks a day in combination with a high dose of acetaminophen (Tylenol) can cause fatal liver damage; thus, 2000 mg of acetaminophen (Tylenol) a day is the recommended limit for people who simultaneously consume alcohol (Burchum & Rosenthal, 2018). Alcohol enhances acetaminophen (Tylenol) metabolism by CYP2E1 into a toxic byproduct potentially causing liver damage (Weathermon & Crabb, 1999). It is especially important to educate patients to read labels to see hidden doses of acetaminophen (Tyleonol) in medications combined with codeine and oxycodone (Percocet).

Alcohol elevates blood pressure when used chronically and thus tends to counteract the effects of antihypertensive medications. When alcohol was given to 10 healthy male subjects after they received multiple doses of the antihypertensive verapamil (Calan), significantly higher blood alcohol levels were detected (Chan & Anderson, 2014). Verapamil (Calan) also increased the subjects' perceptions of alcohol intoxication. These are likely the result of inhibition of CYP2E1 (Chan & Anderson, 2014).

Alcohol and the anti- diabetic medication metformin (Glucophage) interact. Metformin inhibits the mitochondrial oxidation of lactic acid and thereby can result in the adverse effect of lactic acidosis (Burchum & Rosenthal, 2018). Metformin (Glucophage) may cause an increase in lactic acid in the blood after alcohol consumption.

Methods

Sample

All 50 second-year nursing students at a Midwestern liberal arts university was asked to participate in this study. Of the 50 students that were asked to participate, the final sample utilized for data analysis was 48 matched surveys. The eligibility requirement for this study was current enrollment as second-year nursing student in the N217 Pathophysiology and Pharmacology course. The study was conducted during the students' N217 Pathophysiology and Pharmacology lecture time. IRB approval was granted from the Illinois Wesleyan University IRB. Consent was conferred upon completion of the survey (Appendix A).

Data Collection

Data collection occurred at two different points. The data collection consisted of one pretest and one posttest. The pretest occurred on August 27th, 2018, the first day of class and before the students had received any pharmacology content. The AI medications lecture was delivered on November 19, 2018. The posttest was administered December 10, 2018, 21 days after the AI medication lecture. Forty-eight students responded to the survey, resulting in a 96% response rate. No students were excluded in this study. The final sample size was 48 students.

Pedagogical approach to AI medications

Given the rise in use of AI medications and the lack of professional nurses' knowledge of AI medications (Serafico & Jarvis, 2017), one approach to deliver AI content to undergraduate nursing students was explored. A single traditional one-hour lecture was delivered to the N217 Pathophysiology and Pharmacology class using a power point format. The lecture was delivered by the student researcher. The student researcher was a senior level nursing major who had successfully completed the N217 course. The lecture was rehearsed three times and reviewed by the student researcher's faculty advisor. The lecture contained information on the metabolism of alcohol as well as information on the interactions that alcohol has with several medications. The lecture included AI medications that were both on the Jarvis Nursing Knowledge of AI Medications (JaNKAM) instrument and medications that were not tested on the JaNKAM. The lecture lasted 45 minutes and there were five minutes after to answer questions. The PowerPoint slides utilized in the AI medication lecture can be found in Appendix B.

Instrument

Knowledge of AI medications was measured by using the JaNKAM Survey, which was developed in 2016 (Appendix C). The survey includes questions on 14 of the 25 most commonly prescribed medications in the United States, using data from the Centers for Medicare and Medicaid Services, the Institute for Healthcare Informatics, and the Centers for Disease Control and Prevention (CDC, 2016). There are 14 questions, and for each question there are five potential answers. Each survey question asks the participant to select the correct interaction of alcohol and a selected medication from a multiple choice listing. The multiple choices include four potential mechanisms alcohol could interact with the given medication as well as a choice

that alcohol and the given medication do not interact. The survey includes both the generic and the brand names of each drug. The JaNKAM survey tool utilized in this study was changed from the tool utilized in Serafico and Jarvis, 2017. The 2017 tool had an alpha of ($\alpha = 0.628$).

Data Analysis

Data analysis was conducted using IBM SPSS Statistics (version 25). Descriptive statistics were used to describe demographic characteristics. Inferential statistics were used to score the accuracy of participants' answers to the JaNKAM survey. Data were analyzed with mean, standard deviation, and paired t-tests to compare means between pre and posttests. Data were analyzed by two methods. The first method was the correct discrimination of a medication as AI or non AI. The second method was the analysis of the correct identification of AI mechanism of interaction. A paired t-test was utilized to evaluate the difference in means between the pretest and the posttest.

Results

Demographics

The majority of participants in the sample were female (n = 43, 89.6%). The age distribution ranged from 18-21 years old (M=19.3; SD= 0.56). Most participants identified as Caucasian (n=35, 73%). The students had a variety of previous medically related experiences. The majority of previous experience was cardiopulmonary resuscitation certification (n= 21, 43.8%). There were 16 missing responses to the question on previous medially related experiences; thus demographic data is incomplete for that question. Complete demographic data can be found in Appendix D**.**

Nursing Students Knowledge of AI Medications

The first aim of this study was to identify nursing students' knowledge of AI medications. The measure of knowledge was determined by the number of correct answers on the pretest. Knowledge was assessed by two methods. The first method was the correct discrimination if the medication was AI or non-AI. The mean score of correctly identifying medications as AI or non-AI was 68.5%, SD= 6.3, range 50%-79%. The most correctly identified medication was hydrocodone (Vicodin) (100%). The least correctly identified medication was simvastatin (Zocor) (8.2%). The second method was the correct identification of the mechanism of interaction if the medication was AI. The mean pretest score for correct identification of the mechanism was 27.5%, SD=14.2, range 0% to 62%. The most correctly identified medication for correct AI mechanism of interaction was Ibuprofen (Advil) (49%). The least correctly identified medications were prednisone (Deltasone) (4.1%) and simvastatin (Zocor) (8.2%), both of which are not AI medications.

Efficacy of a Single Lecture of AI Medication Content

The second aim of this study was to identify if a single one-hour lecture was sufficient to educate undergraduate nursing students on AI medication content. The measure of AI medication content knowledge was determined by the number of correct answers on the posttest. The posttest was evaluated by two methods.

The first method was the ability to discriminate if the medication was AI or non-AI. The mean score of correctly identifying medications as AI or non AI was 71.6%, SD=6.6, range 57%-86%. The most correctly identified medications were furosemide (100%), metoprolol (Lopressor) (100%), and gabapentin (Neurontin) (100%), all of which are AI medications.

Hydrocodone (Vicodin) decreased from the pretest score to posttest score (100% vs. 94%; t=1.78; p>.05). The least correctly identified medication was prednisone (2.1%). Simvastatin (Zocor) increased from pretest score to posttest score (8.2% vs 18.6%; t=1.53; p>.05). A significant increase was found between pretest and posttest scores for several medications. Omeprazole (Prilosec) increased 21.2% (14.3% vs 35.4%; t=2.34; p< .05), furosemide (Lasix) increased 16.3% (83.3% vs 100%; t=3.07; p<.02), and metoprolol (Lopressor) increased 8.2% (91.8% vs 100%; t=2.07; p<.05). A comparison between pretest and posttest correct discrimination of AI vs Non AI medication can be found in Table 3 (Appendix E).

The second method was the percentage of correct responses on the identification of the correct mechanism of interaction for an AI medication. The mean posttest score for identification of the correct mechanism was 37.3%, SD= 16.6, range 7% to 71%. The most correctly identified medication was hydrochlorothiazide (Aquazide H, Hydrocot, Microzide, Zide) (70.8%). Correct identification of ibuprofen (Advil) improved from pretest score to posttest score (49% vs 69%; t =2.34; p<.02). The least correctly identified medication was prednisone (Deltasone) which decreased from pretest score to posttest (4.1% vs 2.1%; t=.057; p> .05), Simvastatin (Zocor) improved from pretest score to posttest score (8.2% vs 18.8%; t=1.53; p>.05). A significant increase was found in several medications pretest to posttest score. Metoprolol (Lopressor) had 44% increase (18.4% vs 62.5%; t=5.23; p < .02), ibuprofen (Advil) had a 19% increase (50% vs 69%; t= 2.34; p< .02), and hydrochlorothiazide (Aquazide H, Hydrocot, Microzide, Zide) had a 38.1% increase (32.7% vs 70.8%; t= 3.86; p <.02). Levothyroxine (Synthroid) had a significant decrease from pretest to posttest (31.3% vs 6.30%; t=3.86; p< .02). A comparison of pretest and

posttest scores for each individual medication on the correct identification of the mechanism of interaction can be found in Table 2 (Appendix E).

A significant increase was found between pretest and posttest mean score on correct identification of mechanism (27.47 ± 14.18 vs 37.33 ± 16.60; t =3.15; $p < .02$). A significant increase was also found between pretest and posttest mean scores on the ability to discriminate a medication as AI or Non-AI (68.5% ± 6.3 vs 71.5% ± 6.5; t= 2.5; $p <. 02$). A comparison of pretest and posttest mean scores can be found in Table 4 (Appendix E).

Reliability of the JaNKAM

The third aim of this study was to determine the reliability of the JaNKAM AI Medication Survey tool. To determine the reliability of the JaNKAM, internal consistency was measured using Cronbach's alpha. Internal consistency is the degree to which all items on a scale measure the same concept (Polit & Beck, 2013). Cronbach's alpha is a reliability index that is used for nominal data that are scored dichotomously (Waltz, Stickland, & Lenz, 2010). Cronbach's alpha is appropriate for the JaNKAM because the survey was scored dichotomously in a correct/incorrect fashion. The JaNKAM survey demonstrated a low level of reliability (α = 0.549) on the posttest.

Discussion

This study examined nursing students' knowledge of AI medications and if a single one-hour lecture would be sufficient to increase knowledge. The findings demonstrated that a single one-hour lecture was effective in increasing knowledge. A significant increase was observed in both the correct identification of the mechanism of interaction and correct discrimination of AI

vs non AI. The students' ability to identify the correct mechanism was, however, lower than their ability to correctly discriminate if a medication was AI or non AI.

Both alcohol and AI medications are common in today's society, with 56% of the US population 18 and older drinking alcohol on a monthly basis and 41.5% of that population also taking an AI medication (Breslow et al., 2015). Older adults are particularly at risk for AI medications and alcohol interactions as 16.2 million adults aged 65 or older reported that they drank alcohol in the past month (Mattson, Lipari, Hays& Horn, 2017). In the US 90.6% of adults aged 65 and older reported taking a prescription drug in the last 30 days. Patient safety and patient education are both key components of a nurse's job description and therefore nurses need AI medication knowledge in order to do their job effectively. Practicing nurses need both the ability to discriminate a medication as AI or non AI, and the ability to correctly identify the mechanism. To safely provide patient care and education, professional nurses need knowledge of the specific mechanism of interaction and not just possess the ability to discriminate an AI medication vs a non AI medication. Patients need to be educated on the specific mechanism of action so that the patient can report to a healthcare provider if he/she experiences adverse effects. A patient will not know whether they are experiencing an adverse effect of alcohol and the medication interacting or if it is an effect of just the medication, without proper education by a nurse.

To the researchers' knowledge, no other comparable studies have been conducted in undergraduate nursing programs to assess nursing students' knowledge of AI medications. Overall, undergraduate nursing curricula is deficient in AI medication content. The reported curricula focus on alcohol abuse, addiction and withdrawal (Mollica, Hyman, & Mann, 2011).

Thus, this study is the first to focus on assessment and improvement of undergraduate nursing students' knowledge of AI medications.

The implications of these findings inform nursing schools' curricula decisions. The findings show that sophomore nursing students are able to learn AI medication content from a single one-hour traditional lecture. Based on the pretest vs posttest scores, the students' knowledge was greater when it came to discriminating between AI vs non AI than correctly identifying a mechanism of interaction. These findings inform curricular decisions by demonstrating a need for increased emphasis in education on the mechanism of interaction of AI medication. Undergraduate nursing schools could use these findings to make an informed decision on inclusion of AI medication content into the sophomore year curriculum. Undergraduate nursing schools can also utilize these findings to evaluate the effectiveness of a single traditional one-hour lecture as the method of delivery for AI medication content or if another method of instruction could potentially be more beneficial for their specific institution.

Limitations

The convenience sample (n=48) was taken from 50 undergraduate sophomore nursing majors in a Pathophysiology and Pharmacology course enrolled at a small Midwestern liberal arts university. The sample is representative of the study body, at this School of Nursing. Both the university demographic profile and the single-site convenience sample of students decreases the generalizability of the findings to the entire United States undergraduate nursing student population. The survey had a lower level of reliability (a=0.549), and due to this lower reliability the findings from this study need to be interpreted with caution. The lower reliability

of the survey used in this study compared to the survey used in Serafico and Jarvis, (2017) could be partially attributable to the smaller amount of questions (14) (Tavakol & Dennick, 2011).

The traditional one-hour lecture on AI medications was taught by the senior level student researcher. The student researcher had no prior training in education methods. The undergraduate nursing program that the student researcher attended prepared students to be generalist nurses not nurse educators. The student researcher had no prior experience delivering a lecture for 50 minutes. Due to the lack of experience of the student researcher in education, the students may not have learned the AI medication content thoroughly. The experience of the student researcher in lecturing is a potential limitation in the study. It cannot be determined without a true experimental design to what degree the lecture delivered by the student researcher affected the scores of the JaNKAM.

Certain medications were taught in the N217 Pathophysiology and Pharmacology curriculum, by the professor in the time between the pretest and posttest. The following medications were taught in between the pretest and posttest: simvastatin (Zocor), hydrochlorothiazide (Aquazide H,Hydrocot, Microzide, Zide), ibuprofen (Advil), Lisinopril (Prinivil, Zestril), amlodipine (Norvasc), furosemide (Lasix), metoprolol (Lopressor), and losartan (Cozzar). The students improved on their ability to discriminate AI vs non AI on the following meds: hydrochlorothiazide (Aquazide H, Hydrocot, Microzide, Zide), ibuprofen (Advil), Lisinopril (Prinivil, Zestril), amlodipine (Norvasc), furosemide (Lasix), metoprolol (Lopressor), and losartan (Cozzar). The students also improved on their ability to identify the correct mechanism of an AI medication for the following meds: simvastatin (Zocor), hydrochlorothiazide (Aquazide H, Hydrocot, Microzide, Zide), ibuprofen (Advil), Lisinopril

(Prinivl, Zestril), furosemide (Lasix), metoprolol (Lopresor), and losartan (Cozzar). The following medications are on the JaNKAM survey, but are taught in spring N218 Pathophysiology and Pharmacology II not in N217 in the fall: hydrocodone (Vicodin), omeprazole (Prilosec), levothyroxine (Synthroid), prednisone (Deltasone), metformin (Glucophage), and gabapentin (Neurontin). There was no way to determine if the increase in JaNKAM scores was a result of the AI medication lecture alone or if the content delivered in N217 Pathophysiology and Pharmacology contributed to the increase. This study was not constructed in a true experimental design and therefore that difference is cannot be assessed. The knowledge gained in N217 independent of the AI medication lecture could have positively affected the students' scores on those medications. It cannot be determined if the increase in ability to discriminate AI vs non AI and the correct identification of the mechanism was due to the AI medication lecture, the information presented by the N217 professor, or a combination of both.

Future Research

The students' knowledge needs to be assessed longitudinally to determine if retention of the knowledge has been achieved. The same posttest administered in this study will be administered at the end of the students' sophomore year (April,2019), in order to assess retention. In future research the identical posttest could be administered during the students' junior year and senior year. Effectiveness on the basis of retention of the AI medication lecture may be determined based on the posttest scores at each interval. The current findings, due to the sample demographics have limited generalizability. To increase the generalizability of the findings, other undergraduate nursing programs could be invited to participate in future studies.

To address the lower reliability of the JaNKAM survey ($\alpha = 0.549$), review of each question will be undertaken to assess for phrases or words that may mislead participants.

Conclusion

A key role of nurses is to be vigilant of health risks, such as AI medications, and provide health education accordingly, as defined by *The Essentials of Baccalaureate Education for Professional Nursing Practice* (American Association of Colleges of Nursing, 2008). Current undergraduate nursing curricula may be deficient in AI medication content. Nursing students, however, are capable of learning AI medication content, thus preparing them to be safe and effective practicing nurses. The results of this study should be interpreted with caution due to the limited generalizability due to the small sample size that is non representative of all undergraduate nursing students in the United States. Although the results should be interpreted with caution, undergraduate nursing programs could utilize these findings and data from future studies to make informed curricular decisions. These findings can be utilized by undergraduate nursing schools to determine if and how they want to incorporate AI medication content into their curricula.

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Appendix A

Invitation Asking Participants to Complete Survey & Consent

Jarvis Survey on Alcohol Interactive Medications

Please participate in a research study on knowledge of prescription medications and alcohol drinking. Your responses are important in helping us plan education for students. This study is conducted by Dr. Carolyn Jarvis from Illinois Wesleyan University.

This study will take about 15 minutes of your time. You will be asked a few basic demographic questions, and questions about specific commonly prescribed medications and their interaction with alcohol.

Your decision to participate in this study is completely voluntary and does not count toward you course grade. Please answer all the questions. If you do not know an answer, give it your best guess.

Your participation in this research will be completely confidential and data will be averaged and reported in aggregate. Possible outlets of these data may be conferences and publication in peer-reviewed journals. Although your participation in this research may not benefit you personally, it will help us very much in planning nursing education regarding alcohol-interactive medications and patient safety.

The primary risk in this study is loss of confidentiality. But, because no identifiable data are being kept, the risk to loss of confidentiality is minimal. All information will be locked and remain confidential.

If you have questions about this project, you may contact Carolyn Jarvis, PhD, APRN (309-556-3297; cjarvis@iwu.edu). If you have any questions about your rights as a participant in this study or any concerns or complaints, please contact the Illinois Wesleyan University IRB at 309-556-3255 or via email at irb@iwu.edu.

By completing this survey, you are consenting to participate in this study. Please ask for a print copy of this consent form for your records, if you wish.

Your decision to participate will have no effect on your current status or future relations with Illinois Wesleyan University. By completing this survey, you are consenting to participate in this study.

Appendix B

AI Medications Lecture PowerPoint Slides

Pharmacodynamics/Pharmacokinetics of Alcohol and Alcohol Interactive (AI) Medications

Andrew Coop

Alcohol Metabolism

- Constant rate metabolism
- Rate = 15ml per hour or 1 standard drink
- Gastric ADH = Alcohol Dehydrogenase
- Woman = Less Alcohol Dehydrogenase

Pharmacokinetics of Alcohol

- 20% of by the stomach
- 80% by small intestines
- Gastric emptying is rate limiting
- Alcohol= 7kcal per gram
- Lipid and water soluble
- Women= Less body water percentage

Alcohol Metabolism

- Constant rate metabolism
- Rate = 15ml per hour or 1 standard drink
- Gastric ADH = Alcohol Dehydrogenase
- Woman = Less Alcohol Dehydrogenase

Cytochrome P450 metabolism

- Cytochrome P450s = oxidation of steroids, fatty acids, medications
- Chronic drinker ➡ Increased enzyme activity ➡ Increased drug metabolism
- Medications Compete for Cytochrome P450 metabolism
- Increased Medication Half lives

Central Nervous System (CNS)

- Alcohol = CNS depressant
- GABA, Serotonin (5-hT_3) , and Glutamate receptors targeted
- Sedation, relief of anxiety,slurred speech, ataxia, impaired judgment, and disinhibited behavior
- Chronic drinkers develop tolerance
- Sleep aid?

Cardiovascular

- Dilation of cutaneous blood vessels
- Negative inotropic effect
- Atrial fibrillation
- Acute vs Chronic effect on BP
- Dose dependent elevation of blood pressure
- Raise HDL

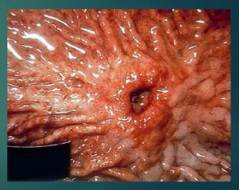
Blood Pressure Category	Systolic mm Hg (upper #)		Diastolic mm Hg (lower #)
Normal	less than 120	and	less than 80
Prehypertension	120 – 139	or	80 – 89
High Blood Pressure (Hypertension) Stage 1	140 – 159	or	90 – 99
High Blood Pressure (Hypertension) Stage 2	160 or higher	or	100 or higher
Hypertensive Crisis (Emergency care needed)	Higher than 180	or	Higher than 110

Liver

- Accumulation of fat and protein
- Cirrhosis

Stomach

- Stimulation of hydrochloric acid secretion
- Erosive gastritis→ Gastric Ulcer



Kidney

- Inhibits ADH- Anti Diuretic Hormone
- Decreased water reabsorption
- Increased urine formation

Alcohol Effects

Alcohol suppresses ADH production by the pituitary

⬇

Without ADH, higher amounts of water stay in the urine

⬇

Urine with high concentrations of water leaves the body

Pancreas and Glucose Metabolism

- 35% of cases of acute pancreatitis
- Suppresses gluconeogenesis
- Blunts after eating rise in blood glucose
- Lowers fasting glucose and insulin
- Increase HDL

Tolerance and Physical Dependence

- Tolerance is both Pharmacokinetic and Pharmacodynamic
- Cross tolerance
- Cross Dependence

CNS Depressants

- Includes Opioids, Benzodiazepines
- Opioids reduce the cough reflex and breathing functions
- Antihistamine CNS effects
- Benzodiazepines alter psychomotor skills

H2 Receptor Blockers

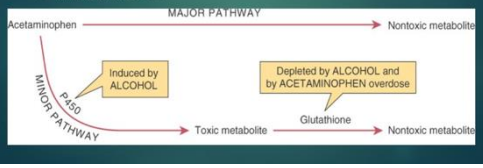
- Metidine and Ranitidine
- Decrease first pass metabolism
- Gastric ADH activity is inhibited

NSAIDs

- Aspirin, Ibuprofen, Naproxen
- Gastric Bleeding
- Aspirin inhibits ADH
- Gastric emptying

Acetaminophen

- Liver damage
- 2,000mg limit
- Inhibits gastric ADH



Diuretics

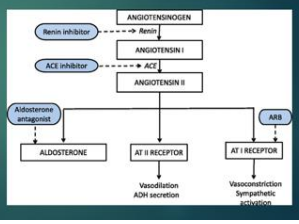
- Hydrochlorothiazide, Furosemide
- Electrolyte loss
- Dehydration
- Hypotension

Anticoagulants

- Heparin, Warfarin
- Acute vs Chronic ingestion

Anti- Hypertensives

- Beta Blocker
- Calcium Channel Blockers
- Ace Inhibitors
- ARBs
- Acute effects of alcohol on BP
- Orthostatic Hypotension



Anticoagulants

- Heparin, Warfarin
- Acute vs Chronic ingestion

Appendix C

Survey Tool: Jarvis Nursing Knowledge of AI Medications (JaNKAM) Survey

1. Please fill in your student ID number

2. What gender?
   - Female
   - Male
   - Transgender

3. Age in years

4. Have you had any of the following
   - CAN
   - CPR
   - EMT
   - Phlebotomist
   - Other medical experience
5. With what ethnic origin do you affiliate?
   - African American
   - Asian
   - Caucasian
   - Latino/a
   - Other

Each question below will identify a common medication given in the hospital or outpatient setting. Assume the patient drinks 1-2 standard alcohol drinks per day. Please look at each medication and answer the way it interacts with alcohol. Please do not use any device or book references as you answer these questions. We just want to learn a baseline for nursing education. It is OK if you do not know an answer.

6. How do Hydrocodone/Acetaminophen (Lortab, Vicodin, Norco) and alcohol interact?
   - Both substances cause respiratory depression and sleepiness
   - Both substances cause high blood pressure crisis
   - Both substances cause kidney toxicity
   - Alcohol decreases pain relief of hydrocodone/acetaminophen
   - Hydrocodone/acetaminophen and alcohol do not interact

7. How do simvastatin (Zocor) and alcohol interact?
   - Both substances increase risk of heart attack

- Both substances increase risk of diabetes
- Alcohol causes high blood cholesterol and cancels statin benefits
- Both substances increase risk of myopathy
- Simvastatin and alcohol do not interact

8. How does hydrochlorothiazide (HCTZ, Hydrocot) and alcohol interact?
    - Both substances increase risk of peripheral edema
    - Both substances increase risk of hearing loss
    - Both substances increase risk of enlarged breasts in men and menstrual irregularities in women
    - Both substances decrease blood pressure
    - Hydrochlorothiazide and alcohol do not interact

9. How do omeprazole (Prilosec) and alcohol interact?
    - Both substances increase risk of pneumonia
    - Both substances increase risk of osteoporosis and fractures
    - Both substances increase risk of intestinal infection with Clostridium difficile
    - Alcohol increases risk of acid rebound when stopping omeprazole
    - Omeprazole and alcohol do not interact

10. How do ibuprophen (Advil) and alcohol interact?
    - Both substances Increase risk of edema
    - Both substances Increase risk for bleeding
    - Both substances Increase risk of kidney toxicity
    - Both substances increase risk of high blood potassium
    - Ibuprophen and alcohol do not interact

11. How do levothyroxine Levoxyl, Synthroid) and alcohol interact?
    - Alcohol reduces levothyroxine absorption
    - Alcohol increases levothyroxine metabolism
    - Both substances increase risk of myxedema coma
    - Both substances increase risk of simple goiter
    - Levothyroxine and alcohol do not interact

12. How do lisinopril (Zestril, Prinivil) and alcohol interact?
    - Both substances increase risk of edema of tongue, lips or eyelids
    - Both substances decrease blood pressure
    - Both substances increase risk of cough
    - Both substances increase risk of high blood potassium
    - Lisinopril and alcohol do not interact

13. How do amlodipine (Norvasc) and alcohol interact?
    - Both substances increase risk of headache
    - Both substances increase risk of peripheral edema
    - Both substances increase risk of constipation
    - Both substances decreased blood pressure
    - Amlodipine and alcohol do not interact

14. How do furosemide (Lasix) and alcohol interact?
    - Both substances decrease blood pressure
    - Both substances increase risk of potassium loss
    - Both substances increase risk of hearing loss
    - Alcohol stops the diuretic effect of furosemide
    - Furosemide and alcohol do not interact

15. How do prednisone and alcohol interact?
    - Both substances suppress the adrenal glands
    - Both substances increase risk of fungal infection
    - Both substances increase physiologic stress
    - Alcohol speeds up glucocorticoid withdrawal
    - Prednisone and alcohol do not interact

16. How do metformin (Glucophage) and alcohol interact?
    - Both substances decrease appetite and cause nausea and vomiting
    - Both substances cause weight gain
    - Both substances allow buildup of lactic acid in the blood
    - Both substances increase risk of acute kidney failure
    - Metformin and alcohol do not interact

17. How do gabapentin (Neurontin) and alcohol interact?
    - Both substances cause dizziness and sleepiness
    - Both substances increase risk for headache
    - Both substances increase risk for peripheral edema
    - Both substances cause blurred vision
    - Gabapentin and alcohol do not interact

18. How do metoprolol (Lopressor) and alcohol interact?
    - Both substances increase risk of hyperthyroidism
    - Both substances cause constriction of breathing tubes
    - Both substances decrease blood pressure
    - Both substances increase the risk of migraine headaches
    - Metoprolol and alcohol do not interact

19. How do losartan (Cozaar) and alcohol interact
    - Both substances cause swelling of the lips, tongue or eyelids
    - Both substances decrease blood pressure
    - Both substances cause kidney failure
    - Both substances increase levels of potassium in the blood
    - Losartan and alcohol do not interact

Appendix D

Table 1: Demographic Characteristics of Participants of Posttest (Sex, Age, Ethnicity, Prior Experience)

Variable	% of total sample
Sex	
Male	10.40
Female	89.60
Ethnicity	
African American	6.30
Asian	6.30
Caucasian	72.90
Latino/a	29.20
Age	
18	2.10
19	75.00
20	18.80
21	4.20
Prior Experience	
CNA	14.5
CPR	52.1
EMT	2.0
Phlebotomist	2.0
Missing data	29.2

Appendix E

Table 2: Comparison of Pretest and Posttest Correct ID of Mechanism of Interaction

Medication	Pretest	Posttest	t(p)
amlodipine	24.5%	24%	0.26(0.80)
furosemide	14.6%	31.6%	1.8(0.07)
gabapentin	35.4%	50.0%	1.5(0.13)
hydrochlorothiazide	32.7%	70.8%	3.9(0.0003)**
hydrocodone	34.7%	50.0%	1.4(0.16)
ibuprofen	50.0%	69.0%	2.3(0.03)*
lisinopril	34.7%	42.6%	0.62(0.54)
levothyroxine	31.3%	6.30%	3.3(0.002)**
losartan	20.4%	29.2%	0.94(0.35)
metformin	20.4%	34.0%	1.4(0.16)
metoprolol	18.4%	62.5%	5.2(0.0001)**
omeprazole	36.7%	31.3%	0.55(0.58)
prednisone	4.1%	2.1%	0.57(0.57)
simvastatin	8.2%	18.8%	1.5(0.13)

*$p < .05$

**$p < .02$

Table 3: Comparison of pretest and Posttest discrimination of AI vs Non AI score

Medication	**Pretest**	**Posttest**	**t(p)**
amlodipine	91.8%	93.5%	0.37(0.71)
furosemide	83.3%	100%	3.07(0.004)**
gabapentin	97.9%	100%	1.00(0.32)
hydrochlorothiazide	87.8%	95.8%	1.66(0.10)
hydrocodone	100%	93.8%	1.77(0.08)
ibuprofen	83.7%	89.6%	0.90(0.37)
lisinopril	95.9%	93.6%	0.44(0.66)
levothyroxine	6.3%	6.3%	0.00(1.00)
losartan	95.9%	83.3%	2.21(0.03)*
metformin	98.0%	89.4%	2.07(0.04)*
metoprolol	91.8%	100%	2.07(0.04)*
omeprazole	14.3%	35.4%	2.34(0.02)*
prednisone	4.1%	2.1%	0.57(.057)
simvastatin	8.2%	18.8%	1.53(0.13)

*$p < .05$

**$p < .02$

Table 4: Comparison of Pretest and Posttest JaNKAM score

Statistic	**Pretest**	**Posttest**	**t(p)**
AI or Non AI	68.5% ± 6.3	71.5% ± 6.5	2.5(0.02)*
Correct mechanism ID	27.3% ± 14.2	37.3% ± 16.6	3.15 (0.003) *

*p < .02