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Familial influence on offspring gambling: a cognitive mechanism for transmission of gambling behavior in families

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ABSTRACT

Background. The problem-gambling literature has identified a range of individual, cognitive, behavioral and emotional factors as playing important roles in the development, maintenance and treatment of problem gambling. However, familial factors have often been neglected. The current study aims to investigate the possible influence of parental factors on offspring gambling behavior.

Method. A total of 189 families (546 individuals) completed several questionnaires including the South Oaks Gambling Screen (SOGS) and the Gambling Related Cognition Scale (GRCS). The relationships were examined using Pearson product-moment correlations and structural equation modeling (SEM) analyses.

Results. Results showed that generally parents' (especially fathers') gambling cognitions and gambling behaviors positively correlated with offspring gambling behaviors and cognitions. However, SEM analyses showed that although parental gambling behavior was directly related to offspring gambling behavior, parental cognitions were not related to offspring gambling behavior directly but indirectly via offspring cognitions.

Conclusion. The findings show that the influence of parental gambling cognition on offspring gambling behavior is indirect and via offspring cognitions. The results suggest a possible cognitive mechanism of transmission of gambling behavior in the family from one generation to the next.

INTRODUCTION

The role familial influences play in the development or maintenance of problem gambling (PG) has been viewed from two perspectives, that is, the genetics and the social learning perspective. Although the research on a genetic link is still in its early stages, several studies have shown a possible genetic link to PG. Several dysfunctional neurotransmitters (e.g. serotonin, noradrenaline and dopamine) have been associated with PG (Blanco *et al*. 2000; Raylu & Oei, 2002). Specific allele variants of genes related to these neurotransmitters have also been linked to PG (e.g. polymorphisms of dopamine receptor genes, the serotonin transporter gene and monoamine oxidase A gene – Comings *et al*. 1996; de Castro *et al*. 1997, 1999; Ibanez *et al*. 2003). The frequency of some of these alleles also varied with the severity of the gambling problem. Winters & Rich's (1998) twin study explored genetic influences on gambling behavior. They found that only for games that involved heavy player promotion and high payoffs, male monozygotic twins were similar in their frequency of gambling. Such a relationship for females was only found for gaming machines. Slutske *et al*.'s (2001) twin study aimed to investigate the contribution of genetic and environmental factors to the relationship between PG and antisocial behaviors, such as antisocial personality disorder, conduct disorders and antisocial behavior, and found a common genetic

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vulnerability between PG and antisocial behaviors among male PGs. A similar study reported a common genetic vulnerability for PG and alcohol dependence in males (Slutske *et al*. 2000).

The social learning perspective suggests that family members as well as friends can often act as significant models for gambling. Hardoon & Derevensky (2002) reviewed the studies conducted in this area (e.g. Gupta & Derevensky, 1997) and reported that 40–68% of youth report gambling with their families, while 55–82% report gambling with their friends. Lorenz & Shuttlesworth (1983) reported that 20% of PGs were raised in environments that included gambling problems. In a significant number of families, parents include children in various forms of gambling (e.g. cards or bingo), bet money together on the lottery, purchase lottery tickets for their children, ask children to purchase lottery tickets for them or receive lottery tickets as presents (Felsher *et al*. 2001; Ladouceur *et al*. 2001; Hardoon & Derevensky, 2002). Kearney & Drabman (1992) found that modeling or social learning increased the chances of risk taking/gambling-like behavior in children as young as 4–5 years old. Several studies have shown a link between parental gambling/PG and offspring gambling/PG (Lesieur & Heineman, 1988; Jacobs *et al*. 1989; Lesieur *et al*. 1991). The familial effect increases in significance with increased severity of PG, especially for males (Eisen *et al*. 1998; Walters, 2001).

The above evidence suggests that genetic transmissions as well as social learning of parents' gambling behaviors can explain the possible relationship between parental gambling/PG and offspring gambling/PG. The gambling literature has, however, shown that a wide range of factors apart from familial factors have been implicated as playing a role influencing gambling behaviors and the development and maintenance of PG, including individual, social, behavioral, emotional and cognitive factors (Raylu & Oei, 2002). In recent years, the role that gambling-related cognitions (GRC) play in the development and maintenance of PG have gained much attention in the treatment of PGs (Raylu & Oei, 2002, 2004). This is mainly because of recent studies and reviews that suggest that (*a*) regular and PGs tend to have significantly more GRC than non-PGs or non-gamblers, (*b*) presently behavioral, cognitive and combined cognitive behavioral therapies have the most outcome research and appear to be most effective at treating gambling problems (Blaszczynski & Silove, 1995; Raylu & Oei, 2002). Behavioral and cognitive behavioral therapy appears to have several advantages such as being cost-effective and allowing for booster sessions (Blaszczynski & Silove, 1995).

Research on GRC

Using a method called 'the thinking aloud method' (a technique where gamblers are asked to provide commentary/verbalizations on everything that is going on in their minds as the game is proceeding, including intentions, urges, ideas and images on their play), several researchers have identified the existence of GRC among frequent and PGs for a variety of games such as blackjack, gambling machines, roulette, lottery and sports betting (Gaboury & Ladouceur, 1989; Ladouceur *et al*. 1991; Coulombe *et al*. 1992; Walker, 1992). Some examples of these gambling-related thinking errors include belief that one could *control* gambling outcomes (e.g. believing that superstitious behaviors such as carrying a rabbit's foot will influence gambling outcomes), belief that one could *predict* gambling outcomes based on salient cues (e.g. the weather or hunches) or based on past wins/losses or *reframing* gambling outcomes that would encourage continued gambling (e.g. attributing successes to one's own skill, and failures to other's influences or luck) (Keren & Wagenaar, 1988; Toneatto *et al*. 1997; Toneatto, 1999; Raylu & Oei, 2004). Other types of cognitions that are less discussed in the gambling literature compared to the alcohol literature are gambling-related expectancies (Walters & Contri, 1998; Raylu & Oei, 2004) and perceived inability to stop gambling (Raylu & Oei, 2004).

Very few studies have looked at GRC in adolescents and children. However, the limited research in this area suggests a link between adolescent PGs and GRC similar to those reported for adult PGs (Fisher, 1993; Moore & Ohtsuka, 1999; Hardoon *et al*. 2001; Derevensky & Gupta, 2002). Existence of GRC have also been reported among children/pre-adolescents such as fourth- to eighth-graders (Frank & Smith, 1989; Derevensky *et al*. 1996; Herman *et al*. 1998). These studies suggest that cognitive errors may be acquired early in life.

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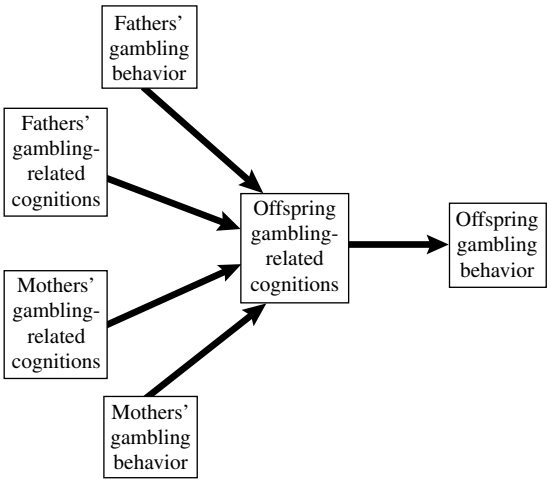

FIG. 1. Proposed relationships between parental and offspring variables.

The discussion above at least suggests that familial factors such as parents' gambling/gambling problems can increase the risk of gambling and PG in offspring. The gambling literature also suggests that GRC play a significant role in the development of PG for adults, adolescents and children. Thus, it is possible that parents' GRC and behavior influences gambling behavior via offspring GRC. The alcohol literature supports the role of cognitions as a mediator between parental alcohol use/problems and offspring alcohol use/problems (Zhang *et al*. 1997; Loveday & Oei, unpublished observations; Oei & Angel, unpublished observations). Thus, based on the above discussion and previous studies we propose to test the following relationships (Fig. 1).

METHOD

Subjects

In total, 189 family units (546 family members) were recruited though offspring who were first-year psychology students at the University of Queensland. While students were given course credit for participating in the study, participation in the study was voluntary. Although questionnaires were distributed to 200 families, only 193 were returned. Data for a further four family units were discarded due to significantly incomplete or missing data. Thus, analyses were carried out using data from the remaining 189 family units. Most of the family-unit data were gathered from both parents ($n = 166$), however, four family units had data from only the father, and 21 family units had data from only the mother. Thus, the resulting sample consisted of 189 child offspring, 170 fathers and 187 mothers. The total number of participants in the study was 546. Of the child offspring, 77·8% were females ($n = 148$) and 22·2% were ($n = 41$) males.

The family units consisted of approximately 82% Caucasians, 10% Chinese, 2·5% other Asian (including Korean, Vietnamese, etc.), 2·5% Indians and 3% other ethnic groups. All groups (mothers, fathers and children) had a South Oaks Gambling Screen (SOGS) score between 0 and 8. Mean age of children was 20·0 years (S.D. = 3·9) and ranged from 17 to 37 years. Mean age for mothers was 48·1 years (S.D. = 5·1), while the age range was 30–70 years. Mean age for fathers was 50·5 (5·5) years, while the age range was 35–73 years.

Measures

South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987)

The SOGS is a 20-item questionnaire based on DSM-III criteria to screen for pathological and PG. It has been used with patients in a therapeutic community (Lesieur & Heineman, 1988), psychiatric admissions (Lesieur & Blume, 1990) and numerous treatment settings as an aid in diagnostic and forensic screening (Rosenthal, 1989). It has been shown to have high validity (by cross-tabulating patients scores with counselors independent assessment scoring → $r = 0{\cdot}86$, $p < 0{\cdot}001$) as well as high internal consistency reliability (Cronbach's $\alpha = 0{\cdot}97$, $p < 0{\cdot}001$).

The Gambling Related Cognitions Scale (GRCS; Raylu & Oei, 2004)

The GRCS is a five-factor 23-item questionnaire. Items for the questionnaire were generated to cover the wide range of gambling-related cognitive errors that have been reported in the gambling literature. Consequently, the original 59-item questionnaire was developed to reflect a range of GRC that have been identified in previous studies (e.g. Toneatto *et al*. 1997; Toneatto, 1999; Gaboury & Ladouceur, 1989; Griffiths, 1994) and those cognitions identified in the general addiction literature that may also apply to PGs (e.g. gambling expectancies). Items were

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constructed by the authors using the examples and/or description of the various categories of GRC provided in previous studies in this area (e.g. Gaboury & Ladouceur, 1989; Griffiths, 1994; Toneatto *et al.* 1997; Toneatto, 1999). Some items (e.g. those reflecting gambling-related expectancies and perceived inability to stop gambling) were also constructed using examples of these specific types of cognitions provided in general addiction studies (e.g. Baldwin *et al.* 1993; Beck *et al.* 1993; Lee & Oei, 1993; Oei & Baldwin, 1994; Oei *et al.* 1998; Lee *et al.* 1999; Oei & Burrow, 2000). Three of the factors are consistent with the categories suggested by previous studies (Toneatto *et al.* 1997; Toneatto, 1999) including illusion of control (e.g. 'I have specific rituals and behaviors that increase my chances of winning'), predictive control (e.g. 'Losses when gambling, are bound to be followed by a series of wins') and interpretative bias (e.g. 'Relating my winnings to my skill and ability makes me continue gambling'). The other two categories were consistent to gambling-related expectancies (e.g. 'Having a gamble helps reduce tension and stress') and perceived inability to stop gambling (e.g. 'My desire to gamble is so overpowering') suggested by other researchers (e.g. Walters & Contri, 1998; Raylu & Oei, 2004). Confirmatory factor analysis (CFA) confirmed that the five-factor solution best fitted the data. Cronbach's α coefficients for the factors ranged from 0·77 to 0·91, and 0·93 for the overall scale. The GRCS also had significant positive correlations with other measures assessing gambling-related variables (i.e. concurrent validity). It could also significantly predict PG (i.e. predictive validity). Furthermore, it showed the ability to discriminate between non-PGs and PGs (i.e. criterion-related validity). The GRCS requires the participants to use a 7-point Likert scale (1 = strongly disagree, 2 = moderately disagree, 3 = mildly disagree, 4 = neither agree nor disagree, 5 = mildly agree, 6 = moderately agree, 7 = strongly agree) to indicate the extent to which they agreed with the value expressed in each statement. Scoring consisted of totaling the values such that the higher the score the higher the number of GRC displayed.

A short questionnaire pertaining to demographic information (e.g. gender, age, employment status, education level and ethnicity) was also included. The above measures used for this study are parts of a larger study examining the effectiveness of a group cognitive behavior therapy program for PGs funded externally by the Queensland government.

Procedure

All families were recruited through undergraduate psychology students. The participants were asked to complete the two measures relevant to the study as well as a range of other questionnaires for another study. The students received credit for participation. To reduce social desirability bias and remove any possible elements of coercion, none of the researchers of this study were part of the teaching faculty of the first-year psychology students. Those students that were interested in participating in the study had to first get permission from their parent(s) to participant in the study. At this stage, the parents were provided with an information sheet outlining the nature and requirements of the study. The information sheet also provided contact details of the researchers so that any questions and concerns about the study could be clarified directly with the researchers. The students were then given the questionnaires to complete (in the presence of an experimenter) only after permission was granted from parent(s). Once students handed in their completed questionnaires, each student was given two stamped addressed envelopes (those that had contact with only one parent or had only one living parent received only one stamped addressed envelope) so that the parent(s) could return their completed questionnaires. This ensured confidentiality of their responses. The response rate (% of questionnaires returned) of students was 98%, while for parents it was approximately 93%. The completion rate (% of returned questionnaires that were completely answered) for students was 98%, while the completion rate of the parents was 90%). Responses were confidential to the researchers and used identifying codes rather than names.

Data analyses

Minor missing data were found for at least five individuals and these were replaced with means. Means for each dependent variable was calculated to demonstrate the distribution of the dependent variables. Analyses of variance (ANOVA) analyses were conducted to compare

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Table 1. *Means (and standard deviations) for GRCS and SOGS scores of offspring and parents*

	Offspring (both males and females)	Male offspring only	Female offspring only	Mothers	Fathers
GRCS	39·05 (16·67)	42·07 (15·15)	38·21 (17·02)	34·17 (17·67)	40·22 (19·64)
SOGS	0·63 (1·26)	0·73 (1·40)	0·61 (1·23)	0·5 (1·34)	0·98 (1·16)

GRCS, Gambling Related Cognitions Scale; SOGS, South Oaks Gambling Screen.

the means of these dependent variables between fathers, mothers, offspring, male-only offspring and female-only offspring. Next, Pearson's correlation analyses were conducted to explore how the variables are related to one another (i.e. nature and strength). Finally, the structural equation modeling (SEM) program, AMOS 4.0 was used to assess the validity of the proposed model compared to alternative models. SEM is based on multivariate statistics and is used to evaluate the nature and size of the effect of one or more postulated causes on one or more postulated effects (Byrne, 2001).

Several goodness-of-fit indices were used to compare the fit of the model to a baseline model. χ^2 statistics were not used in this study because for large sample sizes, significant χ^2 statistics indicate a non-significant result suggesting that the model is not an acceptable fit to the data. Since non-significant χ^2 is difficult to achieve with large sample sizes, a range of other fitted indices was used. The AMOS program produces outputs for Bentler–Bonnet Normed Fit Index (NFI), and the Comparative Fit Index (CFI). These indices were used as they are relatively immune to variations in sample size and type of model (Marsh *et al*. 1988; Bentler, 1990). Values greater than 0·9 are generally accepted as indicating a good fit (Marsh, 1993). Root mean square error of approximation (RMSEA) was also used as an index of fit. The RMSEA is based on population error of approximation measures 'discrepancy per degree of freedom' (Joreskog & Sorbom, 1993, p. 124). A value of 0·05 or less is recognized as suggesting a close fit. However, values up to 0·08 are recognized as a reasonable error of approximation.

RESULTS

Preliminary exploration of data

Data was explored using Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA) to ensure all relevant statistical assumptions were met. The distributions of the GRCS and SOGS scores were all positively skewed. Thus, square-root transformations were performed on these variables so that assumptions of normality, linearity and homoscedasticity had been adequately met. Two outliers were detected using Mahalanobis distance criteria and these cases were excluded from the analyses. Mulitcollinearity was assessed using variance inflation factor (VIF) and all VIF values were below 10 and average VIF was 1·33 suggesting collinearity was not a problem for the model.

Summary of means

A one-way ANOVA showed that there was a significant difference between the four groups (i.e. fathers, mothers, male offspring and female offspring) in relation to mean GRCS scores [$F(3, 45) = 4{\cdot}12$, $p < 0{\cdot}01$]. Tukey's *post-hoc* tests showed that there was a mean difference for the parents, where the mean GRCS scores for fathers were significantly higher than for mothers. A one-way ANOVA showed that there was no significant difference between the four groups in relation to SOGS scores [$F(3, 545) = 2{\cdot}37$, N.S.]. These means of the dependent variables are displayed in Table 1.

Pearson's correlations

The Pearson's correlations (bivariate associations) for both the cognitive variables (offspring, mothers' and fathers' GRCS scores) and gambling behavior variables (offspring, mothers' and fathers' SOGS scores) were calculated and are displayed in Table 2. Bonferroni adjustments of 0·01 were used.

Several conclusions could be drawn from the bivariate correlation analyses. These can be summarized as follows. Significant moderate positive correlations were seen between each individual's GRCS scores and their SOGS scores. Significant moderate positive correlations were

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Table 2. *Pearson's correlations for GRCS and SOGS scores of offspring, mothers and fathers*

	GRCS (O)	SOGS (O)	GRCS (M)	SOGS (M)	GRCS (F)	SOGS (F)
GRCS (O)	1·00	0·27**	0·21**	−0·07	0·29**	0·29**
SOGS (O)		1·00	0·12	0·02	0·24**	0·74**
GRCS (M)			1·00	0·21**	0·31**	0·01
SOGS (M)				1·00	0·01	0·01
GRCS (F)					1·00	0·35**
SOGS (F)						1·00

GRCS, Gambling Related Cognitions Scale; SOGS, South Oaks Gambling Screen.
O, Offspring; M, mothers; F, fathers.
** $p<0·01$.

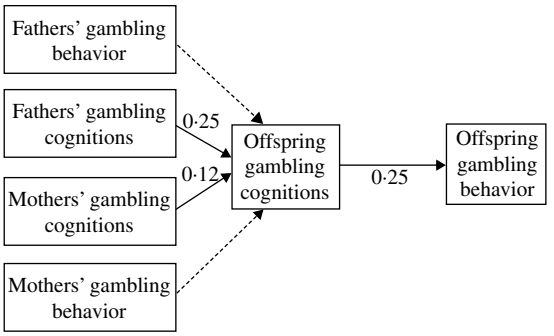

FIG. 2. Model A (indirect model) depicting familial influence indirectly influencing offspring gambling behavior via offspring gambling cognitions – including standardized regression pathways >0·0. (- - -, non-significant pathways). This model did not fit the data.

also established with offspring GRCS scores and both parents' GRCS scores. Although a high positive correlation was established between offspring SOGS scores and fathers' SOGS scores only. There was a significant moderate positive correlation between mothers' and fathers' GRCS scores but no such correlation was found between the SOGS scores of mothers and fathers.

SEM analyses

Initially two models were tested to evaluate the validity of the proposed hypothetical model. Model A (indirect model) that is presented in Fig. 1 tested whether the four parent variables indirectly influence offspring gambling behavior via offspring gambling cognitions (i.e. the hypothesized model). Model B (direct model) on the other hand, tested whether the four parent variables (both parents' gambling behavior and GRC) can directly influence offspring gambling behavior (see Fig. 3). This analysis was conducted to assess the validity of the direct pathways in contrast to the indirect ones of the proposed/hypothesized model. Results of the SEM would assist in distinguishing between the pathways that are valid and those that are not.

Model A (*indirect/hypothesized model*)

The resulting model showed a poor fit to the data as both fit indices that were below the accepted 0·9 level (CFI = 0·70; NFI = 0·68). Furthermore, the RMSEA value was greater than 0·08 (RMSEA = 0·31). Results showed that only certain pathways were significant for this model. It showed that the parents' GRC indirectly influence offspring gambling behaviors via offspring cognitions, however, this was not the case for parents' gambling behaviors. The tested indirect/hypothesized model highlighting the significant pathways, including the standardized regression paths of the model are displayed in Fig. 2.

Model B (*direct model*)

The resulting model showed an unacceptable fit to the data as both fit indices that were slightly below the accepted 0·9 level (CFI = 0·89; NFI = 0·86). Furthermore, the RMSEA value was below 0·08 (RMSEA = 0·20). Results showed that only certain pathways were significant in this model. It showed that the parents' gambling behaviors directly influence offspring gambling behaviors, however, this was not the case for parents' gambling cognitions. The tested direct model highlighting the significant pathways, including the standardized regression paths of the model are displayed in Fig. 3.

Post-hoc *model*

Since both Model A (indirect) and Model B (direct) were an unacceptable fit to the data, a third model (Model C) was tested which consisted of only the significant pathways from models A and B. That is, parents' GRC indirectly influence offspring gambling behavior via their cognitions, while parents' gambling behaviors influence gambling behavior directly (see Fig. 4). This Model C testing was completed just to indicate that *post-hoc* Model C could be confirmed by the SEM. The resulting model showed an acceptable fit to the data as both

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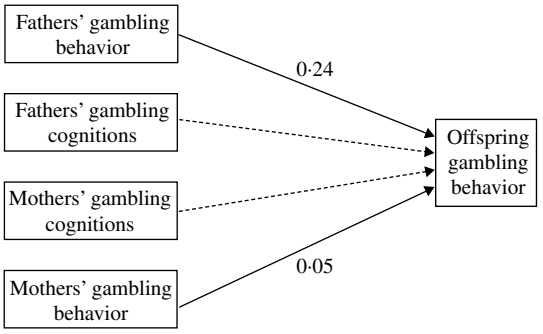

FIG. 3. Model B (direct model) depicting familial influence directly influencing offspring gambling behavior – including standardized regression pathways >0·0. (- - -, non-significant pathways). This model did not fit the data.

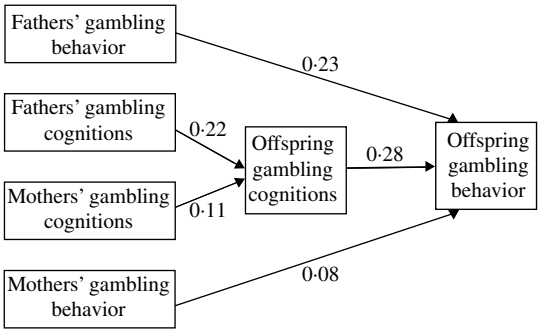

FIG. 4. Resulting model (including standardized regression pathways >0·0) depicting familial influence on offspring gambling-related cognitive errors and gambling behavior. This model best fitted the data.

fit indices that were above the accepted 0·9 level (CFI = 0·92; NFI = 0·91). Furthermore, the RMSEA value was an acceptable 0·08. These significant pathways including the standardized regression paths of Model C are displayed in Fig. 4.

DISCUSSION

The hypothesis that predicted that parents' cognitions and gambling behaviors will influence offspring gambling behaviors via offspring cognitions was only partially confirmed. Results of the study showed that parents' gambling variables (GRC and gambling behaviors) influenced offspring gambling behavior in two parts. Parents' GRC influenced offspring gambling behavior indirectly via offspring GRC, while parents' gambling behaviors influenced offspring gambling behavior directly.

The finding that parents' GRC is indirectly related to offspring gambling behavior via offspring GRC is consistent with earlier studies with other types of addictions such as alcohol (Zhang *et al.* 1997; Loveday & Oei, unpublished observations; Oei & Angel, unpublished observations). The influence of parental values, attitudes and beliefs on offspring's attitudes and beliefs is not a new finding. This has been demonstrated in many publications in the past for a range of factors including values, attitudes and beliefs towards food, weight, shape, diseases such as HIV, AIDS and cancer, occupational aspirations, parenting, sex, religion, smoking and risk-taking (Sigelman *et al.* 1995; Dittus *et al.* 1999; Flor & Knapp, 2001; Lundberg *et al.* 2002; Patel *et al.* 2002). What is new and important is that the present findings showed that parental cognitions also have an indirect contribution in a behavior such as gambling via offspring's cognitions. This adds to the literature, in particular drugs and alcohol literature, which as a whole maintains that the mechanisms of transfer of drug-taking behavior from parents to offspring are via behavior directly or by genetic mechanisms such as A1 dopamine. While data support that it is likely that genetic mechanisms are involved, this finding shows that other possible mechanisms now have to be considered. We suggest that this cognitive mechanism has more utility because cognitive variables are much easier and cheaper to change and thus make prevention and treatment more feasible and cheaper. Since this finding is still in its early stages, more data are needed to show that the cognitive mechanisms are, in fact, firmly supported.

Results showed that parents' gambling behavior influenced offspring gambling behavior directly. This could be attributed to children directly imitating their parents' behavior. This thus supports previous studies that have shown that children tend to gamble with their family members (Gupta & Derevensky, 1997; Hardoon & Derevensky, 2002).

The results also showed that fathers' GRC and gambling behaviors contributed more to offspring gambling behavior than did mothers'. This supports previous researches that have found a stronger familial link among male PGs than female PGs (e.g. Walters, 2001). Research is however, required to investigate this further.

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This study raises several clinical and prevention implications. Since GRC have a significant role in the development and maintenance of gambling problems and that parents can influence these offspring gambling behaviors via their cognitions, it is suggested that prevention can start at home by having early intervention education attempting to modify both parental and offspring's cognition, in particular gambling-related cognition. This suggestion is not new and is consistent with the literature. Although research such as this has not yet been carried out with children with GRC, it has been completed with children with alcohol-related (Kraus *et al*. 1994) and cigarette-related (Oei & Fae, 1987; Oei & Baldwin, 1992) cognitions. Any future prevention or intervention among children needs to take into account the continuing effects of family influences. Previous studies have shown that a significant number of PGs have reported being raised in environments that included gambling problems (Lorenz & Shuttlesworth, 1983; Jacobs *et al*. 1989; Lesieur *et al*. 1991). Thus, any early intervention needs to incorporate the whole family rather than the child in isolation (Oei & Baldwin, 1992).

There were several limitations in this study. First, since this study was cross-sectional in nature, one cannot make conclusions regarding changes in development and factors that influence as individuals age over time. It would, thus, be advantageous for future research to adopt a longitudinal approach to explore these variables. Second, each individual could not complete the questionnaires in controlled and appropriate experimental conditions (e.g. in environmental conditions monitored by the experimenter to ensure each participant completes the questionnaire). Future studies of this type need to take this into consideration. Third, Model C was constructed from significant pathways observed in models A and B. Given the fact that Model C was a *post-hoc* model, findings need to be interpreted with caution and thus, should only be taken as preliminary evidence. Its validity would still need to be confirmed using a new dataset.

Due to the small number of male children in the study, gender differences in familial influences on cognitions and behavior were not explored. The smaller number of male offspring was a reflection of the students enrolled in the first-year psychology course at the university (ratio of 3 : 1 of females to males). Further studies are needed to explore these gender differences using a more balanced sample. Although previous studies using the GRCS (Raylu & Oei, 2004) show that males tend to score higher than females, this study found that only fathers' GRCS scores were significantly higher than mothers'. Such gender differences were not found between male and female children. The small number of male children compared to female children could explain why a significant gender difference was not gained on total GRCS score. Finally, the current sample represented a community sample and thus, approximately 70% of participants were non-PGs. Walters' (2001) review of the familial studies in this area suggested that the familial effect increased in significance with increased severity of PG, especially for males. We remain hopeful that stronger relationships between parental and offspring behaviors will be obtained if clinical samples are used. Thus, the study needs to be replicated using a clinical sample to investigate the generalizability of the findings.

Although the findings of this study account for a percentage of variance in GRC and behavior, it is clear that other factors contribute to these variables. Consistent to social learning theory, other contextual factors including family environment, living arrangement (whether child was living at home or not), family history of gambling, sibling variables, peer gambling behaviors, and cultural values need to be investigated. The study also did not include genetic data on the subjects and thus, it is difficult to establish how much variability in offspring gambling behavior can be explained by genetic factors. Further studies need to conduct similar studies that include data on genetics.

Despite, these limitations it is important to note that this is the first study of its type in the gambling literature and the findings contribute significantly to understanding the role GRC may play in the transmission of gambling behavior from parents to children. The large sample size of this study strengthens these findings. Furthermore, the use of SEM confirms the model proposed.

In conclusion, the current study demonstrates that parents influenced offspring gambling behavior via offspring cognitions. The findings are

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important because this study is the first study to provide a possible cognitive mechanism of transmission of gambling behavior from one generation to the next. The findings have also both theoretical and clinical implications. It not only contributes to the understanding the development and maintenance of gambling problems but also the potential prevention and treatment of gambling problems.

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DECLARATION OF INTEREST

None.

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