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Investigating Possible Reciprocal Relationships between Depressive and Problem Gambling Symptoms in Emerging Adults

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Objective: Previous cross-sectional research has shown that depression and problem gambling co-occur. Longitudinal research, however, allows for a better determination of directionality, as behavioural changes in gambling involvement can be more reliability studied over time. The present study assesses symptoms of depression and problem gambling across four waves and addresses whether their relationship is directional (with one reliably preceding the other), bidirectional, or pathoplastic.

Method: As part of the Manitoba Longitudinal Study of Young Adults, prospective data was collected on Canadian young adults' (Wave 1: N = 679, 51.8% female, ages 18-20) depressive symptoms, involvement in gambling, and risky gambling behaviour. Recruitment and the first cycle of data collection (Wave 1) took place in fall 2007. Three additional waves of data collection then occurred in 12-18 month intervals: fall 2008, spring 2010, and spring 2011. The Problem Gambling Severity Index and the Composite International Diagnostic Interview – Short Form were administered via telephone interview at each wave.

Results: Bivariate growth curve analyses showed that depressive and problem gambling symptoms were positively correlated at Wave 1. Neither disorder was found to be a risk factor for the other, though, and depression and problem gambling were not pathoplastically related (increases in one did not result in increases in the other, and vice versa).

Conclusions: While depression and problem gambling are related, their co-occurrence may be better explained not by depressive- or gambling-related risk, but by the presence of a common underlying factor (like substance abuse).

Clinical Implications:

- Active screening for and treatment of concurrent mental health disorders like depression may be critical for help-seeking gamblers.

- Common underlying vulnerabilities should be actively researched to better understand, prevent, and treat both depression and problem gambling.

Limitations

- Depressive or problem gambling risk may not conferred within the five year span that was studied, at least not among emerging adults.
- While retention rates were fairly high (91.90%, 85.51%, and 78.06% for Waves 2 through 4, respectively), more data was missing over time.
- As might be expected in general population samples, rates of depression and problem gambling were low (despite use of symptom counts).

Key Words: major depressive disorder, problem gambling, emerging adults, longitudinal, comorbid, bi-directional, risk, pathoplastic effect

Abbreviations

CIDI-SF	Composite International Diagnostic Interview – Short Form
MDD	Major Depressive Disorder
MLSYA	Manitoba Longitudinal Study of Young Adults
PG	Problem Gambling
PGSI	Problem Gambling Severity Index

The lifetime prevalence rate of gambling disorder is approximately 0.4-1.0% in the general population, and past-year prevalence is 0.2-0.3%[1]. Gambling disorder typically originates in adolescence or young adulthood – a developmental trend that is demonstrated by

elevated PG risk in college and university samples. Approximately 42-85% of university students gamble, with 3-23% reporting risky weekly gambling patterns[2, 3]. Compared to other age groups, emerging adults (ages 18-25)[4], gamble more frequently and are more likely to have a gambling disorder. For example, adolescents problem gamble 2-4 times more often than adults[5] and, among college students, the lifetime prevalence rate of disordered gambling is 6-11%[6]. Specifically, 6.2% of Canadian undergraduates are moderate-risk gamblers and 1.4% have severe problem gambling (PG)[7]. A critical review concluded that being under the age of 29 was a significant risk factor for the development of PG[8]. These results indicate a propensity for early gambling behaviour may develop into PG. The current generation of North American adolescents and young adults is also the first to grow up with exposure to widespread, legalized, government-operated gambling. This increase in gambling accessibility and salience may mean the likelihood of PG development is also greater[9]. A focus on the correlates of gambling involvement in emerging adults (including risk factors and consequences) is therefore warranted.

Almost 38% of risky gamblers exhibit some form of mood disorder[10]. More specifically, compared to other gamblers, pathological gamblers are more likely to be depressed (26% of pathological gamblers vs. 18% of non-pathological gamblers)[11]. Lifetime major depressive disorder (MDD) is also more than three times more common for participants with lifetime PG than for those without[12]. Therefore, PG and MDD are positively associated, suggesting these disorders often co-occur. This is clinically significant because, among pathological gamblers seeking treatment, those with a comorbid psychiatric disorder tend to exhibit greater gambling pathology and psychopathology[13].

Compared to their non-gambling peers, adolescent problem gamblers have been found to have poor coping skills, have low self-esteem, be depressed, and have attempted suicide[5].

Further, they are at a greater risk for delinquency, academic problems, social problems, and other addictions. Thus, these results demonstrate that PG and depression are related and their co-occurrence can result in serious negative social and academic consequences among emerging adults.

While these studies support the idea that PG and depression are associated, they do not tell us *how* or *why* they are related. If these disorders are causally related, then their association may be explained via one of two pathways. First, depression may lead to escalations in gambling involvement through maladaptive coping. Jacob's general theory of addictions posits that addictive behaviours like gambling are reinforced and maintained by permitting escape from a painful reality[14]. Alternatively, the serious negative consequences associated with PG may be sufficient to trigger MDD[15]. These pathways are not mutually exclusive; a bi-directional relationship between PG and MDD symptoms may also be operative. Further, the two may be pathoplastically related, where an increase in one disorder would result in an increase in the other. To better understand the problematic gambling patterns of emerging adults, it is important that the direction of this relationship be better determined. Findings can inform targeted prevention and early intervention approaches.

The Present Study

The present study uses data collected in four waves over five years as part of the Manitoba Longitudinal Study of Young Adults (MLSYA). Conceptualized as a broad study of the risk and protective factors for PG, the MLSYA dataset provided a unique opportunity for better understanding the possible reciprocal relations MDD and PG in emerging adults. Longitudinal research is particularly helpful when examining gambling antecedents and consequences, as changes in levels of risky gambling can be more reliability studied over time[16].

Hypotheses

H1: In accordance with previous literature demonstrating that depressive and PG symptoms are related, it was hypothesized that MDD would be positively correlated with PG at Wave 1 when participants were aged 18 to 20.

H2: Based on the theory that depression causes escalations in risky gambling behaviour over time via a maladaptive coping process[14], it was hypothesized that higher levels of depressive symptoms at Wave 1 would predict a more pronounced increase in PG over time.

H3: Based on the theory that PG exacerbates depressive symptoms[15], the reverse was also hypothesized: higher levels of PG involvement at Wave 1 would predict a more pronounced increase in depressive symptoms over time.

H4: Finally, pathoplastic effects were also predicted. Increases in MDD would result in increases in PG, and vice versa, over time.

Graphical depictions of hypotheses appear in Figure 1. Income is differentially related to MDD and PG and past year problem gamblers with mood disorders are more likely to have lower incomes[15]. As a result, personal income was used as a covariate in all analyses.

Method

Sampling Procedures

Random-digit dialing was primarily used to recruit the Manitoban participants. News releases and informational posters placed within gambling facilities also invited self-referral for participation. Geographic and gender quotas were established to ensure that male and female, and urban and rural Manitobans were appropriately represented relative to census data. Following recruitment, selected participants were followed over a five year interval. Recruitment and the first cycle of data collection (Wave 1) took place in fall 2007. Three additional waves of

data collection then occurred in 12 to 18 month intervals: during fall 2008, spring 2010, and spring 2011[a].

Participants

At Wave 1, 679 18-20 year olds (mean = 18.92, SD = 0.79) were enrolled. Just over half (51.8%) of the participants were female and most (72.5%) self-identified as being of European descent. In addition to being Canadian, 6.2% of participants were Aboriginal (First Nations or Métis), 6.2% Hispanic, 0.8% White, 0.7% Black, 3.8%, Asian, 0.6% Middle Eastern, and 3.8% reported a primary religious affiliation (Jewish or Mennonite). The mean personal income at Wave 1 was $10,498.87 (SD = $6,837.55, median = $10,000.00).

Materials

The MLSYA battery included over 15 distinct measures, including: personality, emotional disorders, gambling motives, gambling behaviour, gambling problems, and socio-demographics. Only those relevant to the current study are described.

Depressive Symptoms

The Major Depressive Episode (MDE) subscale of the Composite International Diagnostic Interview – Short Form[17] (CIDI-SF) was used to assess depressive symptoms. This structured interview is aligned with International Statistical Classification of Diseases and Related Health Problems – 10th Revision[18] (ICD-10) and DSM-III-R[19] diagnostic criteria and was designed for use in epidemiologic studies.

The MDE subscale uses an established diagnostic algorithm and incorporates skip logic. Only participants' responses to the seven symptom-related questions were examined: feeling

[a] For more information, refer to <http://digitalcollection.gov.mb.ca/awweb/pdfopener?smd=1&did=17604&md=1>.

tired, weight changes, trouble sleeping, trouble concentrating, feeling down, and thoughts about death, and losing interest (e.g., "During a two week period where you felt sad, blue, or depressed, did you lost interest in most things, like hobbies, work, or activities that usually give you pleasure?"). Participants indicated whether they had (1) or had not (0) experienced each symptom.

The CIDI-SF[19] MDE scale was scored continuously: the seven depressive item scores were summed to create a 0-7 total. Continuous scores were deemed to be more appropriate for this community sample (rather than categorical diagnosis vs. non-diagnosis scores) as they allow a greater symptom range to be examined[20, 21].

The CIDI-SF[17] is psychometrically sound. A comprehensive review indicated that its test-retest reliability (κs .95) and inter-rater reliability (κs from .71) was adequate to excellent[22]. Further, 89.6% of major depressive episodes were correctly identified using the CIDI-SF, compared to its longer version[17].

Problem Gambling Symptoms

The Problem Gambling Severity Index[23] (PGSI), a nine-item subscale of the 31-item Canadian Problem Gambling Index[24] (CPGI), was used to assess PG outcomes. Developed using the DSM-IV-TR[25] and the South Oaks Gambling Screen[26] (SOGS) criteria for PG, it measures the extent to which respondents engage in risky or problem gambling behaviours. Five items address PG's diagnostic criteria (e.g., "Have you bet more than you could really afford to lose?") and four address the negative consequences of gambling (e.g., "Has gambling caused any financial problems for you or your household?"). Respondents are asked to indicate the frequency of each behaviour or consequence within the last 12 months. Responses were scored on a four-point Likert scale ranging from 0 (never) to 3 (almost always).

The PGSI[23] was scored continuously: the scores from all nine items were summed to form a 0-27 total. As with depression, continuous scores were deemed to be more appropriate for this community sample[27, 28] (compared to categorical non-gambler, non-problem gambler, low risk, moderate risk, and problem gambler scores[29]).

The PGSI[23] is internally consistent ($\alpha = .84$). Its test-retest reliability over several days is $r = .78$ and it is correlated highly with both the SOGS[26] ($r = .83$) and the DSM-IV[25] ($r = .83$) criteria for PG, thereby supporting its criterion validity.

Procedure

The CPGI[4] and the CIDI-SF[17] were administered at each wave as part of a 30-45 minute telephone interview (that included both open- and closed-ended questions).

Statistical Analysis

Missing data analyses, tests of normality, and descriptive statistics were conducted in SPSS 20.0. After assessing each variable, the appropriate distributions were specified in Mplus 7.1 (normal, poisson, zero-inflated poisson, or dichotomous). Once the distribution was selected, univariate latent growth curve models were tested for both MDD and PG. Time was modeled as linear, with Wave 1 serving as the intercept (time coded as 0, 1, 2 and 3 for each of the four waves). Next, a bivariate growth curve (see Figure 1) was run for MDD and PG. Personal income at Wave 1 was entered as a time-invariant covariate.

This bivariate growth curve contained four effects of interest: (a) the correlation between the intercepts, which represents the correlation between MDD and PG symptoms at Wave 1; (b) the correlation between the MDD intercept and the PG slope, which tests whether depression is a risk factor for increased PG; (c) the correlation between the PG intercept and the

MDD slope, which tests whether PG is a risk factor for increased depression; and (d) the correlation between the slopes, which tests whether MDD and PG are pathoplastically related.

Results

Data Cleaning

Data from 679 participants was analyzed for the purposes of this study. Retention rates were 91.90% (624 participants), 85.12% (578 participants), and 78.06% (530 participants) for Waves 2-4, respectively. Missing data was handled using a full information maximum likelihood approach.

Univariate distributions were assessed by examining skewness, kurtosis, and visual plots (box plots, histograms, P-P plots). At each wave, MDD and PG were substantially positively skewed. To account for this deviation from normality, variables were modeled as negative binomial distributions using the COUNT (nb) command in Mplus 7.1. Robust standard errors were also calculated using the MLR estimator.

We also considered categorical models, poisson models, and zero-inflated models. In the categorical model, MDD and PG were dichotomized to indicate the likely presence or absence of that disorder. We decided against this method because symptom count scores tend to be more appropriate for community samples. Many participants experienced only a few symptoms of MDD and/or PG, and did not meet the full diagnostic criteria. However, our method of calculating total scores allows for symptom counts below this threshold to be meaningful and allows for distinctions in symptom frequency and severity to be more readily made[b].

[b] Categorical models were run, but the results replicate those presented below (using derived symptom count variables).

Poisson models, which are discrete probability distributions, were also examined. Here, the linear predictor of the regression model was connected to the outcome via a natural logarithm function[30]. Another property of the poisson model, however, is that the mean must equal the variance. When the variance exceeds the mean, as it did with our data, then a negative binomial model is more appropriate (the standard error will be reliably larger, reflecting the additional outcome variance). When the data are over dispersed, the poisson models yield statistical tests that are too liberal[c], thereby increasing the probability of Type I error[30].

Finally, zero-inflated models were considered. Here, 0 scores were modelled as a separate process from the poisson portion of the model[30, 31]. While potentially appropriate for use in univariate growth models, this approach lacks parsimony when used with bivariate growth curves. Indeed, adding a zero-inflated component to the model outlined in Figure 1 would have doubled the number of parameters required (through simultaneous modeling of both the zeroes and poisson distributed portions) making the model more difficult to interpret, as well as increasing the Type I error rate (by doubling the number of paths in the model)[d].

Thus, having concluded that the negative binomial models with robust standard errors were the best fit for the data, we proceeded with this approach as the primary analytic strategy. Lastly, by converting the Wave 1 personal income values to z-scores and using 3.29 as a cut off, it was determined that many univariate outliers existed. All 21 of these outliers were removed[e].

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[c] When poisson models were run, many previously non-significant pathways became significant. Given the limitations of poisson models discussed above, we attributed this discrepancy to Type I error.

[d] Negative binomial inflated zero models were run, but since these more complicated models replicated the main analyses reported, we decided not to report them in the interest of maintaining model parsimony.

[e] At Wave 1, participants were 18-20 years old so it is highly unlikely they would have high annual personal incomes. It was assumed the question was misinterpreted so the outliers were

Univariate Growth Curves

Means and standard deviations at each wave are presented in Table 1. Before examining the bivariate growth curve, univariate linear growth curves for both variables of interest were modelled. Looking first at the univariate growth curve for MDD, the intercept (intercept = -1.69, $p < .01$) and the variability of the intercept ($\sigma = 5.52$, $p < .05$) were significant but the slope (slope = -0.58, $p = .198$) and the variability of the slope ($\sigma = 0.13$, $p = .132$) were not. This suggests there are between-subject differences in MDD symptoms, with some emerging adults experiencing more symptoms at Wave 1, but no within-subject change in symptoms over time. For PG, the intercept and slope were significant (intercept = -1.17, $p < .001$; slope = -0.84, $p < .001$), suggesting there is systematic change in individual levels of PG across waves. There was also significant variability in the intercept ($\sigma = 2.50$, $p < .001$) and the slope ($\sigma = 0.26$, $p < .001$), suggesting on average, some emerging adults experience increased symptoms while others saw a decrease.

Bivariate Growth Curve

Refer to Table 2 and Figure 2 for a summary of the bivariate growth curve results. Looking at MDD: its intercept and slope were uncorrelated, suggesting having symptoms of MDD at Wave 1 is unrelated to future changes in MDD symptoms. Neither MDD's intercept nor its slope was correlated with personal income, suggesting that Wave 1 income is unrelated to MDD symptoms at Wave 1 or over time. PG's intercept and slope were positively correlated, suggesting greater symptoms of PG at Wave 1 were related to greater escalations in PG over time. The intercept of PG was positively related to personal income, suggesting, at Wave 1,

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deleted. When the outliers were retained or Winsorized, the models failed to converge because the matrix was not positive definite.

emerging adults with a higher income tend to exhibit more symptoms of PG. The slope of PG, however, was uncorrelated with personal income, suggesting that income at Wave 1 is unrelated to changes in PG involvement over time.

The intercepts of MDD and PG were positively correlated, suggesting that, at Wave 1, emerging adults experiencing MDD symptoms also tend to exhibit symptoms of PG, and vice versa. The intercept of MDD and the slope of PG were uncorrelated, suggesting Wave 1 MDD symptoms are unrelated to changes in PG involvement over time. The intercept of PG and the slope of MDD were also uncorrelated, suggesting Wave 1 PG symptoms are unrelated to changes in MDD symptoms over time. Finally, the slopes of MDD and PG were uncorrelated, suggesting changes in one disorder are not associated with changes in the other.

Discussion

The bivariate growth curve showed that MDD and PG symptoms were positively correlated at Wave 1, which supports H1 and is consistent with previous literature[11, 12]. Contrary to H2 and H3, neither disorder was found to be a risk factor for the other. Furthermore, MDD and PG were not pathoplastically related, thus failing to support H4.

It may be that symptoms do not vary sufficiently within the emerging adult period. Depressive symptoms tend to emerge between ages 13-15 (for both overall rates of MDD and new cases)[32]. From 15-18 symptom rates then increase dramatically (for both genders), before plateauing from 18-21 (overall rates of MDD do not continue to increase and new cases decline). These results are consistent with our univariate MDD growth curve, which showed between- but not within-subject symptom change over time. Since there was little evidence of improvement or decompensation in depressive symptoms between 18-25 years, this age range may have been ill-suited for the longitudinal study of MDD risk factors. It is therefore unsurprising that PG did not

increase MDD risk. Likely, its risk factors exert a greater influence earlier in life (i.e., in adolescence).

PG, on the other hand, appears to be more transitory and episodic. Mid-adolescent gambling involvement is thought to lead to gambling problems which may, in turn, predict continuance of adult gambling problems. Alternatively, adolescents may mature out of their gambling problems[33]. These results are consistent with our univariate PG growth curve, which showed systematic change in individual symptoms levels over time. Intercept and slope variability suggested some emerging adults experienced increased symptoms while others saw a decrease. This systematic and within-person PG change suggests that we had a reasonable chance of finding MDD as a risk factor for PG. However, this hypothesis was unsupported by the data. It is therefore possible the co-occurrence of depressive and gambling symptoms at Wave 1 may be better explained not by MDD related risk but by the presence of a common underlying causal factor.

A systematic meta-analytic review found that, in addition to depression (weighted mean effect size of 23.1%), disordered gamblers also experienced high levels of alcohol misuse (28.1%), illicit drug use (17.2%), and nicotine dependence (60.1%)[11]. The link between MDD and substance use has been clearly established; both are also related to PG involvement. Substance use may therefore be a "third variable" underlying the MDD-PG relationship at Wave 1. For example, in a sample of 1,430 undergraduate students, disordered gamblers exhibited disproportionately higher depression (40.0% vs. 23.3% in the general population) and problem drinking (81.6% vs. 28.1% in the general population) co-morbidity rates[34]. Furthermore, participants who met the threshold for problem drinking also evidenced higher gambling rates

(9.0% vs. 4.2% for the entire sample) and participants who met the threshold for depression were engaging in more problematic gambling (9.4%, vs. 4.2% for the entire sample).

Limitations

At Wave 1, in fall 2007, participants were aged 18-20. Three additional waves of data collection occurred in fall 2008, spring 2010, and spring 2011. At Wave 4, participants were approximately 23 to 25 years old. As previously discussed, it is possible that peak MDD and/or PG symptoms occur before or after this age range or that risk is not conferred within five years.

Second, while retention rates were fairly high, more PGSI[23] and CIDI-SF[17] data was missing over time. This is a problem if data was missing not at random; for example, if the participants with more severe psychopathology participated less over time. However, maximum likelihood approaches, which were used in the present study, are among the most effective in handling missing data[35].

Finally, rates of depression and problem gambling involvement were low in our sample, despite the use of symptom count scores. However, our use of negative binomial distributions and robust standard errors helps mitigate this limitation.

Conclusion

The bivariate growth curve supports the co-occurrence of MDD and PG in emerging adults. Therefore, active screening for and treatment of co-occurring mental health disorders like depression may be critical for help-seeking gamblers. For example, among pathological gamblers seeking treatment, those with a comorbid psychiatric disorder have been shown to exhibit greater severity of both gambling pathology and psychopathology[13]. Further, MDD is associated with suicide, and in a sample of 500 problem gamblers, 48% reported suicide ideation and 13% reported suicide attempts[36]. MDD and bipolar disorder outpatients with comorbid PG

were more than twice as likely to have attempted suicide in the past month as were those without[15].

Given the co-occurrence of MDD and PG, clinicians should screen for and provide treatment for both disorders. We cannot assume that treating one will resolve the other, as these disorders were found to be unrelated over time. Once common factors underlying the MDD-PG relationship are identified, these should be further targeted in preventative interventions. Although individuals with comorbid disorders tend to have lower psychosocial functioning, treatment has been found to effective and satisfying regardless of co-occurring disorder frequency[37].

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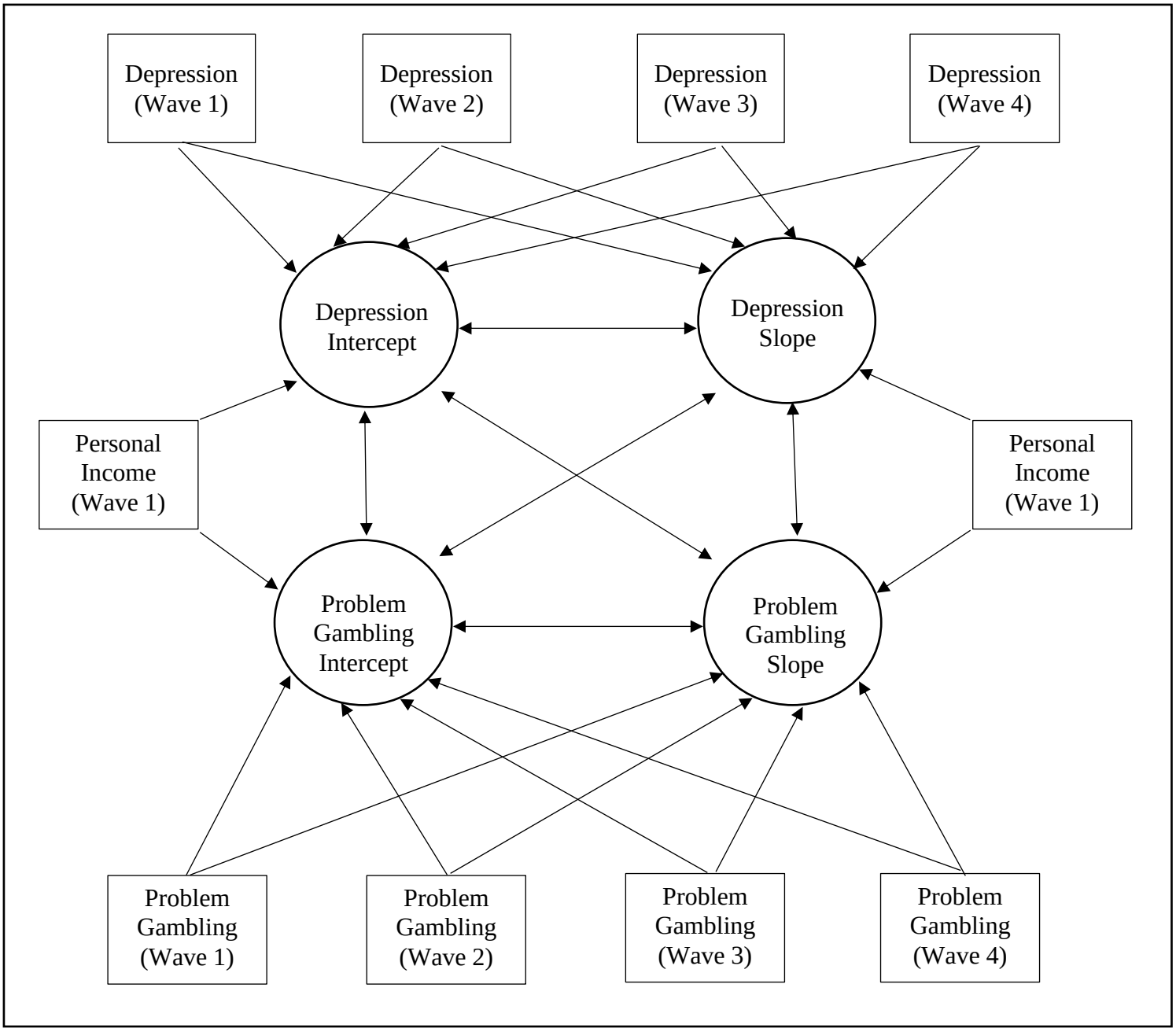
Figure 1 Model of bivariate growth curve hypotheses



Note. Bivariate growth curve analysis of emotional disorder and gambling symptomology.

Rectangles represent measured variables and ovals represent latent intercepts and slopes.

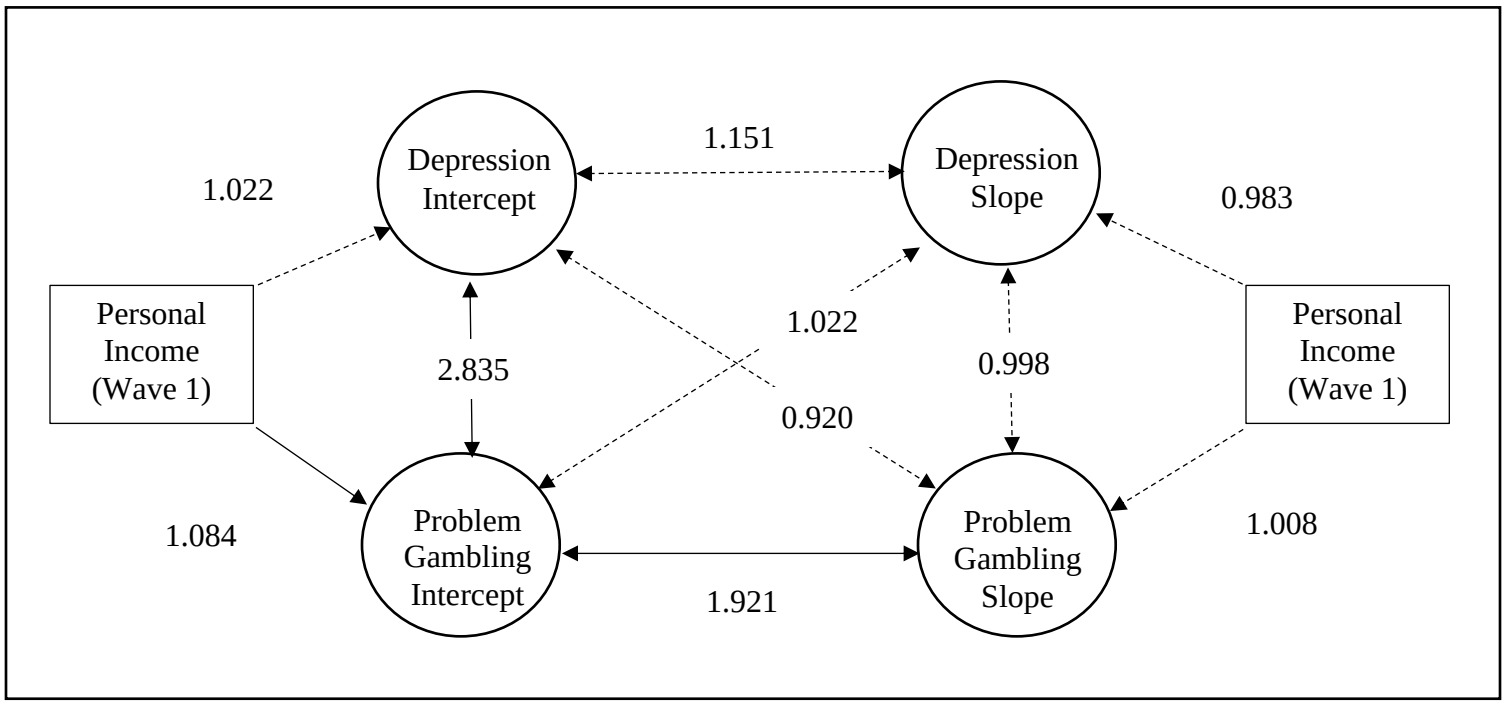
Double-headed arrows represent factor loadings.

Table 1 Sample descriptive statistics

	Wave 1	**Wave 2**	**Wave 3**	**Wave 4**
Depression Symptomology	0.79 (1.81)	0.85 (2.32)	0.92 (2.40)	0.43 (1.45)
Problem Gambling Symptomology	0.97 (2.01)	0.66 (1.77)	0.59 (1.96)	0.41 (1.48)
Personal Income	10,495 (6,837.55)			

Note. Mean (standard deviation) presented for each variable of interest at each applicable wave.

Figure 2 Model of depression and problem gambling bivariate growth curve



Note. Bivariate growth curve analysis of depressive and gambling symptomology. Rectangles represent measured variables and ovals represent latent intercepts and slopes. Double-headed arrows represent factor loadings. Single-headed arrows represent paths. Solid arrows represent statistically significant pathways and dotted arrows represent non-significant pathways. Numbers represent incident rate ratios for paths and correlations.

Table 2 Depression and problem gambling results

	B	p	Standard Error	Incident Rate Ratio	99% Confidence Interval of Incident Rate Ratio
Depression Intercept on Depression Slope	0.141	.943	1.965	1.151	[.007, 181.454]
Gambling Intercept on Gambling Slope	0.653	.000	0.123	1.921	[1.402, 2.635]
Depression Intercept on Gambling Slope	-0.083	.751	0.262	0.920	[.469, 1.808]
Gabling Intercept on Depression Slope	0.022	.952	0.370	1.022	[.394, 2.651]
Depression Intercept on Gambling Intercept	1.042	.002	0.337	2.835	[1.190, 6.753]
Depression Slope on Gambling Slope	-0.002	.953	0.030	0.998	[.924, 1.079]
Depression Intercept on Personal Income	0.022	.197	0.025	1.022	[.958, 1.092]
Depression Slope on Personal Income	-0.017	.399	0.010	0.983	[.958, 1.008]
Gambling Intercept on Personal Income	0.081	.000	0.001	1.084	[1.058, 1.112]
Gambling Slope on Personal Income	0.008	.288	0.006	1.008	[.993, 1.022]

Note. Statistically significant pathways ($p < .05$) are bolded.