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FIRST NATIONS COMMUNITIES AND TOBACCO TAXATION: A COMMENTARY

Hasina Samji, B.A., Sc.M. and Dennis Wardman, M.D., F.R.C.P.C., M.C.M.

Abstract: Taxation of tobacco is a widely used strategy that promotes smoking cessation among adults and reduces cigarette consumption among continuing smokers. First Nations (FN) populations' tobacco use is estimated to be 2-3 times that of other Canadians and, in part, a reflection that tobacco products purchased on reserve by FN people are tax exempt. The purpose of this paper is to present a commentary related to the implementation and impact of a tobacco tax within an FN community.

INTRODUCTION

Tobacco use in Canada is estimated to result in the deaths of more than 47,000 people annually (Makomaski Illing & Kaiserman, 2004). Smoking is a major cause of chronic airway disease, lung cancer, ischemic heart disease, and stroke (Health Canada, 2005). Smoking has also been implicated as a risk factor in the development of diabetes (Houston et al., 2006).

Comprehensive public health strategies to prevent and control tobacco use include mass media campaigns, smoking cessation services, community awareness initiatives, smoke-free spaces, litigation, and taxation of tobacco products (U.S. Department of Health and Human Services [U.S. DHHS], 1999). Taxation of tobacco is a widely used strategy that prompts smoking cessation and reduces cigarette consumption (U.S. DHHS, 2000).

While national studies have demonstrated a decreasing prevalence of smoking—50% of Canadians aged 15 years or older smoked in 1965, compared to 20% in 2004—these trends are not uniform across the nation and specific populations, such as First Nations peoples, continue to exhibit high rates of tobacco use. “First Nations” (FN) refers to those persons who are Registered Indians or Inuit as defined by the Indian Act, and whose names appear on the Indian Registry maintained by the Department of Indian and Northern Affairs. This designation does not include Métis people or non-Status Aboriginals. There are approximately 760,000 FN people in Canada; they are a young population, and an estimated 45% live off reserve (Indian and Northern Affairs Canada [INAC], 2007).

FN populations' tobacco use is estimated to be 2-3 times that of the Canadian smoking rate (First Nations Centre, 2005; Health Canada, 2006a). A significant difference is observed in the prevalence of smoking between on-reserve and off-reserve adults, with smoking rates of 58.5% and 36%, respectively (First Nations Centre). FN smokers consumed an average of 10 cigarettes per day, a lower consumption rate than for other Canadians, who smoked an average of 15 cigarettes per day. (First Nations Centre). Smoking is a major source of morbidity and mortality in FN populations: Adjusted smoking-attributable mortality rates are almost 1.5 times those of the general population, and smoking is responsible for almost one in every fifth FN adult death in British Columbia (BC) (Wardman & Khan, 2004). There are several possible reasons why rates in FN communities are higher (as well as why rates on reserve are higher), including the spiritual role that tobacco plays in many FN cultures, the role of family members who smoke, and the level of education achieved (First Nations Centre; French, 2000; Kegler, Cleaver, & Yazzie-Valencia, 2000). Another contributing factor pertains to the exemption of tobacco products purchased on reserve by FN people from taxation (Fiscal Realities, 1997).

HISTORICAL OVERVIEW OF TOBACCO PRICES IN FN COMMUNITIES

The final selling price for tobacco products includes the Federal excise duty, collected during manufacturing for Canadian products and at the time of importation for imported products; the provincial tobacco taxes, which are established by the finance ministries of provinces and territories; as well as the product price. Taxes per 200 cigarettes vary greatly by province and territory, with Quebec setting the tax at \$20.60 and the Northwest Territories at \$42.00 (Health Canada, 2006b). The Indian Act (section 87) states that personal property of a Registered Indian or a band situated on a reserve is exempt from taxation; thus, the only cost added to tobacco products purchased by an FN person on reserve is the Federal excise duty. Therefore, the average cost for a carton of cigarettes sold on reserve to an FN person is CAD\$39.74 (CAD\$23.87 + CAD\$15.87), which is 44% less than if purchased off reserve (Fiscal Realities, 1997; Smoking and Health Action Foundation, 2003).

In 1997, the Federal government passed Bill C-93, the Budget Implementation Act, which grants authority to FN communities to tax the sale of tobacco and tobacco products to Natives and non-Natives. This is otherwise known as the FN Tax (FNT) and is identical to the Federal Goods and Sales Tax (GST). The Bill also contains provisions that allow FN communities to impose a tax similar to that imposed under a provincial tobacco tax act (Fiscal Realities, 1997). The Westbank First Nation in BC was the first to implement the FNT in 1998, and 10 other nations have followed suit (Canada Revenue Agency [CRA], 2006).

Although Registered Indian communities now have the ability to collect tax on tobacco products and direct the use of these revenues, the strategy is underutilized—less than 2% of bands collect a tobacco tax (CRA, 2006). In order to further reduce smoking rates among FN populations, the Federal government has embarked on several initiatives to inform bands about the tax and to support FN communities in the area of tobacco taxation. A commentary of some of the initial efforts to implement the tax will be presented here.

BENEFITS OF TAXATION

Unfortunately, there is little or no published research on the effect of tobacco product taxation or price increases on tobacco utilization rates within FN populations. Among non-FN populations, it is estimated that a 10% price increase will decrease smoking rates by 4% (U.S. DHHS, 2000). In addition, a study evaluating the Federal impact of a tobacco tax identified a 1.12-pack per capita decrease for every 1-cent increase in tobacco tax (Meier et al, 1997). If we assume an inverse relationship between price and tobacco use among FN populations, then the implementation of an FNT (7%) may reduce tobacco use by 2.8% ($4\%/10\% \times 7\%$). Furthermore, if an FN community were to collect the provincial tax (mean of \$34.85 per carton of cigarettes), the price would increase 53.3% ($\$39.74/(\$39.74 + \$34.85)$), followed by a potential reduction in tobacco use of 22.5% ($4\%/10\% \times 56.3\%$; Ontario Tobacco Research Unit, 2007).

The other potential health benefit of a tobacco tax is that a reduction in the consumption of tobacco may be accompanied by a reduction in the misuse of alcohol and other drugs (Prochaska, Delucchi, & Hall, 2004). A smoking cessation intervention examined by the authors showed a 25% increase in the likelihood of abstinence from drugs and alcohol for participants receiving the intervention compared to the control group (Prochaska et al.). Given that the misuse of substances is a major concern for many FN communities, taxing tobacco products is another mechanism by which they can address this most urgent issue.

In addition to the enormous health benefits of tobacco use reduction, tobacco taxation can serve as a source of community revenues. Once again, there is a lack of data available to indicate the amount of revenue a tobacco tax would generate for FN communities. However, it is possible to estimate the amounts. The government generated CAD\$1.15 billion from tobacco taxation during 2006 in BC, and if we assume that FN people represent 3.7% of the population, with approximately half living off reserve, CAD\$21,275,000 would potentially be generated annually from FN tobacco taxes. This amount breaks down to CAD\$106,375 per community, as there are 200 bands in BC (Physicians for A Smoke-Free Canada, 2008; British Columbia Vital Statistics Agency & First

Nations and Inuit Health Branch, 2002; INAC, 2006). Assuming FN smoking rates are higher than those of the general BC population (i.e., up to 3 times higher), revenues may be even higher, perhaps up to \$196,020 per community. These estimates include the provincial tax and the GST.

OBSTACLES TO IMPLEMENTING THE TAX

In spite of the substantial benefits of tobacco taxation to FN communities, very few presently collect this tax. There are several possible reasons why this is so, including lack of awareness of the policy that allows FN communities to tax tobacco products; vendors' reluctance to support a tax due to loss of revenue; worry about the possibility of reduced Federal transfers, the end of statutory exemption for Status Indians, and barriers against using tobacco for traditional purposes; and backlash against the revenue being collected from populations where many are faced with poverty (Fiscal Realities, 1997). However, despite these potential barriers, there appears to be considerable FN community support for tobacco taxation—a tobacco use survey in 2005 revealed that 50% of FN people dwelling on reserve supported a tobacco tax in their community (unpublished data, First Nations and Inuit Health Branch, Pacific Region, 2005).

In addition to the obstacles listed above, another concern regarding the tax pertains to the potential increase in tobacco smuggling in response to the tax. For many FN communities, however, smuggling is probably minimal, as is the case with non-FN communities (U.S. DHHS, 2000).

EXPERIENCE LAUNCHING THE TAX IN AN FN COMMUNITY

Having provided a general overview of the history and potential benefits and obstacles of FN tobacco taxation, it will be beneficial to examine an actual scenario of a tobacco tax being implemented in an FN community. As mentioned previously, tobacco taxation is a contentious issue in FN communities, and those that collect a tax are often reluctant to discuss specifics with outsiders. For this reason, and to maintain the confidentiality of those communities, the scenario presented is primarily based on one community but also draws on the collective experiences of communities that presently tax tobacco products. This amalgamation of communities will be referred to as Community A for ease of understanding.

The community presented is, in many ways, a typical FN community. It is located in a rural setting and has a population of 500 people, with 50% living off reserve. The population is young, with 40% aged 19 years or younger. (INAC, 2006). Members of Community A have often expressed their concerns about a high rate of cancer and problems with alcohol and drugs in the community, but there are no data available to quantify cancer rates and level of substance use in the community.

A survey of Community A revealed that 55% of the population smokes and tobacco is sold at 3 vendors within the community. The community receives \$15,000 annually from Health Canada to support tobacco control activities and has designated a champion—an individual with substantial community support—as part of the strategy. The community's tobacco control strategy has focused on awareness, including the establishment of a tobacco information booth at the community health fair, where information related to health effects is provided; the development of marketing products (i.e., posters); presentations at the local school; and the sponsorship of tobacco walks (exercising while discussing the effects of tobacco use). There have also been efforts to include tobacco cessation advice within existing community programs, such as the prenatal program. Finally, band-operated buildings are smoke-free, although most other public spaces in the community are not. Because there are no staff exclusively hired to focus on tobacco control activities, such activities are often carried out by the tobacco control champion, who manages several other programs. Thus, tobacco programming in Community A often does not receive the resources and support it requires.

The regional First Nations and Inuit Health Program (FNIH) office has provided information related to tobacco taxation to bands in BC, as well as the opportunity to apply for one-time funding of \$5000 per community to support programming related to tobacco taxation. These funds can be used for supporting an initial community dialogue that explores tobacco taxation. The community champion from Community A was interested in the initiative and accessed the funds to support a community forum that would allow such a discussion.

The tobacco control champion, supported by the regional FNIH Program, arranged a community forum that included a traditional feast, a tobacco taxation presentation by a public health professional, and a performance by an Aboriginal hip-hop group, which included a song on the benefits of not smoking. Overall, the forum was well attended (approximately 100 people). A representative cross-section of the community that varied widely in age, and which included members of the elected leadership (Chief and Council), was present.

The presentation focused on the benefits and harms of tobacco taxation in an FN context, followed by a community dialogue session. Community members raised questions and concerns, which centered on how the tobacco tax revenues would be utilized, as well as the fact that raising the price of tobacco products might not reduce tobacco use. During a discussion in response to the first concern, participants decided to vote to determine the level of support for the tobacco tax, and to gather suggestions from community members on how the tax revenue should be utilized. Based on the vote, 94% of participants, who represented a majority of the community's on-reserve membership at that time, supported the tax. The following areas for funding were identified: youth, Elders,

housing, tobacco/addiction prevention activities, and an identified priority area that would change yearly. Taxation information resources were provided to community members to aid their decision making (CRA, 2006; U.S. DHHS, 1999; Fiscal Realities, 1997; Wardman & Khan, 2005).

After a short break, participants discussed the fact that addiction disorders (including tobacco addiction) are treatable, and that many persons overcome their addiction without any professional intervention, in part due to maturation (O'Brien & MacLellan, 1996; Frances & Miller, 1998; Vaillant, 1988). In response to the second issue that had been raised earlier (namely, that tobacco cost and use were not directly correlated), participants discussed studies from different cultures and socioeconomic groups that have shown consistently that a decrease in smoking follows an increase in the price of tobacco products (U.S. DHHS, 2000). Based on their comments, those present appeared to accept that a tax would reduce tobacco consumption and result in health benefits. In summary, the forum allowed information to be presented to community members and leadership to aid their decision making about the tobacco tax. The forum was deemed a success.

During the next phase, the tobacco control champion presented voting results and the identified target areas for funding from the community forum to Community A's elected leadership. The presentation was similar to that made at the community forum, and was also followed by a dialogue. Based on this discussion, the leadership decided that the funds remaining from the \$5,000 Health Canada taxation programming grant would be used to gather additional information by communicating with communities that already taxed tobacco products to identify (1) how they had advanced the implementation of the tax and (2) what the resulting implications for their communities were. A report based on this information would be presented to the Chief and Council before a final decision was made about implementing a tobacco tax in Community A.

Four months later, the report was completed and presented to the leadership. Generally, little information could be gleaned from the experience of other communities that collected the tax because they did not assess the tax's impact on smoking. As well, these communities often included the tobacco tax revenues in their general revenues without specifying which areas had been targeted. However, one community noted that they had used tax revenues to buy back traditional land. Based on the information gathered in the report and what is known about the health benefits related to tobacco taxation, the leadership voted to implement the tax. The leadership then met with the CRA to discuss collection of the tax. It was decided that vendors would collect the tax and submit the funds to the CRA, which would then forward the funds to the band. The leadership arranged for presentations on the tax to be made in leadership meetings and other appropriate venues, and included an article in the community newsletter related to the tax and how the revenue would be used. Approximately 3 months before implementation, notices were posted throughout the community and placed in the community newsletter.

Once the leadership made the announcement, there was a small, vocal group who raised concerns. They argued that more community discussions were needed as the tax could have major implications for the community, including the erosion of tax-exempt status and loss of Federal government funding. The leadership met with the group to discuss the matter; it was decided that the tax would be implemented for 1 year so that its impact could be assessed and this information shared with the community. A plebiscite would then be held, allowing community members to decide whether the tax would continue.

ONE YEAR LATER

The tax was implemented with generally positive feedback and some minor complaints. A Tobacco Prevention Coordinator was hired (see below) who made several presentations on the tax and what it meant to the community. During the year, the community newsletter carried messages to keep members abreast of progress in implementing the tax. Approximately \$200,000 was generated; this money was equally distributed to the five areas chosen in the initial community forum (i.e., each program area received \$40,000). The revenue was utilized as follows:

- *Youth:* Trips to sporting events and conferences, team uniforms and sporting equipment, partial support of a youth drop-in center, and a parent-youth support group (development of life skills, including parenting skills)
- *Elders:* A transportation program and a Meals for Elders group
- *Housing:* Partial support of a community home mold remediation program
- *Tobacco/Addiction Prevention Activities:* Hiring a Tobacco Prevention Coordinator to develop and implement prevention activities, including the creation of smoke-free spaces; enhancing Driving While Impaired (DWI) penalties (including the exploration of an alternative justice program for individuals convicted of DWI); and support of an alcohol server trainer program
- *Priority Area:* Investigation of the impact that a local industry plant is having upon traditional foods, including human health effects

Once the tax had been in place for a year, the Tobacco Prevention Coordinator spearheaded a community forum at which tax revenue and program information was presented to community members and leadership. In general, the majority of community members present agreed that the tax was beneficial and thought that it should continue. Following the forum, similar presentations were made by the Coordinator to elected leadership, with positive feedback. The information was also presented in the community newsletter, and the Coordinator disseminated copies of a report summarizing information about the tax revenue and its program implications throughout the community.

Once the presentations were complete and the community had opportunity to review the report, a plebiscite was held in which 60% of band members indicated that they supported the tobacco tax; thus, it remained in place. Occasionally there are complaints from community members who oppose the collection of the tax. Annual updates on the use of the revenues are provided in the community newsletter, at Chief and Council meetings, and via dissemination of an annual summary report. In summary, the majority of community members feel the tax benefits the community and continue to support its collection.

CONCLUSION

FN people smoke at a higher rate than other Canadians, in part because they are exempt from taxation of tobacco products purchased on reserve. Tobacco taxation has economic and health benefits but is underutilized by FN communities. A commentary on tobacco taxation is presented to provide additional information to FN communities in their efforts to reduce tobacco use. Significant benefits (i.e., decreasing tobacco consumption, creating funding for community programs) may be derived from implementing the tax in communities where support can be mobilized.

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REFERENCES

- British Columbia Vital Statistics Agency & First Nations and Inuit Health Branch. (2004). *Regional analysis of health statistics for Status Indians in British Columbia, 1991-2002*. Vancouver, BC: Author.
- Canada Revenue Agency. (2006). *First Nations tax*. Retrieved March 10, 2009 from <http://www.cra-arc.gc.ca/E/pub/gp/rc4072/README.html>
- First Nations Centre. (2005). *First Nations Regional Longitudinal Health Survey (2002/03)* (pp. 105-112). Ottawa, ON: Author.
- First Nations and Inuit Health Branch, Pacific Region. (2005). [Community Survey]. Unpublished data.

- Fiscal Realities. (1997). *First Nation Taxation and New Fiscal Relationships*. Report presented to The Indian Taxation Advisory Board and the Research and Analysis Directorate, Policy and Strategic Direction Branch of The Department of Indian Affairs and Northern Development. West Vancouver, BC: Author.
- Frances, R.J., & Miller, S.I. (Eds.). (1998). *Clinical textbook of addictive disorders* (2nd ed.). New York : The Guilford Press.
- French, L.L. (2000). *Addictions and Native Americans*. Westport, CT: Praeger.
- Health Canada (2005). *Tobacco Factsheets*. Retrieved October 23, 2006 from http://www.hc-sc.gc.ca/hl-vs/tobac-tabac/fact-fait/fs-if/index_e.html
- Health Canada (2006a). *Canadian Tobacco Use Monitoring Survey, 2003*. Retrieved October 23, 2006 from http://www.hc-sc.gc.ca/hl-vs/tobac-tabac/research-recherche/stat/ctums-esutc/fs-if/2003/index_e.html
- Health Canada. (2006b). *Report to the Conference of the Parties on the Implementation of the Framework Convention on Tobacco Control*. Retrieved October 23, 2006 from <http://www.hc-sc.gc.ca/hl-vs/pubs/tobac-tabac/cop-cdp/part-section2-eng.php>
- Houston, T.K., Kiefe, C.I., Person, S.D., Pletcher, M.J., Liu, K. & Iribarren, C. (2006). Active and passive smoking and development of glucose intolerance among young adults in a prospective cohort: CARDIA study. *BMJ*, 332(7549),1064-1069.
- Indian and Northern Affairs Canada. (2007). *Registered Indian Demography - Population, Household and Family Projections, 2004-2029*. Retrieved March 29, 2009 from <http://www.ainc-inac.gc.ca/ai/rs/pubs/re/rgd/rgd-eng.asp>
- Kegler, M.C., Cleaver, V.L., & Yazzie-Valencia, M. (2000). An exploration of the influence of family on cigarette smoking among American Indian adolescents. *Health Education Research*, 15(5), 547-557.
- Makomaski Illing E.M., & Kaiserman, M.J. (2004). Mortality attributable to tobacco use in Canada and its regions, 1998. *Canadian Journal of Public Health*, 95(1), 38 - 44.
- Meier, K.J., & Licari, M.J. (1997). The effect of cigarette taxes on cigarette consumption, 1955 through 1994. *American Journal of Public Health*, 87(7), 1126-1130.
- O'Brien, C.P., & McLellan, A.T. (1996). Myths about the treatment of addiction. *Lancet*, 347, 237-240.
- Ontario Tobacco Research Unit. (2007). *The Tobacco Control Environment: Ontario and Beyond. Special Reports: Monitoring and Evaluation Series, 2006-2007 (Vol. 13, No. 1). Fact Sheet 1.5: Tobacco Taxes*. Toronto, ON: Author.
- Physicians for A Smoke-Free Canada. (2008) *Tax revenues from tobacco sales*. Retrieved March 29, 2009 from <http://www.smoke-free.ca/factsheets/pdf/totaltax.pdf>

- Prochaska, J.J., Delucchi, K., & Hall, S.M. (2004). A meta-analysis of smoking cessation interventions with individuals in substance abuse treatment or recovery. *Journal of Consulting & Clinical Psychology*, 72(6), 1144-1156.
- Smoking and Health Action Foundation. (2003). Average price of a carton of 200 cigarettes. Retrieved October 23, 2006 from <http://www.hpclearinghouse.ca/tobacco/files/taxmap24april03.pdf>
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (1999). *Best practices for comprehensive tobacco control programs*. Atlanta, GA: Author.
- U.S. Department of Health and Human Services, Centers for Diseases Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. (2000). *Reducing Tobacco Use: A Report of the Surgeon General*. Atlanta, GA: Author.
- Vaillant, G.E. (1988). What can long-term follow up teach us about relapse and prevention of relapse in addiction? *British Journal of Addiction*, 83, 1147-1157.
- Wardman, D., & Khan, N. (2004). Smoking-attributable mortality among Columbia's First Nations population. *International Journal on Circumpolar Health*, 63(1), 81-92.
- Wardman, D., & Khan, N. (2005). Registered Indians and tobacco taxation: A culturally appropriate strategy? (Commentary). *Canadian Journal of Public Health*, 96(6), 451-453.

INTIMATE PARTNER VIOLENCE AND ALCOHOL, DRUG, AND MENTAL DISORDERS AMONG AMERICAN INDIAN WOMEN FROM SOUTHWEST TRIBES IN PRIMARY CARE

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Abstract: The relationship of intimate partner violence (IPV) with mental disorders was investigated among 234 American Indian/Alaska Native female primary care patients. Results indicated that unadjusted prevalence ratios for severe physical or sexual abuse (relative to no IPV) were significant for anxiety, PTSD, mood, and any mental disorder. Adjusted prevalence ratios showed severe physical or sexual IPV to be associated with any mood disorder. Patterns of IPV and mental health have implications for detection and service utilization.

The annual economic victim-related costs of intimate partner violence (IPV) in the U.S. have been estimated at \$67 billion (Miller, Cohen, & Wiersema, 1996). These costs are associated with the severe and negative health and social consequences of violence to victims of IPV. These consequences include worse physical health, (Brokaw et al., 2002; Hathaway et al., 2000; Silverman, Raj, Mucci, & Hathaway, 2001) worse mental health, (Hien & Bukszpan, 1999; Roberts, Williams, Lawrence, & Raphael, 1998; Woods, 2000) and lower employment status (Browne, Salomon, & Bassuk, 1999; Bryne, Resnick, Kilpatrick, Best, & Saunders, 1999). For alcohol, drug, and mental health (ADM) outcomes specifically, IPV is associated with a variety of outcomes including phobias (Roberts et al.), depression (Hathaway et al.; Roberts et al.), dysthymia (Roberts et al.), anxiety (Hathaway et al.; Roberts et al.), substance dependence (Roberts et al.; Silverman et al.), somatization (Roberts et al.), suicidal ideation (Hathaway et al.; Silverman et al.), and PTSD (Hien & Bukszpan; Woods).

While the prevalence and consequences of IPV are well established in the research literature in general, there are certain limitations for American Indian/Alaska Native (AI/AN) populations. First, estimates for AI/ANs are based on limited research; thus, it is important for future research to provide more baseline information on the prevalence of different categories of IPV (e.g., different levels of severity and emotional abuse; Oetzel & Duran, 2004). Second, IPV is underreported in

primary care settings. A dramatic illustration of the difficulty of identifying IPV is evident at the Albuquerque Indian Hospital, the site of this study. In the entire history of hospital ambulatory and outpatient records, only 123 AI women (1.6%) have been identified as victims of domestic violence, and 58 of those women were identified as late as 1996 (Clark, 2001). These statistics strongly suggest that episodes of IPV experienced by AI/AN women are seriously underdetected and underreported in primary care settings. Third, while the ADM consequences of IPV are clear for the general population, there is limited research on AI/AN women. Further, the research tends to examine the impact of IPV in general (emphasizing the most physically violent behaviors)—not the different levels of severity (including emotional abuse)—on ADM disorders.

These limitations are significant for several reasons. First, primary care prevalence information is crucial for estimation of unmet need and for planning public health prevention and clinical services. The highest research priority in the area of AI/AN mental health is the need for estimations of illness (including IPV and ADM disorders), and for a better understanding of service utilization and help-seeking for these problems (Indian Health Service, 1995). In particular, health care providers need to have an understanding of the rates of IPV and ADM disorders to improve their detection and treatment of these problems. Second, data on violence and on mental illness risk and protective factors foster the development of culturally specific etiological hypotheses and treatment and prevention models (Jenkins, 2001). Cultural factors, such as meanings and orientations towards psychological distress, may contribute to the lower minority access to care as much as do economic factors (Wells, Klap, Koike, & Sherbourne, 2001). Finally, to address the racial disparity in disease and social problems, the lack of data on important social problems among communities of color must be addressed (United States Commission on Civil Rights, 2000; Wells et al.). Thus, the purpose of this study is to examine the relationship between the severity level of IPV and five different categories of ADM disorders (any anxiety, PTSD, any substance abuse, any mood, and any disorder) in AI/AN women from Southwest tribes (SW) presenting for primary care.

METHODS

Study Location and Sampling Design

The study was conducted at the outpatient appointment and urgent care clinics of the Indian Health Service (IHS) hospital in Albuquerque, New Mexico. The hospital is part of a regional IHS unit, which provides health care to five tribes and the urban AI population in the immediate area. The Albuquerque service unit records approximately 97,000 visits per year, 60% of them at the hospital site. Women were approached in the waiting area to determine their eligibility and were considered eligible if they were between 18-45 years of age, received their medical care from IHS

facilities, and were willing to sign the informed consent form. The women were fluent in English (a criterion for inclusion) and were given an incentive of \$20 per hour for their participation. The local IHS Unit Health Board approved this study, as did the Institutional Review Boards of both the University of New Mexico Health Sciences Center and the IHS National Research Office.

The study was designed as a two-stage procedure because of this method's efficacy in epidemiological studies (Miller, 1996). Stage I used the 12-item General Health Questionnaire (GHQ-12; Goldberg & Williams, 1988) as a screener for mental distress. The GHQ-12 assesses the severity of a mental problem over the past few weeks. Each item has a 4-point response scale; in this study, we used the scoring system of 0-0-1-1, as recommended by Goldberg and Williams. Scores ranged from 0-12, with high scores indicating worse health. The GHQ was chosen because of its high estimates of internal validity, its established validity in a number of countries, and its ease of use; and because it is the most common measure of mental well-being (Jackson, 2007). Subjects were stratified according to high (≥ 3) or low (≤ 2) GHQ scores. These numbers were selected to ensure adequate sample size in high and low groups and to provide dichotomous groups (i.e., not people in the middle); this approach follows prior research (Duran et al., 2004). A total of 489 eligible study subjects completed the GHQ, with 246 (50.3%) obtaining high scores and 243 (49.7%) obtaining low scores. Of the 246 women with high GHQ scores, 97% ($n = 238$) were selected for Stage II interviews, as were a random sample of 65% of those with low GHQ scores ($n = 158$). These percentages were chosen in order to have a sample size with adequate power. Of these 396 women selected for Stage II interviews, 162 either could not be located by information taken during Stage I or refused to participate in Stage II. It is known that the population is transient, and many of the women did not come back to care. Presumably, they returned to their home reservations for care, although we have no documentation to that effect. Stage II interviews were completed within four months of initial contact.

Measures

The interviews consisted of the University of Michigan version of the Composite International Diagnostic Interview (CIDI), the Revised Conflict Tactics Scale (CTS2), and demographics. The CIDI, developed jointly by the World Health Organization and the Substance Abuse and Mental Health Services Administration, is a clinical instrument that determines psychiatric (including alcohol and drug) diagnoses through interviews by lay interviewers. The validity of the instrument has been established in cross-cultural settings (Wittchen, 1994; Wittchen, Robins, Cottler, Sartorius, Burke, & Regier, 1991). The CIDI version used allows for case ascertainment based on the Diagnostic Statistical Manual IV (American Psychiatric Association, 2000; Mitchell, Beals, Novins, Spicer, & AI-SUPERPFP Team, 2003; Whitesell et al., 2006). Diagnoses were divided into five general

categories of past-year ADM disorders (any anxiety, PTSD, any substance abuse/dependence, any mood, and any disorder). For this study, we examined PTSD as a separate category from anxiety because of its relationship with IPV in prior research (e.g., DeJonghe, Bogat, Levendosky, & von Eye, 2008; Kaminer, Grimsrud, Myer, Stein, & Williams, 2008).

The CTS2 (Straus, 1979) was used to determine both the presence and dimensions of IPV. The CTS2 has operationalizations of minor and severe levels of conflict, is easily self-administered in populations with varying educational backgrounds, and takes only 10 minutes to complete. The CTS2 has been validated for U.S. minority populations (Kolbo, 1996; Malcoe, Duran, & Fickel, 2002).

Statistical Analyses

Log-Binomial models were used to estimate the prevalence ratios between IPV and each of the ADM categories. The SAS (Version 8.1) procedure GENMOD was used to accomplish this task. Prevalence ratios were estimated both on the univariate level (i.e., individual log-Binomial models were fitted with one of the independent variable or covariates at a time), and on the multivariate level (i.e., log-Binomial models were fitted with both the independent variable and the covariates at the same time). Therefore, both unadjusted and adjusted prevalence ratios were obtained. The “copy method” was used to obtain convergence of all multivariate models (Deddens, Peterson, & Lei, 2003).

A purposeful selection method was used to help select the final “best” models in the multivariate analyses. The following steps outline the specific model fitting procedures:

- 1) Preliminary bivariate analyses between demographics and IPV revealed a potential quadratic trend for age and the dependent variables and potential IPV*family history of alcohol and IPV*age interactions. Thus, these interaction terms were also included when fitting the multivariate models.
- 2) Univariate/unadjusted models for IPV, demographics, age², IPV*age, and IPV*family history of alcohol, respectively were analyzed.
- 3) A multivariate model included IPV and all covariates with *p* values less than .25, and used backward elimination to refine the model until the reduced model only contained variables significant at the .10 level (IPV was included regardless of *p* value).
- 4) Covariates that were significant at a .10 level and not originally selected were added back into the model one at a time.

RESULTS

Demographics and IPV Categorization

All participants were enrolled with a tribe, and over 90% were members of SW tribes. Approximately 61% were married or living in common-law relationships, and most (88%) lived in urban areas. More than one quarter (29%) of participants reporting income information lived below the federal poverty level, and only 33% lived above 185% of the poverty level. Table 1 displays the descriptive statistics for the variables included in this study.

Table 1
Demographics (N = 234)

	Frequency	Percent
IPV		
None	48	20.5
Any lifetime minor or severe psychological	84	35.9
Any lifetime severe	102	43.6
Employment		
Employed	151	64.5
Unemployed	44	18.8
Other	39	16.7
Debt		
None	65	27.8
Some	105	44.9
Very Much	62	26.5
Education		
< HS	25	10.7
HS	68	29.1
> HS	141	60.3
Family History of Alcohol Abuse		
No	71	30.3
Yes	163	69.7
Age	30.02 (M)	7.54 (SD)

Note: Any lifetime minor includes physical, sexual, or injury or severe psychological aggression.

Any lifetime severe includes physical, sexual, or injury

The primary independent variable was IPV exposure. Initially, IPV was coded as a five-category variable: (a) no physical/sexual/injury and no severe psychological; (b) minor physical/sexual/injury; (c) past-year severe physical/sexual/injury, (d) lifetime severe physical/sexual/injury, and (e) severe psychological but no severe physical/sexual/injury). Prevalence rates of each of the five dependent variables across these five categories of IPV were estimated (Table 2).

Table 2
Past-Year Prevalence Rates of Mental Disorders by IPV (N = 234)

IPV	Any Anxiety		PTSD	
	Freq (%)	95% CI	Freq (%)	Freq (%)
None	15(31.25)	(17.6,44.9)	2(4.17)	(0,10.0)
Minor	20(47.62)	(31.9,63.4)	4(9.52)	(0.3,18.8)
Past Sev	31(49.21)	(36.5,61.9)	16(25.40)	(14.3,36.4)
Life Sev	21(53.85)	(37.5,70.2)	8(20.51)	(7.3,33.8)
Sev Psy	19(45.24)	(29.5,60.9)	4(9.52)	(0.3,18.8)
Total	106(45.30)	(38.9,51.7)	34(14.53)	(10.0,19.1)

IPV	Any Substance Abuse		Any Mood		Any Disorder	
	Freq (%)	Freq (%)	Freq (%)	95% CI	Freq (%)	95% CI
None	6(12.50)	(2.8, 22.2)	5(10.42)	(1.5, 19.4)	18(37.50)	(23.3,51.7)
Minor	7(16.67)	(4.9,28.4)	9(21.43)	(8.5,34.4)	27(64.29)	(49.2,79.4)
Past Sev	13(20.63)	(10.4,30.9)	13(20.63)	(10.4,30.9)	46(73.02)	(61.7,84.3)
Life Sev	6(15.38)	(3.5,27.2)	12(30.77)	(15.6,45.9)	29(74.36)	(60.0,88.7)
Sev Psy	4(9.52)	(0.3,18.8)	5(11.90)	(1.7,22.1)	24(57.14)	(41.5,72.8)
Total	36(15.38)	(10.7,20.0)	44(18.80)	(13.8,23.8)	144(61.54)	(55.3,67.8)

Note: Minor = minor physical, sexual, or injury
 Past Sev = past-year severe physical, sexual, or injury
 Life Sev = lifetime severe physical, sexual, or injury
 Sev Psy = lifetime psychological aggression

These estimates and the preliminary analyses suggested that this five-category IPV variable should be collapsed into a three-category variable: (a) no physical/sexual/injury and no severe psychological; (b) severe psychological or minor physical/sexual/injury; and (c) severe physical/sexual/injury. The preliminary analyses suggested that there were not sufficient cell sizes warranted for the IPV prevalence ratio analysis controlling for the covariates. Additionally, the prevalence ratios suggested that severe psychological and minor physical/sexual/injury had similar relationships with the ADM disorders as did past-year and lifetime severe physical/sexual/injury.

Multivariate Models

Table 3 presents both the unadjusted and adjusted prevalence ratio estimates with 95% confidence intervals from the univariate and multivariate prevalence ratio analyses. For any anxiety disorder, lifetime severe IPV and a lot of debt had significant unadjusted prevalence ratios. Women experiencing severe IPV were 60% more likely to have an anxiety disorder than women who had

no experience with IPV. For the multivariate model, a lot of debt was significant, and there was also a significant IPV*family history of alcohol interaction. Women experiencing severe IPV with a family history of alcohol were almost 2.5 times more likely to have an anxiety disorder than women not experiencing IPV and a family history of alcohol. IPV did not have a relationship with anxiety disorder if there was no family history of alcohol.

Table 3
Prevalence Ratios of IPV and Covariates for ADM Disorders

	Any Anxiety PR (95% CI)		PTSD PR (95% CI)		Any Substance PR (95% CI)	
	Unadjusted	Adjusted	Unadjusted	Adjusted	Unadjusted	Adjusted
IPV		See IPV*FHALC				See Age*IPV
None	1.00	interaction	1.00	1.00	1.00	interaction
Lifetime minor	1.49(0.92,2.40)		2.29(0.51,10.33)	1.66(0.37,7.44)	1.05(0.41,2.65)	
Lifetime severe	1.63(1.03,2.59)*		5.65(1.39,22.93)*	3.97(0.98,16.14)	1.49(0.64,3.49)	
Age	1.01(0.99,1.03)		1.03(0.99,1.07)		1.00(0.96,1.05)	See Age*IPV
Employment						
Employed	1.00		1.00		1.00	1.00
Unemployed	0.91(0.61,1.35)		0.62(0.23,1.71)	0.56(0.21,1.49)	0.41(0.13,1.30)	0.31(0.10,0.97)
Other	1.14(0.80,1.62)		1.41(0.68,2.92)	1.76(0.93,3.32)	1.24(0.61,2.53)	1.35(0.67,2.70)
Debt						
None	1.00	1.00	1.00	1.00	1.00	
Some	1.38(0.93,2.05)	1.33(0.90,1.96)	2.63(0.93,7.48)	2.56(0.93,7.07)	1.47(0.68,3.16)	
Very Much	1.62(1.08,2.44)*	1.55(1.04,2.32)*	3.15(1.07,9.23)*	3.09(1.10,8.74)*	1.05(0.42,2.62)	
Education						
< HS	1.16(0.79,1.71)		1.41(0.58,3.41)		1.99(0.87,4.56)	1.35(1.03,5.35)
HS	0.73(0.51,1.05)		0.93(0.45,1.94)		1.59(0.82,3.07)	2.05(1.06,3.95)
> HS	1.00		1.00		1.00	1.00
Family Hist of A		See IPV*FHALC				
No	1.00	Interaction	1.00	1.00	1.00	
Yes	1.16(0.84,1.60)		3.27(1.20,8.93)*	2.98(1.11,7.99)*	1.13(0.58,2.22)	
IPV*(FHALC=no)						
None		1.00				
Lifetime minor		1.00(0.53,1.91)				
Lifetime severe		0.89(0.44,1.81)				
IPV*(FHALC=yes)						
None		1.00				
Lifetime minor		2.16(0.95,4.92)				
Lifetime severe		2.43(1.09,5.43)*				
Age ²						
Age*(IPV=None)						
Age*(IPV=Life.minor)						0.84(0.73,0.97)
Age*(IPV=Life.severe)						0.95(0.86,1.06)

Table 3, Continued
Prevalence Ratios of IPV and Covariates for ADM Disorders

	Any Mood PR (95% CI)		Any Disorder PR (95% CI)	
	Unadjusted	Adjusted	Unadjusted	Adjusted
IPV				
None	1.00	1.00	1.00	Interaction
Lifetime Minor	1.49(0.92,2.40)	1.72(0.67,4.44)	1.62(1.08,2.42)*	See IPV*FHALC
Lifetime Severe	1.63(1.03,2.59)*	2.53(1.05,6.09)*	1.96(1.34,1.88)*	
Age	1.01(0.99,1.03)	0.72(0.55,0.95)	1.01(0.99,1.02)	
Employment				
Employed	1.00		1.00	
Unemployed	0.91(0.61,1.35)		0.99(0.76,1.29)	
Other	1.14(0.80,1.62)		0.95(0.71,1.27)	
Debt				
None	1.00		1.00	1.00
Some	1.38(0.93,2.05)		1.38(1.03,1.84)*	1.32(1.01,1.72)*
Very Much	1.62(1.08,2.44)		1.45(1.07,1.97)*	1.46(1.12,1.90)*
Education				
< HS	1.16(0.79,1.71)	1.33(0.66,2.68)	1.27(1.00,1.60)*	
HS	0.73(0.51,1.05)	0.34(0.15,0.77)	0.82(0.63,1.06)	
> HS	1.00	1.00	1.00	
Family Hist of A				
No	1.00		1.00	See IPV*FHALC
Yes	1.16(0.84,1.60)		1.22(0.95,1.55)	Interaction
IPV*(FHALC=no)				
None				1.00
Lifetime Minor				1.20(0.73,1.97)
Lifetime Severe				0.96(0.53,1.73)
IPV*(FHALC=yes)				
None				1.00
Lifetime Minor				2.31(1.12,4.74)*
Lifetime Severe				3.08(1.53,6.20)*
Age ²		1.00(1.00,1.01)		

Note: Any lifetime minor includes physical, sexual, or injury or severe psychological aggression

Any lifetime severe includes physical, sexual, or injury

FHALC = Family history of alcohol

* = $p < .05$

For PTSD, severe IPV, a lot of debt, and family history of alcohol all had significant unadjusted prevalence ratios. Women experiencing severe IPV were over five times more likely to have PTSD than women who had no experience with IPV. A lot of debt and family history of alcohol remained in the multivariate model, but severe IPV was not significant at the .05 level (although the prevalence ratio for severe IPV relative to no IPV was almost four).

For any substance abuse disorder, there were no significant unadjusted prevalence ratios for any of the variables. The multivariate model did include education; women with more than a high school education were less likely to experience a substance abuse disorder than either women with only a high school education or less than a high school education. In addition, there was a significant age² X IPV interaction. Specifically, the quadratic age term was significant for lifetime minor IPV relative to no IPV. However, this finding appears to be an artifact of the sample, as there were certain ages without any data. Thus, we have chosen not to further interpret this finding.

For any mood disorder, severe IPV and a lot of debt had significant unadjusted prevalence ratios. The multivariate model included only severe IPV. Specifically, women experiencing severe IPV were over 2.5 times more likely to have a mood disorder than women who had not experienced IPV.

For any disorder, severe IPV and a lot of or some debt had significant unadjusted prevalence ratios. For the multivariate model, a lot of or some debt was significant, and there was also a significant IPV*family history of alcohol interaction. Women experiencing severe IPV with a family history of alcohol were over three times more likely to have any disorder than women not experiencing IPV and a family history of alcohol. Additionally, women experiencing minor IPV or severe psychological aggression with a family history of alcohol were over two times more likely to have any disorder than women not experiencing IPV and without a family history of alcohol. IPV did not have a relationship with any disorder if there was not a family history of alcohol.

DISCUSSION

The univariate findings regarding severe IPV and ADM outcomes are largely consistent with research in other settings and populations (Hathaway et al., 2000; Hien & Bukszpan, 1999; Roberts et al., 1998; Silverman et al., 2001; Woods, 2000). Severe IPV has been associated with depression, anxiety, and PTSD in a variety of culturally diverse clinical and community samples.

One contradiction with prior research is the lack of a relationship of severe IPV to substance abuse in the current sample (Roberts et al., 1998; Silverman et al., 2001). To interpret this finding, it is important to put substance use (particularly alcohol) into context. Research on AI/AN drinking indicates that (a) alcohol consumption and abuse levels vary by tribe and over time (Beauvais, 1998; May, 1996; O'Connell et al., 2006; Whitesell et al., 2006), (b) alcohol consumption is higher in urban areas than on reservations (Beauvais; Costello, Farmer, Angold, Burns, & Erkanli, 1997; May), (c) women have high rates of alcohol abstention (Beauvais; Costello et al., 1997; May; O'Connell et al., 2006), and (d) alcohol consumption patterns are bimodal—there are large numbers of both abstainers and heavy binge drinkers in these populations (Beauvais; May; O'Connell et al.;

Whitesell et al.). The latter two points likely impact the relationship between IPV and substance abuse in that, as a cultural trend, there are significant numbers of women who do not use alcohol regardless of their traumatic experiences. Alternatively, substance use may be underreported in this sample, which further limits the likelihood of finding a relationship between IPV and substance abuse. Although the DSM-IV criterion for diagnosis of substance alcohol abuse may be problematic in AI/AN populations (May), it is plausible that the unreliability of recall as a measure of use may equally lead to underestimates of the prevalence of substance abuse. Finally, substance use is both a cause and a consequence of IPV (Anderson, 2002). This study only examined cross-sectional data; thus, the relationship between IPV and substance abuse may be diminished because this study cannot clearly identify the sequential pathway.

The multivariate findings suggest that IPV is only a factor for anxiety or any disorder in women with a family history of alcohol. One explanation is that both IPV and family history of alcohol are associated with anxiety disorders (Preuss, Schuckit, Smith, Barnow, & Danko, 2002; Schuckit et al., 2003). The combination of both factors places a “double impact” on the victim. However, for women without a family history of alcohol, other significant factors (i.e., debt) likely account for the experience of anxiety disorders.

Theoretically, our analyses recognize that, in general, family violence occurs within a broader context of social, economic, historic, and cultural factors. This recognition warrants adjustment for other variables, such as number of children, low educational level, parental substance abuse, poverty, early behavioral problems and marital disruption, that may contribute to negative outcomes (Dube et al., 2001; Horwitz, Widom, McLaughlin, & White, 2001; Kendler et al., 2000; Widom, 1999). Within an AI/AN context, adverse family experiences reach beyond immediate family characteristics and include exposure to misguided educational and child welfare policies, such as forced boarding school attendance and racist practices of child protective service agencies (Cross, Earle, & Simmons, 2000; Madrigal, 2001; Mannes, 1995).

Additionally, the univariate and multivariate findings suggest that minor IPV (severe psychological or minor physical/sexual) generally is not associated with ADM outcomes. Specifically, minor IPV was only associated with any disorder when there was a family history of alcohol. The lack of a relationship is contradictory to some research on other ethnic groups, such as White and Black women (Wagner, Mongan, Hamrick, & Hendrick, 1995). However, the findings are consistent with research showing that severe physical and sexual IPV has a stronger relationship with ADM disorders than psychological aggression. Consistent with this assertion, the findings of this study may reflect the fact that psychological aggression is a precursor to the development of physical aggression (Murphy & Hoover, 1999; Stith, Smith, Penn, Ward, & Tritt, 2004). The prevalence ratios generally reflected an increased pattern from minor IPV to severe IPV.

Implications

This study suggests the importance of improving identification of IPV in the primary care setting. Unfortunately, the identification rate of IPV in patients is poor in both mainstream and AI/AN populations. For example, one study found that physicians' files documented only 1% of possible cases in a population with an IPV prevalence of 30% (including physical and emotional violence; Martins, Holzapfel, & Baker, 1992).

Lack of routine screening for IPV in primary care settings can result in the unintended consequence of continued IPV. Because of the confidentiality inherent in medical care, victims may feel more comfortable reporting their IPV experience here than in other settings. Unfortunately, there is often a breakdown in transmission of assistance (Little & Kaufman Kantor, 2002). Many health care providers are uncomfortable addressing IPV and do not feel that health care settings, including emergency rooms, are appropriate for such intervention (Ramsay Richardson, Carter, Davidson, & Feder, 2002). However, one study found that 43 to 85% of women (especially those who are IPV victims) believe that screening in health care settings is appropriate (Rosenberg & Fenley, 1991). Further, primary care identification and intervention efforts could reduce IPV incidence by 75% (Rosenberg & Fenley).

Ideally, health care providers would have a policy of routine screening to detect cases of IPV. Clark examined the screening rates of IHS facilities and found that facilities with policies and procedures related to domestic violence were more likely to screen than facilities without such policies (Clark, 2001). Having a domestic violence committee also increased the likelihood of screening. Additionally, health care providers need training on how to screen and talk about IPV with patients since, from the patient perspective, shame, fear of criminal justice involvement, and fear of more violence may prevent honest disclosure (Chester, Robin, Koll, Lopez, & Golden, 1994; Duran Duran, & Brave Heart, 1998).

Finally, we would be remiss if we did not acknowledge that programs servicing AI/AN populations are woefully underfunded. Annual per capita expenditures for AI/AN health care programs fall below the level for every other federal medical program and standard; the difference has been characterized by the United States Commission on Civil Rights (2004) as a "revolting disparity." On a systems level, our study documents unmet mental health service needs and supports the Commission's contention that there are multiple barriers to care that must first be overcome by an increase in funding. Without addressing these barriers, there is limited hope to increasing the identification and treatment of mental health outcomes associated with IPV.

Limitations and Conclusions

There are several limitations of this study. The first is that the CIDI, which is widely used for psychiatric studies, may be less accurate than structured diagnostic interviews conducted by culturally competent, licensed mental health professionals. This observation may be particularly true with regard to the applicability of the DSM-IV alcohol abuse and dependence criteria to the unique (high quantity, sporadic, binge frequency) drinking style of many AI/AN groups (May & Gossage, 2001). Second, data were collected at a single site from women 18-45 in primarily SW tribes, which limits the general applicability of these findings to other AI/AN populations. Third, this study used a cross-sectional design, which limits our ability to draw causal relations. Future research will have to determine the causal pathway for IPV and ADM disorders. Fourth, the sample of participants with PTSD was small; thus, firm conclusions about the relationship between IPV and PTSD cannot be drawn from these data. Finally, this study only examines women as victims of IPV. There is a growing literature examining the more complex nuances of violence among intimate partners (Frieze, 2005; Graham-Kevan & Archer, 2005; Richardson, 2005). Research suggesting that gender is a risk factor for IPV is limited because, generally, this research does not consider women as perpetrators and men as victims. Accordingly, there is limited research about AI/AN women as perpetrators of violence.

Despite these caveats, the findings illustrate that IPV is a common occurrence for AI/AN women presenting in primary care settings and has significant associations with ADM disorders. The study suggests the importance of developing procedures for identifying, reporting, and/or treating IPV and ADM disorders in primary care settings. The challenge will be training health care providers on these procedures, as IPV and ADM are historically underidentified and underreported.

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REFERENCES

- Anderson, K.L. (2002). Perpetrator or victim? Relationships between intimate partner violence and well-being. *Journal of Marriage and Family*, 64, 851-863.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: Primary care version* (4th ed.). Washington, DC: Author.
- Beauvais, F. (1998). American Indians and alcohol. *Alcohol Health & Research World*, 22, 253-259.
- Brokaw, J., Fullerton-Gleason, L., Olson, L., Crandall, C., McLaughlin, S., & Sklar, D. (2002). Health status and intimate partner violence: A cross-sectional study. *Annals of Emergency Medicine*, 39, 31-38.
- Browne, A., Salomon, A., & Bassuk, S.S. (1999). The impact of recent partner violence on poor women's capacity to maintain work. *Violence Against Women*, 5, 393-426.
- Bryne, C.A., Resnick, H.S., Kilpatrick, D.G., Best, C.L., & Saunders, B.E. (1999). The socioeconomic impact of interpersonal violence on women. *Journal of Consulting and Clinical Psychology*, 67, 362-366.
- Chester, B., Robin, R.N., Koll, M.P., Lopez, J., & Goldman, D. (1994). Grandmother dishonored: Violence against women by male partners in American Aboriginal communities. *Violence and Victims*, 9, 249-258.
- Clark, D.W. (2001). Domestic violence screening, policies, and procedures in Indian Health Service facilities. *Journal of the American Board of Family Practice*, 14, 252-258.
- Costello, E.J., Farmer, E.M., Angold, A., Burns, B.J., & Erkanli, A. (1997). Psychiatric disorders among American Indian and white youth in Appalachia: The Great Smoky Mountains Study. *American Journal of Public Health*, 87, 827-832.
- Cross, T.A., Earle, K.A., & Simmons, D. (2000). Child abuse and neglect in Indian country: Policy issues. *Families in Society*, 81(1), 49-58.
- Deddens, J.A., Peterson, M.R., & Lei, X. (2003). *Estimation of prevalence ratios when PROC GENMOD does not converge*. SAS Users Group International Proceedings. Retrieved February 20, 2009 from <http://www2.sas.com/proceedings/sugi28/270-28.pdf>
- DeJonghe, E.S., Bogat, G.A., Levendosky, A.A., & von Eye, E. (2008). Women survivors of intimate partner violence and post-traumatic stress disorder: Prediction and prevention. *Journal of Postgraduate Medicine*, 54, 294-300.
- Dube, S.R., Anda, R.F., Felitti, V.J., Chapman, D.P., Williamson, D.R., & Griles, W.H. (2001). Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span: Findings from the adverse childhood experiences study. *Journal of the American Medical Association*, 286, 3089-3096.

- Duran, B., Duran, E., & Brave Heart, M.Y.H. (1998). American Indian and/or Alaska Natives and the trauma of history. In R. Thornton (Ed.), *Studying Native America: Problems and prospects* (pp. 60-76). Madison, WI: University of Wisconsin Press.
- Duran, B., Sanders, M., Skipper, B., Waitzkin, H., Malcoe, L. H., Paine, et al. (2004). Prevalence and correlates of mental disorders among Native American women in primary care. *American Journal of Public Health, 94*, 71-77.
- Frieze, I.H. (2005). Female violence against intimate partners: An introduction. *Psychology of Women Quarterly, 29*, 229-237.
- Graham-Kevan, N., & Archer, J. (2005). Investigating three explanations of women's relationship aggression. *Psychology of Women Quarterly, 29*, 270-277.
- Goldberg D.P., & Williams, P.(1988). *A user's guide to the General Health Questionnaire*. Windsor: NFER-Nelson.
- Hathaway, J.E., Mucci, L.A., Silverman, J.G., Brooks, D.R., Mathews, R., & Pavlos, C.A. (2000). Health status and health care use of Massachusetts women reporting partner abuse. *American Journal of Preventive Medicine, 19*, 302-307.
- Hien, D., & Bukszpan, C. (1999). Interpersonal violence in a "normal" low-income control group. *Women & Health, 29*, 1-15.
- Horwitz, A.V., Widom, C.S., McLaughlin, J., & White, H.R. (2001). The impact of childhood abuse and neglect on adult mental health: A prospective study. *Journal of Health and Social Behavior, 42*, 184.
- Indian Health Service. (1995). *National plan for Native American mental health services, amended*. Rockville, MD: Department of Health and Human Services, Mental Health Programs.
- Jackson, C. (2007). The General Health Questionnaire. *Occupational Medicine, 57*, 79.
- Jenkins, R. (2001). Making psychiatric epidemiology useful: The contribution of epidemiology to government policy. *Acta Psychiatrica Scandinavia, 103*, 2-14.
- Kaminer, D., Grimsrud, A., Myer, L., Stein, D. J., & Williams, D. R. (2008). Risk for post-traumatic stress disorder association with different forms of interpersonal violence in South Africa. *Social Science & Medicine, 67*, 1589-1595.
- Kendler, K.S., Bulik, C.M., Silberg, J., Hettema, J. M., Myers, J., & Prescott, C.A. (2000). Childhood sexual abuse and adult psychiatric and substance use disorders in women: An epidemiological and cotwin control analysis. *Archives of General Psychiatry, 57*, 953-959.
- Kolbo, J.R. (1996). Risk and resilience among children exposed to family violence. *Violence and Victims, 11*(2), 113-128.
- Little, L., & Kaufman Kantor, G. (2002). Using ecological theory to understand intimate partner violence and child maltreatment. *Journal of Community Health Nursing, 19*, 133-145.

- Madrigal, L. (2001). Indian child and welfare act: Partnership for preservation. *American Behavioral Scientist*, 44, 1505.
- Malcoe, L.H., Duran, B.M., & Ficek, E.E. (2002). Social stressors in relation to intimate partner violence against Native American women. *Annals of Epidemiology*, 12, 525.
- Mannes, M. (1995). Factors and events leading to the passage of the Indian child welfare act. *Child Welfare*, 74, 264.
- Martins, R., Holzapfel, S., & Baker, P. (1992). Wife abuse: Are we detecting it? *Journal of Womens Health*, 1, 77-80.
- May, P. (1996). Overview of alcohol abuse epidemiology for American Indian populations. In G.D. Sandefur, R.R. Rindfuss, & B. Cohen (Eds.), *Changing numbers, changing needs: American Indian demography and public health* (pp. 235-261). Washington, DC: National Academy Press.
- May P.A., & Gossage, J.P. (2001). New data on the epidemiology of adult drinking and substance use among American Indians of the northern states: Male and female data on prevalence, patterns, and consequences. *American Indian and Alaska Native Mental Health Research*, 10(2), 1-26.
- Miller, T.R., Cohen, M.A., & Wiersema, B. (1996). *Victim costs and consequences: A new look*. Washington, DC: National Institute of Justice.
- Mitchell, C. M., Beals, J., Novins, D. K., Spicer, P., & AI-SUPERPFP Team (2003). Drug use among two American Indian populations: Prevalence of lifetime use and DSM-IV substance use disorders. *Drug and Alcohol Dependence*, 69, 29-41.
- Murphy, C.M., & Hoover, S.A. (1999). Measuring emotional abuse in dating relationships as a multifactorial construct. *Violence and Victims*, 14, 39-53.
- O'Connell, J., Novins, D. K., Beals, J., Croy, C., Barón, A. E. Spicer, P., et al. (2006). The relationship between patterns of alcohol use and mental and physical health disorders in two American Indian populations. *Addiction*, 101, 69-83.
- Oetzel, J., & Duran, B. (2004). Intimate partner violence in American Indian and/or Alaska Native communities: A social ecological framework of determinants and interventions. *American Indian and Alaska Native Mental Health Research*, 11(3), 49-68.
- Preuss, U.W., Schuckit, M.A., Smith, T.L., Barnow, S., & Danko, G.P. (2002). Mood and anxiety symptoms among 140 children from alcoholic and control families. *Drug and Alcohol Dependence*, 67, 235-242.
- Ramsay, J., Richardson, J., Carter, Y.H., Davidson, L.L., & Feder, G. (2002). Should health professionals screen women for domestic violence? Systematic review. *BMJ*, 325, 315- 318.
- Richardson, D. (2005). The myth of female passivity: Thirty years of revelations about female aggression. *Psychology of Women Quarterly*, 29, 238-247.

- Roberts, G.L., Williams, G.M., Lawrence, J.M., & Raphael, B. (1998). How does domestic violence affect women's mental health? *Women & Health*, 28, 117-129.
- Rosenberg, M., & Fenley, M.A. (1991). *Violence in America: A public health approach*. New York: Oxford University Press.
- Schuckit, M.A. Smith, T.L., Barnow, S., Preuss, U., Luczak, S., & Radzinski, S. (2003). Correlates of externalizing symptoms in children from families of alcoholics and controls. *Alcohol*, 38, 559-567.
- Silverman, J. G., Raj, A., Mucci, L.A., & Hathaway, J.E. (2001). Dating violence against adolescent girls and associated substance use, unhealthy weight control, sexual risk behavior, pregnancy, and suicidality. *Journal of the American Medical Association*, 286, 572-579.
- Stith, S.M., Smith, D.B. Penn, C.E., Ward, D.B., & Tritt, D. (2004). Intimate partner physical abuse perpetration and victimization risk factors: A meta-analytic review. *Aggression and Violent Behavior*, 10, 65-98.
- Straus, M.A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics Scales. *Journal of Marriage and the Family*, 7, 75-88.
- United States Commission on Civil Rights. (2000). *The health care challenge: Acknowledging disparity, confronting discrimination, and ensuring equality. Volume I: The role of government*. Washington, DC: Author.
- United States Commission on Civil Rights. (2004). *Broken promises: Evaluating the Native American health care system*. Retrieved February 2, 2009 from <http://purl.access.gpo.gov/GPO/LPS69594>
- Wagner, P.J., Mongan, P., Hamrick, D., & Hendrick, L.K. (1995). Experience of abuse in primary care patients: Racial and rural differences. *Archives of Family Medicine*, 4, 956-962.
- Wells, K., Klap, R., Koike, A., & Sherbourne, C. (2001). Ethnic disparities in unmet need for alcoholism, drug abuse, and mental health care. *American Journal of Psychiatry*, 158, 2027-2032.
- Whitesell, N.R, Beals, J., Mitchell, C.M., Novins, D.K., Spicer, P., & Manson, S.M. (2006). Latent class analysis of substance use: Comparison of two American Indian reservation populations and a national sample. *Journal of Studies on Alcohol*, 67, 32-43.
- Widom, C.S. (1999). Posttraumatic stress disorder in abused and neglected children grown up. *American Journal of Psychiatry*, 156, 1223-1229.
- Wittchen, H. (1994). Reliability and validity studies of the WHO Composite International Diagnostic Interview (CIDI): A review process. *Journal of Psychiatric Research*, 28, 57-84.
- Wittchen, H., Robins, L.N., Cottler, L.B., Sartorius, N., Burke, J.D., & Regier, D. (1991). Cross-cultural feasibility, reliability, and sources of variance of the Composite International Diagnostic Interview (CIDI): The multicentre WHO/ADAMHA field trials. *British Journal of Psychiatry*, 159, 645-658.

Woods, S.J. (2000). Prevalence and patterns of posttraumatic stress disorder in abused and postabused women. *Issues in Mental Health Nursing*, 21, 309-324.

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THE EFFECT OF NEAR-MISS RATE AND CARD CONTROL WHEN AMERICAN INDIANS AND NON-INDIANS GAMBLE IN A LABORATORY SITUATION: THE INFLUENCE OF ALCOHOL

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Abstract: Twelve American Indian (AI) and 12 non-AI participants gambled on a slot-machine simulation and on video poker. Prior to the gambling sessions, half of the participants consumed alcohol while the other half consumed a placebo beverage. They then played the slot-machine simulation three times, with the percentage of programmed “near misses” varying across sessions. They also played video poker three times, with the control the players had over holding and discarding cards varying across sessions. Results showed that AI participants played significantly fewer poker hands than did non-AIs and that participants played most when they had the least control over what cards were played. No significant effect of alcohol consumption was observed. Likewise, results failed to show a significant effect of the percentage of near misses when participants played the slot-machine simulation. The present results lend support for the idea that the differences in gambling problems between AI and non-AI reported in the literature are not a function of ethnicity per se. They may also suggest that providing video poker players with accurate information may have the unintended effect of increasing the rate at which they gamble.

Over the past several decades there has been a large expansion of gambling in the United States, at least partially driven by the Indian Gaming Regulatory Act (Wardman, el-Guebaly, & Hodgins, 2001). With this expansion has come increased concern about problem gambling and the factors that lead to it. “Pathological gambling” became an official impulse-control disorder in 1980 (American Psychiatric Association, 1980). Although prevalence rates are difficult to accurately determine, Petry (2005) estimated that 1-3% of the population suffers from pathological gambling.

Importantly, the prevalence of pathological gambling is not equal across different populations. Pertinent to the current investigation, research indicates that American Indians (AIs) suffer from pathological gambling at a much higher frequency than non-AIs. Raylu and Oei (2002), for instance, reviewed numerous studies on the relationship between ethnicity and/or Indigenous cultures and gambling. They reported that results consistently demonstrate a higher rate of pathological gambling among Indigenous populations than in the majority culture. This conclusion was consistent with the findings of Volberg and Abbott (1997), who interviewed a random sample of AI and Caucasian residents of North Dakota about gambling behavior. AIs reported gambling more frequently than Caucasians and also reported spending more money on certain forms of gambling. Wardman et al. (2001) reported that rates of pathological gambling in Indigenous populations were 2.2 to 15.69 times higher than in non-Indigenous populations.

There are many potential reasons why AIs may display heightened rates of pathological gambling. One possibility is that ethnicity (e.g., genetics) is directly related to the disorder. A second possibility is that cultural factors, such as differences in beliefs and norms, may contribute to gambling problems by influencing individuals' gambling patterns (see Raylu & Oei, 2004). Another possibility is that mediating factors (e.g., socioeconomic status, drug use) related to both ethnic minority status and pathological gambling account for the increased prevalence (see Petry, 2005).

Recent results from our laboratory would appear to be consistent with the last of these possibilities. McDougall, McDonald, and Weatherly (2008) had non-pathological AI and non-AI participants play a slot-machine simulation in the presence or absence of an AI or non-AI confederate. No significant differences were observed in the gambling behavior of the AI and non-AI participants, nor were there significant differences in how they were influenced by the presence or actions of the confederate. Likewise, Gillis, McDonald, and Weatherly (2008) studied the gambling behavior of AI and non-AI participants who were high or low sensation seekers. Participants played a slot-machine simulation in three different sessions, across which the simulation paid out at three different rates. Again, no differences in gambling behavior were observed between AI and non-AI participants. It is important to note, however, that the findings of both of these studies represent null results and therefore cannot conclusively eliminate the possibility that ethnicity and/or cultural factors influence gambling behavior.

It is certainly possible to identify potential mediators that may be responsible for the heightened rates of pathological gambling in the AI population. One possibility is drug use. According to Petry (2005), drug use is largest risk factor for pathological gambling. Specifically, results from LaBrie, Shaffer, LaPlante, and Wechsler (2003) suggest that alcohol use is strongly tied to problem gambling. Similarly, Welte, Barnes, Wieczorek, Tidwell, and Parker (2001) determined

that drinkers who consumed an average of two ounces or more of alcohol daily were five times as likely as abstainers to be problem or pathological gamblers. This factor is relevant because research indicates that AIs display heightened rates of substance abuse relative to the majority population (e.g., see McDonald & Chaney, 2003).

AI and non-AI cultures may also differ in how individual members interpret environmental events and/or view themselves in relationship to the environment (e.g., see Raylu & Oei, 2004). These differences may be important because research on gambling has indicated that aspects of games of chance and/or the player-game interaction can influence how people gamble. For example, persistence of play on a slot machine has been shown to differ as a function of the frequency of near misses (i.e., winning symbols in all but one of the necessary positions and just above or below the remaining one). Kassinove and Schare (2001) showed that participants who experienced near misses on 30% of the trials persisted longer in playing than those who experienced near misses on 15% or 45% of trials. The authors suggested that 30% was apparently the “optimal range.”

Others have found that dispositional factors, such as the illusion of control (Langer, 1975), can influence gambling behavior. The illusion of control occurs when active participation enhances one’s sense of having control over the outcome when, in fact, such participation has no influence over the result. Dixon, Hayes, and Ebbs (1998), for instance, found that participants were willing to purchase the opportunity to place their own bets when playing roulette rather than have the researcher choose the bet for free. Similarly, Dixon (2000) found that participants wagered more chips when they had control over the numbers bet than when the experimenter controlled bet placement. However, when studying video-poker play, Dannewitz and Weatherly (2007) found that participants played more hands and bet more credits when the game chose the cards that would be “held” versus when the player chose the cards.

Thus, it is possible that the differences in gambling behavior between AIs and non-AIs reported in literature (e.g., Wardman et al., 2001) represent ethnic differences. However, the differences may be related to other factors such as alcohol use, how individuals persist in the face of situations such as near misses, or how they display the illusion of control. The present study was an initial attempt to assess these possibilities. AI and non-AI participants were recruited to participate. Half of the participants consumed alcohol whereas the other half consumed a placebo beverage. All participants then played a slot-machine simulation three different times, with the simulation differing across periods in terms of the percentage of near misses that were programmed. They also played video poker three different times, with the game differing across periods in how much control players had over which cards were played.

Given that previous experimental research from our laboratory (e.g., Gillis et al., 2008; McDougall et al., 2008) failed to demonstrate differences in the gambling of AI and non-AI participants, we were not optimistic that a main effect of ethnicity would be observed in the present study. Rather, because the literature suggests that alcohol use is related to heightened levels of gambling (e.g., LaBrie et al., 2003), we predicted that participants who consumed alcohol would display heightened levels of gambling relative to participants who did not consume alcohol. Consistent with Kassinove and Schare (2001), we predicted that participants would gamble most on the slot-machine simulation when 33% of the outcomes were near misses (vs. 0 or 67%). Consistent with Dannewitz and Weatherly (2007), we predicted that participants would gamble most on the video-poker game when the game, rather than the player, chose which cards would be held or discarded.

METHOD

Participants

Twenty-four participants were recruited through the Psychology Department participant pool and advertisements on a local public-access television channel. All participants were males¹ 21 years of age or older. Twelve were of AI descent and 12 were of non-AI descent, both determined by self report. Participants were non-pathological gamblers, as determined by scores on the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987). They also did not abuse alcohol, as determined by scores on the Khavari Alcohol Test (KAT; Khavari & Farber, 1978) and the Michigan Alcoholism Screening Test (MAST; Selzer, 1971). Participants were in good health when the experimental sessions were conducted, as determined by scores on a medical questionnaire.

Materials

Participants were asked to complete a series of paper-and-pencil measures. The first of these was the SOGS (Lesieur & Blume, 1987). The SOGS is a 20-item questionnaire that asks respondents about their gambling history. It is the most widely used screening measure for gambling problems (see Petry, 2005), with a score of 5 or more indicating the potential presence of pathology. Research indicates that the SOGS has adequate internal consistency (Stinchfield, 2003) and good reliability (Lesieur & Blume, 1987).

The second measure was the KAT (Khavari & Farber, 1978). The KAT consists of three different sets of three items. The first set addresses the frequency of drinking beer, wine, and liquor. The second set addresses the average number of drinks of beer, wine, and liquor the person has at

one sitting. The third set addresses the maximum number of beers, glasses of wine, or drinks with liquor consumed at one time. The cutoffs for these values were set according to diagnostic criteria for alcohol abuse (Khavari & Farber, 1978).

The third measure was the MAST (Selzer, 1971), which consists of 25 yes/no statements that address the participant's drinking habits and history. Item responses are weighted with a specific point value. A combined score of 16 or greater is indicative of an alcohol abuse problem. The MAST has been shown to identify individuals with a history of drunk and disorderly conduct as well as driving under the influence of alcohol (Selzer, 1971).

The fourth measure was a brief medical questionnaire created for the present study. The questionnaire was employed to rule out current illness. It asked if the participant had been diagnosed with any chronic medical conditions, such as heart disease or diabetes, as well as whether he was taking any prescription or over-the-counter medication at the time. In addition to ruling out illness, the medical questionnaire asked if the participant had ever undergone treatment for addiction, had received a DUI within the past two years, or had consumed any illicit drugs during the past month. These questions were used to rule out participants who may have had a history of substance abuse. The researcher reviewed the sheet to ensure no prescription medicines and no over-the-counter cold medicines were currently being taken.

Apparatus

Experimental sessions were conducted in a windowless room that measured approximately 1.5 m by 1.5 m. The room contained two small desks, each equipped with an IBM-compatible computer. One computer was used to collect data with the slot-machine simulation customized from MacLin, Dixon, and Hayes (1999). The simulation differed from the original in that the outcomes of the individual trials could be predetermined by the researcher. The other computer was used to collect data with the video-poker game, which was a 5-card-draw poker game by Zamzow (2003). The software allowed a variety of games to be played. The present study used only Jacks-or-Better poker. The software allowed the researcher to program whether the game indicated to the player which cards should be held or discarded each hand.

Blood alcohol levels (BAL) were determined by using a breathalyzer (Alcomonitor CC Series 02.XX; Intoximeters Inc., St. Louis, MO). The breathalyzer was located in a room directly adjacent to the one housing the computers. This adjacent room was approximately 5 m by 5 m and had two windows and several tables. Participants completed the paper-and-pencil tasks and consumed the alcoholic or placebo beverages in this room before going to the other room for the gambling session.

Procedure

All the procedures were reviewed and approved by the Institutional Review Board at the University of North Dakota. Participants were run individually. When the participant entered the room, the researcher checked his identification to ensure he was at least 21 years of age. The researcher then initiated the process of receiving informed consent. The participant granted informed consent and completed the paper-and-pencil measures. Once the participant had completed the SOGS, KAT, MAST, and medical questionnaire, the researcher scored these measures to determine whether the participant qualified to continue. One participant was dismissed due to an elevated SOGS score of 5. He was replaced by an additional participant.

The researcher then read the participant a prepared script informing him about the procedure of the study. The researcher then weighed the participant and subsequently mixed the beverage the participant would consume. The drink was a mixture of 0.66 ml/kg dose of either 95% ethanol or water mixed with soda in a 1:5 dose/soda ratio. This mixture was divided equally into three separate cups; the participant was required to consume the contents of one cup every 5 min. Breathalyzer readings were taken every 5 min following the final consumption period until the participant exhibited a second consecutive drop in BAL. Those participants who did not receive alcohol were yoked with other participants who did receive alcohol regarding the time interval between drink consumption and the start of the gambling sessions. The dose of alcohol administered to participants was chosen because research (e.g., Davidson, Carnara, & Swift, 1997) suggests that a dose of at least 40 ml/kg is necessary for participants to discriminate receiving alcohol and to report positive subjective effects of its administration.

Before the start of the gambling sessions, the researcher read the participants the following instructions:

You will be given the opportunity to play two different computer simulations. One simulation will be a computer-simulated slot machine. This slot machine is programmed identical to those found in actual gaming establishments. Each possible winning outcome is scheduled at a constant probability and each individual play is independent of the previous play. Before you begin this session, you will be given instructions on how the program works and the payoff table. The other simulation will be a 5-card-draw video poker game. This game is also programmed identical to those found in actual gaming establishments. Each possible winning outcome is scheduled at a constant probability and each individual play is independent of the previous play.

Each of these 15-minute sessions will be divided into three 5-minute periods. For each period, you will be staked with 100 credits. Each credit is worth \$0.05. This means that after you receive instructions on how to play the game, you will be given 5 minutes to play. When those 5 minutes are up, or you reach 0 credits, the researcher will reset the game with 100 credits and you are able to play again for another 5 minutes with those 100 credits.

The participant then completed a total of six 5-min gambling sessions. In three of the six sessions, participants played the customized version of the MacLin et al. (1999) slot-machine simulation. These sessions were identical with the exception of the rate at which near misses occurred during losses (0, 33, or 67%). Near misses were defined as winning symbols falling on the first two positions on the win line but not in the third position. Prior to the first slot-machine session, the researcher read the participant the following directions:

You are able to control the amount bet by clicking on either the “Bet 1 Credit” button or by clicking the “Bet Max Credits” button. The maximum number of credits that can be wagered on one spin is 5 credits. After selecting a wager amount, click the “Spin” button. The payout table is located directly to your right.

In the remaining three sessions the participants played video poker (Zamzow, 2003). Participants played Jacks-or-Better 5-card draw in each session. In one, the participant was not provided information on which cards to hold or discard. In another session, the “autohold” feature was enabled, which indicated to the participant which cards should be held to maximize his chances of winning (or minimize his chances of losing). However, the participant was not required to follow the supplied advice. In the third session, the autohold feature was enabled and the participants were required to play as instructed by the software. Prior to the first video-poker session, the researcher read the participants the following instructions:

You are able to control the amount bet by clicking on either the “Bet 1 Credit” button or by clicking the “Bet Max Credits” button. The maximum number of credits that can be wagered on one hand is 5 credits. When you are satisfied with the wager amount, click the “Deal/Draw” button. Depending on the condition, you may be able to choose which cards are held. You can do this by clicking on the cards you would like to hold or clicking the hold button directly beneath the card. Do you have any questions?

The order participants experienced these six 5-min sessions was counterbalanced. At the end of the gambling sessions, all participants were debriefed, paid cash for the number of credits they had accumulated in the gambling sessions, and given extra credit for their psychology course

(if applicable). Participants who consumed the placebo beverage were then dismissed. Those who had consumed alcohol remained under supervision until their BAL had reached 0.02, at which time they were dismissed.

Dependent Measures

There were two main dependent measures for the slot-machine sessions. The first was the total number of trials played, which can be taken as a measure of rate and persistence. The second was the total amount bet, which can be taken as a measure of risk. Ideally, the same two measures would be analyzed for the video-poker session. However, due to a recording error, the total number of credits bet per session was not recorded for enough participants to allow analyses to be conducted on this measure. Analyses were therefore conducted on the total number of credits remaining each session, which is an indirect, but imperfect, measure of how much participants risked during the session. Due to a wide range of variance across participants, a square-root transformation was completed on all data. A square-root transformation was chosen because this transformation is recommended for ratio-scale data, which the present data represent.

RESULTS

An independent samples *t*-test showed that AI participants were significantly older than non-AI participants, $t(22) = 4.646$, $p < 0.001$. There was no significant difference in age between those participants who received alcohol and those who received the placebo, $t(22) = 0.655$. Results from these, and all the following, analyses were considered significant at $p < .05$. Because of the significant difference in age, age was used as a covariate in preliminary analyses. However, age never accounted for a significant amount of variance in any analysis and was therefore dropped from the analyses reported below.

Each participant began the gambling sessions after the second consecutive drop in BAL or at the assigned time if placebo was received. Those who received alcohol had an average BAL of 0.071 at the start of the gambling sessions. Those who received placebo had a BAL of 0.00.

Slot-Machine Gambling

The number of trials played by individual participants was analyzed by conducting a three-way (Ethnicity by Drink by Percentage of Near Misses) mixed-model analysis of variance (ANOVA) on the transformed data. Ethnicity (AI or non-AI) and drink (alcohol or placebo) served as between-subject variables. Percentage of near misses (0, 33, and 67%) served as the repeated measure. The main effect of ethnicity, $F(1, 20) = 1.75$, $p = 0.201$, $\eta^2 = 0.081$, drink, $F < 1$, $\eta^2 =$

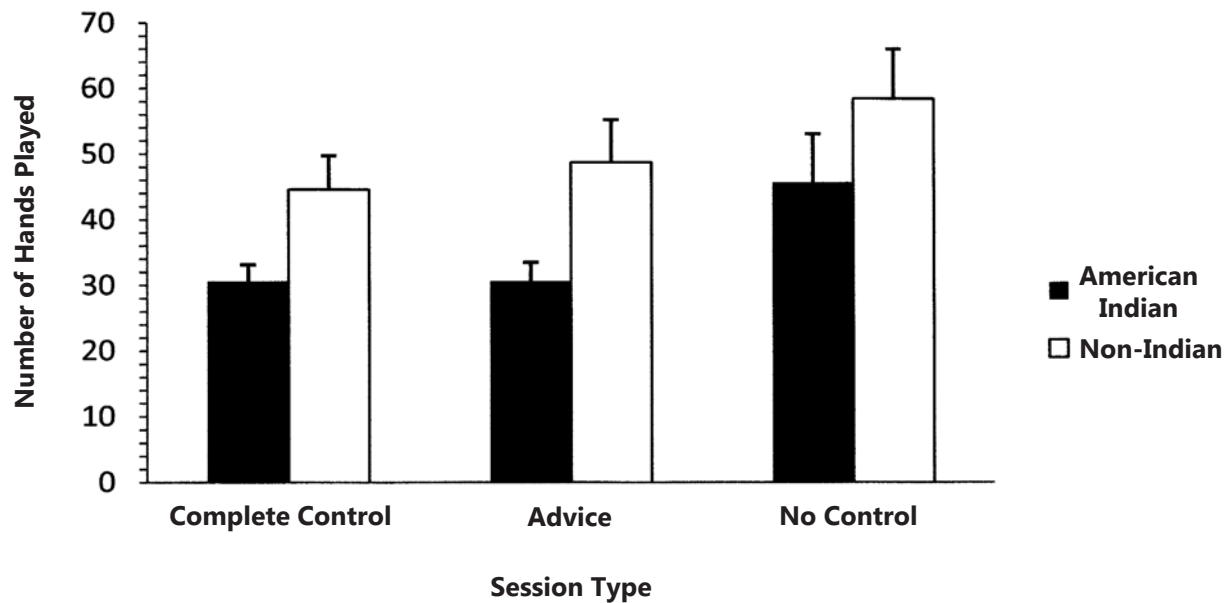
0.000, and percentage of near misses, $F < 1$, $\eta^2 = 0.034$, each failed to reach statistical significance. The interactions between ethnicity and drink, $F < 1$, $\eta^2 = 0.021$, between ethnicity and percentage of near misses, $F(2, 40) = 2.51$, $p = 0.094$, $\eta^2 = 0.111$, between drink and percentage of near misses, $F < 1$, $\eta^2 = 0.034$, and the three-way interaction, $F < 1$, $\eta^2 = 0.007$, also each failed to reach statistical significance. Together, these results indicate that none of the independent variables significantly altered the number of times participants played the slot-machine simulation.

An identical analysis was conducted on the total number of credits bet per session. That analysis indicated that the main effect of ethnicity, $F < 1$, $\eta^2 = 0.001$, drink, $F < 1$, $\eta^2 = 0.017$, and percentage of near misses, $F(2, 40) = 1.12$, $p = 0.336$, $\eta^2 = 0.053$, each failed to reach statistical significance. The interaction between ethnicity and drink, $F(1, 20) = 0.048$, $p = 0.829$, $\eta^2 = 0.002$, and between ethnicity and percentage of near misses, $F(2, 40) = 2.765$, $p = 0.075$, $\eta^2 = 0.121$, also failed to reach statistical significance. The interaction between drink and percentage of near misses was significant, $F(2, 40) = 3.856$, $p = 0.029$, $\eta^2 = 0.162$. However, tests of simple effects indicated that the differences were not significant between the alcohol and placebo groups at any percentage of near misses, all $F_s(1, 23) \leq 1.95$, $p > .177$, $\eta^2 < 0.081$. Furthermore, betting did not differ significantly across the three conditions for either participants who received a placebo, $F(2, 22) = 2.387$, $p = 0.115$, $\eta^2 = 0.178$, or an alcoholic beverage, $F(2, 22) = 2.399$, $p = 0.114$, $\eta^2 = 0.179$. Finally, the three-way interaction was not significant, $F < 1$, $\eta^2 = 0.006$.

Video-Poker Gambling

The number of trials played by individual participants during the three video-poker sessions was analyzed by conducting a three-way (Ethnicity by Drink by Control of Choice) mixed-model ANOVA on the transformed data. Ethnicity (AI or non-AI) and drink (alcohol or placebo) again served as between-subject variables. Control of choice (Complete Control, Advice, No Choice) served as the repeated measure. In this analysis, the main effect of ethnicity was significant, $F(1, 20) = 6.21$, $p = 0.022$, $\eta^2 = 0.237$. AI participants played significantly fewer trials than non-AI participants. The main effect of drink was not significant, $F < 1$, $\eta^2 = 0.010$, but the main effect of control of choice was significant, $F(2, 40) = 5.86$, $p = 0.006$, $\eta^2 = 0.227$. A *post hoc* Tukey HSD calculated by hand indicated that participants played more trials in the No Choice condition than in either the Complete Control or Advice sessions. The effect of ethnicity and control of choice can be seen in Figure 1. There was no significant interaction between the ethnicity and drink, $F < 1$, $\eta^2 = 0.043$, between ethnicity and control of choice, $F < 1$, $\eta^2 = 0.010$, or between drink and control of choice, $F(2, 40) = 2.83$, $p = 0.071$, $\eta^2 = 0.124$. The three-way interaction also failed to reach statistical significance, $F < 1$, $\eta^2 = 0.047$.

Figure 1
The number of Poker Hands Played Per 5-min Session by American Indian and Non-Indian Participants as a Function of Amount of Control Participants Had Over the Game



An identical analysis was conducted on the number of credits remaining at the end of each session. The main effect of ethnicity, $F < 1$, $\eta^2 = 0.012$, drink, $F(1, 20) = 1.69$, $p = 0.209$, $\eta^2 = 0.078$, and control of choice, $F < 1$, $\eta^2 = 0.002$, each failed to reach significance. The interactions between ethnicity and group, $F < 1$, $\eta^2 = 0.009$, ethnicity and control of choice, $F(2, 40) = 2.79$, $p = 0.074$, $\eta^2 = 0.122$, and drink and control of choice, $F < 1$, $\eta^2 = 0.016$, were not significant, nor was the three-way interaction, $F(2, 40) = 2.11$, $p = 0.134$, $\eta^2 = 0.096$.

DISCUSSION

Although the literature suggests that AIs suffer from pathological gambling at a greater frequency than non-AIs (e.g., Raylu & Oei, 2002), previous research from our laboratory (Gillis et al., 2008; McDougall et al., 2008) has failed to find significant differences in gambling between non-pathological AIs and non-AIs. We reasoned that the difference reported in the literature might be the outcome of some other factor(s), such as alcohol use or differences in responding to the gambling environment. These possibilities were tested in the present study by having AI and non-AIs consume an alcoholic or placebo beverage and then gamble on a slot-machine simulation across three sessions in which the percentage of near misses was varied and on video poker across three sessions in which player's control over the cards was varied. Overall, participants' gambling on

the slot machine was not influenced by ethnicity, type of beverage, or percentage of near misses. Participants' play on video poker, however, was significantly altered by both ethnicity and control over the game. Importantly, AI participants played fewer hands than did non-AI participants. Consistent with previous research from our laboratory (Dannewitz & Weatherly, 2007), participants also played the most when they had the least control over the game.

Unlike prior research from our laboratory, the present study produced a significant effect of ethnicity. This difference, observed for video-poker play, was in the opposite direction of the prevalence rates for pathological gambling in that AI participants played fewer hands of poker than non-AI participants. The present procedure included a number of features that could have potentially influenced the results, such as a relatively small sample size and 5-min sessions. Despite these features, a significant effect was still observed. Thus, the idea that the difference in gambling problems between AIs and non-AIs is due solely to ethnicity is not supported by the present results. It should be noted, however, that despite the fact that AI participants played fewer poker hands than did non-AI participants, they did not end the session with significantly more credits than did the non-AI participants.

The present study was designed to assess whether factors other than ethnicity would potentially influence gambling behavior. Alcohol use is linked to problem gambling (e.g., LaBrie et al., 2003) as well as with AI populations (e.g., McDonald & Chaney, 2003). Thus, we predicted that participants who drank alcohol would gamble more than those who did not. However, there was only one significant effect involving alcohol, an interaction between whether or not the participant received alcohol and the percentage of near misses programmed on the slot-machine simulation when amount wagered was the dependent measure. However, none of the subsequent *post hoc* tests revealed a significant effect of alcohol.

Several aspects of the present procedure may have limited the effect of alcohol consumption. One was the dosage level. The average participant peaked at a BAL below 0.08, which is the legal limit for intoxication in most states in the United States. An effect of alcohol might have been observed if higher doses had been administered and/or if we had recruited heavy drinkers. With that said, the observed BAL in the present study exceeded what prior research suggests can be discriminated by participants and that produces positive subjective effects (e.g., Davidson et al., 1997).

For the slot-machine simulation sessions, the percentage of near misses was varied across the sessions. Unlike the results reported by Kassinove and Schare (2001), no significant differences in the amount wagered or the number of trials played was observed as a function of the percentage of near misses. This failure may be the outcome of the short sessions that were employed in the present study. However, this possibility must be interpreted in light of the fact that a significant

effect of number of hands played was observed when participants played video poker in sessions of the same length. The different outcomes quite likely can be traced to other procedural differences. Kassinove and Schare had participants play a set number of trials and then measured how long they continued to play when they could freely choose to proceed or quit. The present procedure measured how much participants would play and bet from the beginning of their experience with the different percentages of near-miss outcomes.

For the different poker sessions, participants were provided with differing levels of perceived control to determine if it had an effect on gaming behavior. Participants played more hands when they had the least control over the game, a result that replicates previous research (Dannewitz & Weatherly, 2007). This result is likely the outcome of a decrease in decision-making time when the game automatically informs the player of the decision that needs to be made. However, finding an increase in gambling when this function is presented is of importance because, intuitively, one might think that providing the gambler with accurate information might safeguard him/her. Although doing so may indeed help maximize the player's chances of winning, one must weigh that against the possibility that it may also increase how rapidly the person gambles.

In closing, there are several limitations that should be noted regarding this research. First, data were collected from a relatively small area on a Midwestern university campus. Although not all of the participants were students, they all resided in the surrounding area. Also, blood-quantum level was not obtained for AI participants. This fact may be important because it has been hypothesized (Raylu & Oei, 2004) that AIs who are more traditional possess more risk factors which are correlated with problem gambling behavior. Blood-quantum level is one way of measuring the ancestry of an individual, so one could argue that those with higher blood-quantum levels would be raised in a more traditional manner. In addition, this research did not document the specific tribes to which AI participants belonged, which may limit the scope of AIs to which the results could be applied. Future research should specifically target demographic information such as educational level, socioeconomic status, and marital status. Such factors have been documented as risk factors for pathological gambling (Petry, 2005) and could aid in interpretation of the results of studies like the present one. Unfortunately, such data were not collected for the present study; thus, it is not possible to determine whether additional conclusions could have been drawn with this information.

The present study also suffered from relatively low power. Prior studies that have suggested differences in gambling behavior between AIs and non-AIs (e.g., Raylu & Oei, 2004; Volberg & Abbott, 1997; Wardman et al., 2001) did not report effect size. When determining the number of necessary participants, we therefore relied on previous work from our laboratory, which had found moderate effect sizes (e.g., Weatherly & Meier, 2007; Weatherly, Austin, & Farwell, 2007). Unfortunately, however, we did not ultimately observe moderate effect sizes and thus the design

suffered from low power across many of the analyses, suggesting that future research should utilize a greater number of participants than used in the present study. With that said, a *post hoc* analysis run with the same statistical program indicated that the present study would have required 180 participants to detect significant differences with a medium effect size. Given the present procedure, such a number is rather excessive.

Lastly, the present design had participants gamble across six relatively short (i.e., 5-min) sessions; therefore, one could legitimately argue that our measure of persistence in gambling was constrained. Such short sessions were employed to try to ensure that participants who had consumed alcohol were under the influence of alcohol during all six sessions. It is possible that significant effects would have been observed had we conducted sessions that were longer than 5 min. Future research that investigates the influence of alcohol on gambling might benefit from fewer independent variables (e.g., near misses or card control, but not both) and longer sessions at each level of the independent variable.

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REFERENCES

- American Psychiatric Association (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, D.C.: Author.
- Dannewitz, H., & Weatherly, J.N. (2007). Investigating the illusion of control in mildly depressed and nondepressed individuals during video-poker play. *The Journal of Psychology*, 141, 307-319.
- Davidson, D., Carnara, P., & Swift, R. (1997). Behavioral effects and pharmacokinetics of low-dose intravenous alcohol in humans. *Alcoholism: Clinical and Experimental Research*, 21, 1294-1299.
- Dixon, M.R. (2000). Manipulating the illusion of control: Variations in gambling as a function of perceived control over chance outcomes. *Psychological Record*, 50, 705-719.
- Dixon, M.R., Hayes, L.J., & Ebbs, R.E. (1998). Engaging in "illusory control" during repeated risk-taking. *Psychological Reports*, 83, 959-962.

- Gillis, A., McDonald, J.D., & Weatherly, J.N. (2008). American Indians and non-Indians playing a slot-machine simulation: Effects of sensation seeking and payback percentage. *American Indian and Alaska Native Mental Health Research*, 15, 18-32.
- Kassinove, J.I. and Schare, M.L. (2001). Effects of the “near miss” and the “big win” on persistence at slot machine gambling. *Psychology of Addictive Behaviors*, 15(2), 155-158.
- Khavari, K. A., & Farber, P. D. (1978). A profile instrument for the quantification and assessment of alcohol consumption: The Khavari Alcohol Test. *Journal of Studies on Alcohol*, 39(9), 1525-1538.
- LaBrie, R.A., Shaffer, H.J., LaPlante, D.A., & Wechsler, H. (2003). Correlates of college student gambling in the United States. *Journal of American College Health*, 52, 53-62.
- Langer, E.J. (1975). The illusion of control. *Journal of Personality and Social Psychology*, 32, 311-328.
- Lesieur, H., & Blume, S.B. (1987). The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. *American Journal of Psychiatry*, 144, 1184-1188.
- MacLin, O.H., Dixon, M.R., and Hayes, L.J. (1999). A computerized slot machine simulation to investigate the variables involved in gambling behavior. *Behavior Research Methods, Instruments, & Computers*, 31(4), 731-734.
- McDonald, J.D, & Chaney, J. (2003). Resistance to multiculturalism: The “Indian problem.” In J. Mio & G. Iwamasa (Eds.) *Culturally diverse mental health: The challenges of research and resistance*. New York: Brunner-Routledge.
- McDougall, C.L., McDonald, J.D., & Weatherly, J.N. (2008). The gambling behavior of American Indian and non-Indian participants: Effects of the actions and ethnicity of a confederate. *American Indian and Alaska Native Mental Health Research*, 14, 59-74.
- Petry, N.M. (2005). *Pathological gambling: Etiology, comorbidity, and treatment*. Washington, D.C.: American Psychological Association.
- Raylu, N. and Oei, T.P.S. (2002). Pathological gambling: A comprehensive review. *Clinical Psychology Review*, 22, 1009-1061.
- Raylu, N., & Oei, T.P.S. (2004). Role of culture in gambling and problem gambling. *Clinical Psychology Review*, 23, 1087-1114.
- Selzer, M. L. (1971). The Michigan Alcoholism Screening Test: A quest for a new diagnostic instrument. *American Journal of Psychiatry*, 127, 1653-1658.
- Stinchfield, R. (2003). Reliability, validity, and classification accuracy of a measure of DSM-IV diagnostic criteria for pathological gambling. *American Journal of Psychiatry*, 160, 180-182.
- Volberg, R.A., & Abbott, M.W. (1997). Gambling and problem gambling among indigenous peoples. *Substance Use & Misuse*, 32, 1525-1538.

- Wardman, D., el-Guebaly, N., and Hodgins, D. (2001). Problem and pathological gambling in North American aboriginal populations: A review of the empirical literature. *Journal of Gambling Studies*, 17(2), 81-100.
- Weatherly, J.N., Austin, D.P., & Farwell, K. (2007). The role of prior experience when people gamble on three different video-poker games. *Analysis of Gambling Behavior*, 1, 34-43.
- Weatherly, J.N., & Meier, E. (2007). Studying gambling behavior experimentally: The value of money. *Analysis of Gambling Behavior*, 1, 133-140.
- Welte, J., Barnes, G., Wieczorek, W., Tidwell, M.C., & Parker, J. (2001). Alcohol and gambling pathology among U.S. adults: Prevalence, demographic patterns and comorbidity. *Journal of Studies on Alcohol*, 62, 706-712.
- Zamzow Software Solutions (2003). *WinPoker 6.0 [Computer Software]*. Fountain Hills, AZ.

FOOTNOTE

¹Only males were recruited for participation because the literature indicates that males, rather than females, are more likely to display gambling problems. Furthermore, introducing gender as a pseudo-independent variable would have decreased our ability to detect differences in the variables of interest in the present study. Future research will need to determine whether the results of the present study can be generalized to the gambling behavior of females.

AUTHORS' NOTE

The present study was completed by the first author in partial fulfillment of the requirements for a Master's degree.