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Profils de joueurs à problèmes:

un test empirique du modèle des parcours multiples

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Résumé

Le modèle des parcours multiples, proposé par Blaszczynski et Nower (2002), suppose l'existence de trois cheminements distincts menant à un trouble du jeu excessif et résultant en trois profils distincts de joueurs: les conditionnés, les émotionnellement vulnérables, et les biologiquement vulnérables. Dans la description de ces trois profils de joueurs, les auteurs stipulent que certaines composantes affectives et/ou comportementales surviennent avant l'apparition du jeu problématique, alors que d'autres en découlent. Il est donc important d'avoir des données longitudinales afin de pouvoir valider le modèle des parcours multiples. Ce dernier a été validé à quelques reprises auprès d'adultes et à une seule reprise auprès d'adolescents. Aucune de ces études n'a utilisé une perspective longitudinale.

À travers cette thèse, nous présenterons trois articles (dont deux de nature empirique) ayant pour objectif principal la validation du modèle des parcours multiples auprès d'une population d'adolescents suivis jusqu'au début de l'âge adulte. Les deux articles empiriques reposent sur des données recueillies de manière prospective auprès de deux échantillons populationnels. De façon plus précise, le premier article empirique vise à identifier la présence des profils proposés par le modèle des parcours multiples parmi l'ensemble des joueurs problématiques répertoriés au sein des deux échantillons populationnels. Pour ce faire, des indicateurs d'ordre personnel mesurés en début d'adolescence ont été soumis à une analyse de profils latents afin de vérifier si les profils qui en résulteraient sont conformes à ceux proposés par le modèle des parcours multiples. Les indicateurs d'ordre personnel sont la dépression, l'anxiété, l'impulsivité, l'hyperactivité, l'antisocialité, l'agressivité, ainsi que la consommation d'alcool ou de drogues. Les profils ainsi dérivés ont ensuite été comparés au niveau de leur répartition homme/femme, et de leurs

comportements de jeu en fin d'adolescence et au début de l'âge adulte. Le deuxième article empirique utilise les mêmes indicateurs d'ordre personnel et applique la même méthodologie que le premier article, mais auprès de l'ensemble de l'échantillon (c.-à-d., en incluant les non-joueurs), afin d'identifier dès le début de l'adolescence des profils à risque de développer des problèmes de jeu et vérifier si ces profils sont à nouveau conformes à ceux proposés par le modèle des parcours multiples. En outre, divers facteurs de compensation ou de protection mesurés à 14 ans (supervision parentale, attachement à la famille, aux pairs, et la conformité des pairs), et susceptibles de minimiser l'occurrence de problèmes de jeu à la fin de l'adolescence ou au début de l'âge adulte ont été mis en scène. Finalement, le troisième article tente de dégager des pistes pour la prévention et l'intervention qui seraient compatibles avec nos résultats de recherche et le modèle des parcours multiples.

Les résultats du premier article empirique suggèrent l'existence de quatre profils distincts; dont trois ressemblent à ceux décrits par le modèle des parcours multiples. Le quatrième profil présente des caractéristiques similaires à la fois aux joueurs émotionnellement vulnérables et aux joueurs biologiquement vulnérables. Ces résultats sont confortés par ceux du second article empirique qui identifie des profils similaires auprès de tous l'échantillon, même les non-joueurs. Divers facteurs de protection et de compensation ont été également identifiés en fonction du profil des participants, et du temps de mesure des problèmes de jeu (c.-à-d., fin d'adolescence versus début de l'âge adulte). Ensemble, ces résultats militent en faveur d'une approche de prévention et d'intervention différenciée en fonction du profil des participants.

Mots-clés : jeux de hasard et d'argent; devis longitudinal; adolescence; jeune adulte; facteurs de risque; facteurs de protection; modèle des parcours multiples

Abstract

The Pathways Model, theorized by Blaszczynski and Nower (2002), suggests the existence of at least three distinct pathways leading to problem-gambling, resulting in three unique profiles of gamblers: behaviorally conditioned, emotionally vulnerable, and biologically vulnerable gamblers. The authors suggest that each one these profiles is characterized by a set of biopsychosocial characteristics that precede or follow the beginning of problem-gambling. In order to distinguish what precedes from what may follow, it is important to use longitudinal data to effectively validate the Pathways Model. Several attempts have been made to verify the theoretical model's accuracy, only one of which targeted adolescents. However, none of these studies implemented a longitudinal perspective.

Throughout this thesis, three articles will be presented (two of which are empirical studies) with the main goal of validating the Pathways Model among a population of adolescents followed into early adulthood. Both empirical studies have collected data using a prospective approach in two population samples. More precisely, the first empirical study aims to identify the presence of the profiles described by the Pathways Model specifically among problem-gamblers from both samples. To achieve this goal, indicators of personal risk assessed in early adolescence were used for latent profile analyses, in order to explore whether the resulting best-fitting model would bring out profiles consistent with the Pathways Model. These indicators consisted of depression, anxiety, impulsivity, hyperactivity, antisociality, aggressiveness, and drug/alcohol use. The resulting profiles were then compared in terms of their gender distribution, as well as their number of gambling problems in late adolescence and early adulthood. The second empirical article used these same risk indicators and the same statistical techniques among the whole samples (i.e.,

including non-gamblers), in order to identify as early as the beginning of adolescence profiles that could be at risk for developing gambling problems, and to check once again whether these profiles resembled those described by the Pathways Model. Moreover, various compensatory and protective factors assessed when participants were 14 years old (parental involvement, parent-child connectedness, peer conformity, and peer connectedness), and likely to minimize the occurrence of gambling problems, were put into play. Finally, the third article aims to offer various suggestions for prevention and intervention efforts, which are in line with our study results and the Pathways Model.

Results from the first empirical study point toward the existence of four unique profiles of problem-gamblers; three of which closely resemble those described by the Pathways Model. The fourth profile likely represents an overlap of Emotionally and Biologically Vulnerable pathways. These results are comforted by the second empirical study, which identifies four similar profiles, but among the full population sample. Various compensatory and protective factors were also identified, as a function of participants' at-risk profiles, and the time-point when gambling problems were assessed (i.e., late adolescence and early adulthood). Taken together, these results advocate a targeted prevention and intervention approach, tailored to participants' profiles.

Keywords: gambling; longitudinal design; adolescence; early adulthood; risk factor; protective factors; pathways model

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Liste des sigles et abréviations

En français :

c.-à-d.	C'est-à-dire
BV	Biologiquement Vulnérable
CC	Comportementalement Conditionné
EV	Émotionnellement Vulnérable
GRIP	Groupe de recherche sur l'inadaptation psychosociale chez l'enfant
JHA	Jeux de Hasard et d'Argent
p. ex.	Par exemple
SSE	Statut Socio-Économique
TDAH	Trouble du Déficit de l'Attention avec Hyperactivité
TPA	Trouble de la Personnalité Antisociale

En anglais:

ADHD	Attention-Deficit Hyperactivity Disorder
BC	Behaviorally Conditioned
BV	Biologically Vulnerable
DSM-5	Diagnostic and Statistical Manual of Mental Disorders: Fifth Edition
e. g.	For example; from Latin, e*xempli gratia*
EV	Emotionally Vulnerable
GD	Gambling Disorder
i. e.	That is; from Latin, *id est*
LPA	Latent Profile Analysis
M	Mean
PG	Problem-Gambling
SD	Standard Deviation
SES	Socioeconomic Status
SOGS	South Oaks Gambling Screen
SOGS-RA	South Oaks Gambling Screen – Revised for Adolescents
SUD	Substance Use Disorder

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Introduction

Le *Diagnostic and Statistical Manual of Mental Disorders : Fifth Edition* (DSM-5; American Psychiatric Association, 2013) estime que l'exercice de jeux de hasard et d'argent (JHA) devient un trouble lorsqu'un joueur présente un certain nombre de problèmes directement liés au jeu d'un point de vue légal, social et de maîtrise de soi. Par ailleurs, le DSM-5 regroupe le trouble lié aux JHA avec les autres dépendances (p. ex., à la drogue). Toutefois, le nombre et la gamme de problèmes éprouvés par les joueurs troublés (*disordered* en anglais) peuvent varier grandement. En plus des joueurs troublés, il existe des joueurs à risque. Ceux-ci présentent des problèmes, mais pas suffisamment pour répondre aux critères du DSM-5; ces joueurs méritent néanmoins notre attention parce que leur situation pourrait s'aggraver (Billi, Stone, Marden et Yeung, 2014). Dans cette thèse, le terme *joueur problématique* désignera un individu avec au moins un problème de JHA; le terme *joueur à risque* désignera un individu avec un niveau sous-clinique de trouble lié aux JHA; alors qu'un *joueur troublé* désignera un individu ayant atteint le seuil clinique (variable selon l'outil administré et l'âge de mesure).

Au Québec, de 36,6% à 80% (Dubé et al., 2009; Gupta et Derevensky, 1998) des adolescents s'adonnent à des JHA chaque année. L'écart entre ces taux peut s'expliquer par des différences méthodologiques. Par exemple, le taux de participation augmente à travers les années d'école secondaire, donc le temps de mesure est important. De plus, certaines études, comme celle de Dubé et al. (2009), ne prennent pas en compte les élèves décrocheurs, ce qui soulève la possibilité d'une sous-estimation du taux réel. Finalement, le choix des instruments de mesure peut également porter à conséquence, les critères pour identifier un joueur à risque ou troublé pouvant varier de l'un à l'autre. Malgré ces différences, il est important de remarquer que tous les adolescents qui

participent à des JHA ne développent pas de problèmes liés au jeu. En effet, environ 10-15% sont à risque de développer au moins un problème relié au jeu et environ 4% vivent une multitude de problèmes en lien avec leur pratique de JHA.

Quoique certains auteurs jugent qu'il pourrait y avoir une surestimation de la prévalence de JHA chez les adolescents (Derevensky, Gupta et Winters, 2003), la plupart des adultes présentant des niveaux cliniques de JHA déclarent avoir commencé à jouer avant ou pendant leur adolescence (Gupta et Pinzon, 2012). Il est donc important de pouvoir identifier les joueurs à risque dès l'adolescence, afin de mieux diriger les efforts de prévention et ainsi éviter les conséquences négatives que cela pourrait amener à l'individu et à la société.

La problématique abordée dans cette thèse

Les besoins en matière de prévention sont d'autant plus importants que la pratique de JHA a été citée comme étant le plus grand problème comportemental impliquant un risque pour la santé des élèves dans les écoles secondaires du Québec (Gupta et Pinzon, 2012): 28,2% des élèves ont déclaré qu'ils jouaient au moins 1 fois par semaine. Ce comportement est suivi par la cigarette (17,4%), les drogues illicites (13,8%) et l'alcool (13,5%). En revanche, les programmes de prévention en matière de JHA sont inefficaces, notamment parce qu'ils ne tiennent pas compte des besoins particuliers des diverses catégories de joueurs et parce que nos connaissances sur les facteurs de risque et de protection en lien avec les JHA sont limitées, quoique constamment approfondies (Ladouceur, Goulet et Vitaro, 2013).

Dans leur résumé des connaissances acquises en matière de JHA chez les adolescents, Derevensky et Gupta (2004) concluent que, comparés à leurs équivalents sans problèmes, les adolescents ayant des problèmes de JHA ont tendance à : prendre plus de risques, faire plus de

tentatives de suicide, avoir une estime de soi plus basse, être plus impulsifs et défier les normes sociales. Toutefois, tel qu'exposé par la recension de Milosevic et Ledgerwood (2010), les études de cas cliniques suggèrent que tous les joueurs problématiques ne présentent pas nécessairement ce profil de risque qui semble caractérisé à la fois par des problèmes de nature intériorisée (p. ex., symptômes de dépression ou d'anxiété) et par des problèmes de nature extériorisée (p. ex., prise de risques, impulsivité, et antisocialité), ce qui laisse entrevoir des typologies diverses de joueurs avec des profils différents. L'existence de groupes distincts de joueurs présentant chacun une constellation particulière de facteurs de risque ou de protection commanderait alors une perspective différenciée en matière de prévention et d'intervention. Une telle approche est actuellement inexistante dans le domaine de la prévention des problèmes de JHA chez les adolescents (Ladouceur et al., 2013); mais, encore faut-il que l'existence de profils distincts de joueurs soit valide et établie de manière empirique et claire.

En effet, les rares études qui ont examiné et rapporté des profils distincts de joueurs, non seulement ont-elles été menées chez les adultes, mais elles ont utilisé un devis transversal, ce qui limite la portée de leurs résultats. Contrairement à un devis longitudinal, un devis transversal ne permet pas de préciser la direction des liens entre les variables en jeu, de sorte qu'il n'est pas clair si les caractéristiques qui définissent les joueurs à problèmes précèdent ou découlent des problèmes de jeu; d'où la nécessité d'une perspective longitudinale (Vitaro, 2012).

Facteurs prédicteurs des problèmes de JHA : une perspective longitudinale et différenciée

Une perspective longitudinale et différenciée est nécessaire pour alimenter les modèles étiologiques des problèmes de jeu ainsi que les programmes de prévention et d'intervention, dont les cibles et les stratégies devront être ajustées en fonction des constellations possiblement particulières de facteurs de risque et de vulnérabilité, auxquels se greffent les facteurs de compensation et de protection, s'il y a lieu. Comme précisé par Lussier, Derevensky, Gupta et Vitaro (2013), un facteur de compensation diminue la probabilité d'apparition d'un problème de jeu, qui pourrait découler de la présence de facteurs de risque, selon un mode additif (en termes d'effet de régression) alors qu'un facteur de protection y parviendrait selon un mode interactif (c.-à-d., par multiplication ou division). Le premier est par conséquent l'inverse d'un facteur de risque alors que le second est apparenté à un facteur modérateur, tout comme les facteurs de vulnérabilité, mais en sens inverse. Avant d'aborder les facteurs de risque et de protection selon une perspective différenciée, il importe d'en faire un inventaire et une analyse critique sur un plan méthodologique (c.-à-d., en distinguant notamment les études qui ont utilisé une méthodologie transversale et les études qui ont utilisé une méthodologie longitudinale).

Facteurs de risque et de vulnérabilité

Plusieurs aspects de la personnalité d'un individu ont été démontrés comme ayant un lien avec les problèmes de jeu. Ces aspects touchent le domaine comportemental, cognitif, ou affectif. Certains de ces aspects découlent aussi de prédispositions biologiques. Des études faites auprès de jumeaux (en début d'adolescence et à l'âge adulte) ont permis de déterminer qu'il existe des prédispositions biologiques importantes dans le développement de problèmes de jeu (Lobo et

Kennedy, 2009; Shah, Eisen, Xian et Potenza, 2005; Vitaro et al., 2014). Celles-ci peuvent s'exprimer à travers des anomalies cérébrales. Le striatum ventral, couramment appelé « système de récompense », et le cortex orbitofrontal sont deux régions candidates : en plus d'être associées avec la sévérité des problèmes de JHA (Büchel, 2006; van Holst, van den Brink, Veltman et Goudriaan, 2010), elles sont aussi impliquées dans d'autres dysfonctions fréquemment associées au jeu problématique, telle que la consommation abusive de substances psychoactives (Volkow et Fowler, 2000), le trouble de déficit d'attention avec hyperactivité (TDAH; Carmona et al., 2009), l'inhibition de cognitions et un ensemble de comportements impulsifs (Berlin, Rolls et Kischka, 2004).

Au plan endophénotypique (c.-à-d. une dimension intermédiaire entre la biologie et la personnalité), l'impulsivité figure parmi les caractéristiques les plus communément associées aux problèmes de JHA (Dussault, Brendgen, Vitaro, Wanner et Tremblay, 2011; Johansson, Grant, Kim, Odlaug et Gotestam, 2009; Nower, Derevensky et Gupta, 2004). Plus précisément, Dussault et al. (2011) ont utilisé un devis longitudinal pour explorer l'ordre d'apparition de différents facteurs de risque (impulsivité et dépression) par rapport aux problèmes de JHA. Ils ont d'abord montré que le lien à l'adolescence entre la dépression et le jeu problématique s'expliquait par des antécédents communs (p. ex., l'impulsivité). Une fois en place, les problèmes de jeu à l'adolescence ont par la suite expliqué l'aggravation de symptômes dépressifs au début de l'âge adulte. Inversement, les symptômes dépressifs à l'adolescence ont aussi prédit une aggravation des problèmes de jeu de l'adolescence au début de l'âge adulte. Ces résultats montrent bien l'importance d'adopter une perspective développementale qui va au-delà de l'adolescence car le rôle des variables en jeu pourrait varier d'une période développementale à une autre. Ils montrent la préséance de l'impulsivité par rapport à l'émergence des problèmes de jeu et des symptômes

dépressifs à l'adolescence. La présence d'antécédents communs tels l'impulsivité ou des notions connexes telles la recherche des sensations fortes (Johansson et al., 2009) peut également expliquer pourquoi les joueurs à problèmes consomment plus d'alcool à l'adolescence (Scholes-Balog, Hemphill, Dowling et Toumbourou, 2014) et abusent de substances psychoactives (Hardoon, Gupta et Derevensky, 2004). Quelques études longitudinales ont par ailleurs montré que l'impulsivité pouvait expliquer, quoique partiellement, l'association entre les problèmes de jeu et la consommation de psychotropes à l'adolescence (Vitaro, Brendgen, Ladouceur et Tremblay, 2001). Par contre, d'autres études ont trouvé que l'impulsivité expliquait la sévérité de plusieurs problèmes de consommation, mais non l'apparition de problèmes de JHA (Barnes, Welte, Hoffman et Dintcheff, 2005). Cela pourrait être dû à des typologies différentes de joueurs au sein des diverses études, mais aussi à une variété de différences au niveau des instruments de mesure et de l'échantillonnage des participants.

D'autres troubles de comportement ont été reliés aux problèmes de jeu. Par exemple, le trouble lié aux JHA a été associé à une tendance à afficher des comportements antisociaux (Jacobs, 2000; Stinchfield, 2000; Vitaro et al., 2001) ou agressifs (Martins et al., 2013). Plusieurs critères diagnostiques du trouble de la personnalité antisociale (TPA) font écho à un dysfonctionnement du cortex préfrontal et rappellent des facteurs de prédiction déjà évoqués dans le cadre d'études longitudinales en lien avec les JHA (p. ex., comportements impulsifs, agressivité, difficulté à se conformer aux règles). D'autres troubles liés à un tel dysfonctionnement cérébral, tel que le TDAH (un trouble ayant une composante d'impulsivité), sont aussi associés au jeu problématique. Ces différents aspects pourraient donc avoir un antécédent biologique commun, du moins chez certains joueurs.

En ce qui concerne les troubles intériorisés, les joueurs à problèmes présentent des niveaux de dépression ou d'anxiété plus élevés que la normale (Becona, Del Carmen Lorenzo et Fuentes, 1996; Petry, Stinson et Grant, 2005). Ceci est concordant avec les études associatives qui ont trouvé un lien concomitant ou longitudinal entre les problèmes intériorisés et les problèmes de jeu. Rappelons l'étude de Dussault et al. (2011) qui ont trouvé un lien longitudinal entre les symptômes dépressifs et les problèmes de jeu, mais seulement du milieu de l'adolescence au début de l'âge adulte, ou encore celle de Volberg, Reitzes et Boles (1997) qui ont trouvé un lien entre une faible estime de soi et un niveau élevé de problèmes de JHA à l'âge adulte. Ces résultats suggèrent que les troubles intériorisés pourraient contribuer au développement des problèmes de jeu mais à partir de la fin de l'adolescence et pas auparavant. De plus, il est important de noter qu'au sein même des échantillons de joueurs à problèmes, les troubles affectifs ne sont pas uniformément répartis (Kim, Grant, Eckert, Faris et Hartman, 2006). Cela suggère qu'il pourrait exister un sous-groupe spécifique de joueurs avec des problèmes de JHA démontrant des problèmes affectifs, même à l'adolescence, mais ceux-ci passent inaperçus lorsque les résultats sont analysés pour l'ensemble des participants. Par exemple, une analyse en profondeur de leur échantillon a permis à Roy, Custer, Lorenz et Linnoila (1988) de départager plusieurs facettes de la dépression vécue par leurs participants et ainsi déterminer que certains aspects dépressifs découlaient des problèmes de JHA alors que d'autres en étaient indépendants. Encore une fois, ces résultats concernent les adultes et ne nous renseignent pas nécessairement au sujet de joueurs adolescents.

Au niveau des facteurs sociodémographiques, Jacobs (2005) indique que la fréquence de comportements problématiques de JHA est plus élevée auprès des hommes et des minorités ethniques vivant dans des quartiers défavorisés. Auger, Lo, Cantinotti et O'Loughlin (2010) ont aussi mis en évidence le rôle du statut socio-économique (SSE) dans le développement des

problèmes de jeu. Ils ont trouvé que le SSE interagissait avec les niveaux d'impulsivité de joueurs adolescents pour prédire l'émergence d'habitudes de jeu : l'impulsivité était associée à une initiation au jeu précoce chez les participants issus de milieux défavorisés seulement. Cela illustre bien qu'une analyse de facteurs de risque doive se faire en considérant des facteurs potentiellement modérateurs (p. ex., protecteurs) ou compensatoires, poussant ainsi une analyse différenciée au-delà des prédispositions personnelles susceptibles de caractériser les joueurs à problèmes.

Facteurs de compensation et de protection

Malgré que l'étude des facteurs compensatoires et protecteurs soit déficitaire par rapport à l'étude des facteurs de risque, quelques résultats émergent de façon récurrente. Le soutien social de la part des parents ou des pairs est associé à divers résultats positifs chez les adolescents, autant au niveau physique que psychologique (Abazari, Haghdoost et Abbaszadeh, 2014; King, Tergerson et Wilson, 2008). Toutefois, en ce qui concerne les JHA, seulement le soutien de la part de la famille, et non des pairs, diminue le risque d'émergence du jeu problématique (Rasanen, Lintonen, Tolvanen et Konu, 2016). Au niveau familial, Lee, Stuart, Ialongo et Martins (2014) ont constaté qu'une décroissance de la supervision parentale entre 11 et 14 ans est associée à des problèmes de JHA plus sévères au début de l'âge adulte. Dans une étude australienne, l'environnement familial (p. ex., comportements prosociaux au sein de la famille) a été identifié par Scholes-Balog et al. (2014) comme étant un facteur de protection significatif contre les problèmes de jeu, pour les adolescents consommant de l'alcool (mettant une fois de plus en évidence un effet modérateur en lien avec les JHA)

En revanche, les jeunes avec des pairs aux comportements socio-normatifs (c.-à-d., non-délinquants) sont moins enclins à éprouver des problèmes de jeu (Lussier, Derevensky, Gupta et

Vitaro, 2014; Vitaro et al., 2001) ; toutefois, ces effets n'ont été observés que lors d'études transversales. Étant donné que les joueurs pourraient s'affilier à des pairs qui partagent leurs habitudes délinquantes, ou adopter ces habitudes une fois qu'ils y ont été exposés par leurs pairs, il n'est pas clair si la non-conformité des pairs précède ou suit les problèmes de jeu.

La recherche d'appartenance sociale (*social bonding*), composée de la cohésion familiale et de l'engagement scolaire, a aussi été démontrée comme jouant un rôle significatif en tant que facteur de compensation contre les problèmes de jeu (Lussier et al., 2013). Par exemple, l'assiduité et la performance scolaires démontrent un lien négatif avec les problèmes de JHA à l'adolescence (Ladouceur, Boudreault, Jacques et Vitaro, 1999; Winters, Stinchfield et Fulkerson, 1993). En somme, les facteurs de risque associés aux problèmes de jeu sont nombreux et variés, mais ils ne s'appliquent vraisemblablement pas tous de la même manière selon le type de joueur et la période développementale examinée.

Prévention des problèmes de JHA

Les programmes de prévention sont classés en deux catégories principales (Gordon, 1983; Mrazek et Haggerty, 1994). Les programmes universels s'appliquent uniformément à l'ensemble de la population, sans égard aux caractéristiques individuelles de chaque individu. Il pourrait s'agir, par exemple, d'un programme fourni à l'ensemble des élèves à l'école secondaire. D'autre part, les programmes ciblés visent un sous-groupe de la population particulièrement à risque de développer le comportement problématique concerné. Les programmes ciblés de type « indiqués » concernent les individus présentant des caractéristiques personnelles à risque (p. ex., des individus impulsifs), alors que les programmes de type « sélectifs » dépistent les individus à risque en

fonction de caractéristiques environnementales (p. ex., des adolescents avec un entourage de joueurs).

Actuellement, les programmes de prévention de JHA destinés aux adolescents sont tous de type universel (Ladouceur et al., 2013). La plupart de ces programmes visent à corriger les croyances erronées associées aux JHA, telle que la probabilité de gagner à un jeu en particulier. D'autres programmes cherchent à sensibiliser les jeunes aux conséquences néfastes associées au jeu problématique (p. ex., en fournissant des exemples réels de personnes réelles souffrant du trouble), et à leur fournir une panoplie de ressources pouvant servir à une personne de leur entourage ou à eux-mêmes (Ladouceur, Ferland, Vitaro et Pelletier, 2005).

Malheureusement, les résultats de ces programmes sont décevants. Non seulement plusieurs programmes n'ont pas été évalués à long-terme, ceux qui l'ont été démontrent que le comportement de JHA demeure inchangé (Ladouceur et al., 2013), malgré que des connaissances au sujet des JHA, tels que les principes statistiques d'événements aléatoires, aient été effectivement transmises aux jeunes. De plus, aucun de ces programmes n'adopte de perspective différenciée du jeu problématique prenant en considération les profils uniques à risque. Par conséquent, une approche universelle ne serait probablement pas suffisante dans la prévention des problèmes de JHA chez les jeunes à risque.

Groupement des facteurs de risque et de protection par profil de joueur : une approche typologique

Moran (1970) a été l'un des premiers à proposer différentes explications pour rendre compte de l'apparition de niveaux pathologiques de JHA à l'âge adulte, en séparant les joueurs pathologiques en cinq sous-groupes (sous-culture, névrotique, impulsif, psychopathe, symptomatique). Jacobs (1986) a, par la suite, proposé une théorie (*General Theory of Addictions*) qui intègre des aspects biologiques et psychologiques de l'individu. Selon lui, il existerait deux types de personnes pouvant développer une dépendance au jeu (ou à une substance) : les personnes ayant un état physiologique au repos caractérisé par une hypotension et celles ayant un état chronique d'hypertension. De plus, chaque groupe aurait une prédisposition psychologique à adopter des attitudes négatives vis-à-vis de soi-même (p. ex., faible estime de soi) qui mènerait à une quête d'approbation de l'autre. Sharpe (2002) propose un modèle étiologique plus complet en y intégrant les influences sociales. De plus, Sharpe crée une distinction entre les joueurs qui préfèrent les jeux demandant un minimum de connaissances (p. ex., jeux de cartes, course à chevaux) et ceux qui préfèrent les jeux de pure chance (p. ex., machine à sous). Le premier groupe serait à la recherche de sensations fortes (intolérance à l'ennui) alors que le deuxième chercherait à se distraire de ses affects négatifs.

Ces subdivisions supposent des constellations particulières de facteurs de risque (et de protection) qui se combinent de manière additive ou interactive, mais leurs fondements empiriques sont demeurés quasi inexistants jusqu'à récemment. En effet, les regroupements proposés par ces auteurs découlent davantage d'une analyse clinique ou qualitative que d'une démarche empirique. Depuis, un certain nombre d'auteurs ont repris l'idée qu'il existe différents profils de joueurs à

l'âge adulte, mais en faisant appel cette fois à des bases empiriques fondées sur diverses caractéristiques personnelles et environnementales (Gonzalez-Ibanez et al., 2003; Gupta et al., 2012; Ledgerwood et Petry, 2010; Nower et Blaszczynski, 2005).

Le premier modèle avec des fondements théoriques et empiriques, et dont il sera question principalement dans cette thèse, a été proposé par Blaszczynski et Nower (2002). Il s'agit du Modèle des Parcours Multiples (*Pathways Model*) qui décrit trois cheminements menant au développement de niveaux variables de problèmes de JHA. Chacun de ces cheminements est aussi caractérisé par une évolution différente et des conséquences variables des problèmes liées au jeu. Le modèle a été réarticulé pour aussi prendre en compte les adolescents (Nower et Blaszczynski, 2005).

Le *Pathways Model* se présente sous la forme d'un modèle développemental, ce qui est un avantage comparé aux autres modèles étiologiques. Il présente les différents cheminements en commençant tôt dans la vie d'un individu pour finir avec le développement des comportements problématiques. Un tel modèle développemental pourrait faciliter le dépistage précoce des facteurs de risque pertinents avant que des problèmes associés aux JHA ne surviennent, en tenant compte des cheminements en jeu. Il offre aussi l'avantage d'expliciter les facteurs de risque impliqués ainsi que leur agencement temporel au point d'en permettre une évaluation empirique. Ainsi, Milosevic et Ledgerwood (2010) ont analysé 17 études abordant la classification de joueurs pathologiques, et ont conclu qu'un modèle à trois groupes ressort de manière récurrente. Ces trois groupes ressemblent aux trois profils décrits par le *Pathways Model*. Par conséquent, ces auteurs recommandent aux études subséquentes de focaliser sur ce modèle en particulier en tentant de combler les lacunes des études existantes (explicitées plus loin).

Pathways Model

Dans leur chapitre sur l'étiologie des problèmes de JHA, Blaszczynski et Nower (2007) avisent qu'un modèle complet doit intégrer les perspectives biologiques, psychologiques et sociales. Effectivement, le *Pathways Model* intègre des facteurs biologiques, cognitifs, personnels et environnementaux selon une perspective développementale pour clairement délimiter les frontières entre chaque profil de joueur. Ce modèle propose trois cheminements pour expliquer l'apparition de profils distincts de pratique de JHA. Tout en admettant la possibilité qu'il en existe d'autres, Nower et Blaszczynski (2005) décrivent les trois profils initiaux comme suit :

#1 : Comportementalement Conditionnés (« Behaviorally-Conditioned »). Les joueurs dits « Comportementalement Conditionnés » (CC) n'ont pas d'historique de psychopathologie. Les causes à la source de leurs problèmes de jeu sont principalement fonction des déformations cognitives découlant des effets de conditionnement liés aux récompenses du jeu, plutôt qu'à une difficulté à se contrôler. En fonction des influences sociales et des opportunités auxquelles ils sont soumis, les joueurs conditionnés fluctuent entre des périodes de jeu acceptable et de jeu excessif. Les problèmes liés au jeu (affectifs, comportementaux, cognitifs, sociaux, financiers) sont conçus comme découlant de leur pratique de JHA et non comme une cause du jeu.

#2 : Émotionnellement Vulnérables (« Emotionally Vulnerable »). À l'inverse, les joueurs « Émotionnellement Vulnérables » (EV) ont un historique de problèmes affectifs (p. ex., anxiété, dépression, faible estime de soi) et de stresseurs familiaux et/ou environnementaux. Ils sont aussi sujets aux mêmes distorsions cognitives que les joueurs CC. Cependant, la motivation principale derrière leur pratique de JHA serait qu'ils chercheraient à réguler leurs états affectifs (p. ex., pour se distraire de leur détresse). Ce profil de joueurs ressemblerait au profil « hypotension » de Jacobs

et au profil 1 de Sharpe. Ces joueurs seraient plus résistants au traitement, comparés au profil CC, à cause de leurs psychopathologies préétablies. La rémission du trouble lié au jeu serait conditionnelle à la guérison de ces psychopathologies.

#3 : Biologiquement Vulnérables (« Biologically Vulnerable »). Initialement surnommé « Antisocial-Impulsiviste », le troisième sous-groupe a depuis été rebaptisé « Biologiquement Vulnérable » (BV; Blaszczynski, 2014). Ces joueurs possèdent, comme les joueurs du profil EV, des prédispositions psychosociales. Toutefois, en plus d'une possible prédisposition affective, le profil BV est principalement caractérisé par des difficultés de nature extériorisée qui prennent la forme de conduites impulsives, délinquantes et antisociales (dont les fondements biologiques et génétiques sont bien documentés; Baker, Bezdjian et Raine, 2006). Ces caractéristiques sont centrales à la personnalité des joueurs BV et entrainent des comportements problématiques d'ordre légal et une consommation excessive et variée de substances psychotropes. Fait important, ces problèmes de comportement et d'impulsivité précèdent leurs problèmes de jeu et sont susceptibles d'expliquer leur cooccurrence avec d'autres problèmes, telles que la criminalité ou la consommation de drogues. Le groupe BV ressemblerait au profil « hypertension » de Jacobs et au profil 2 de Sharpe. Il serait le plus résistant au traitement et le plus persistant dans les problèmes de jeu.

Validation auprès des adultes

Quelques études ont tenté de démontrer la présence des trois profils du *Pathways Model* chez une population adulte (Ledgerwood et Petry, 2010; Moon, Lister, Milosevic et Ledgerwood, 2016; Nower, Martins, Lin et Blanco, 2013; Valleur et al., 2015). Nower et al. (2013) ont tenté de valider le modèle théorique auprès de répondants présentant des niveaux problématiques de JHA et

recrutés au sein d'un échantillon représentatif de la population américaine (i.e. la National Epidemiologic Survey on Alcohol and Related Conditions, 2001-2002). Ceux-ci devaient avoir joué au moins 5 fois dans la dernière année et présenter au moins 3 critères sur 10 du DSM-IV-TR en matière de jeu pathologique (n = 581). Cette étude a intégré les variables suivantes : anxiété, dépression, abus d'alcool, dépendance à la nicotine, automédication, impulsivité, trouble de la personnalité antisociale, problèmes légaux, tendances suicidaires, antécédents familiaux d'antisocialité et de consommation d'alcool, problèmes avec la loi, tendance suicidaire, mauvaises relations sociales, TDAH, mort ou divorce des parents, présence de quelconque trouble de la personnalité (à vie) et la présence d'altercations physiques dues à l'alcool. Grâce à des analyses de classes latentes, les auteurs ont aussi démontré la présence de trois profils correspondant aux trois cheminements proposés par le *Pathways Model*. Mais, contrairement à ce que le modèle suggère, la présence de TDAH n'a pas distingué les profils EV et BV. Il y a toutefois eu une présence accrue de troubles de la personnalité antisociale et d'abus de drogues au sein du profil BV.

Ledgerwood et Petry (2010) ont aussi utilisé le *Pathways Model* pour classifier des joueurs en traitement afin d'examiner leurs progrès cliniques en fonction de leur profils définis à partir de trois variables centrales au *Pathways Model* (niveau d'anxiété, dépression et impulsivité). De façon plus précise, les auteurs ont arbitrairement créé trois groupes conformes aux trois profils du *Pathways Model* (niveaux bas sur les trois variables; niveaux élevés d'anxiété et dépression; niveaux élevés sur les trois variables). Après avoir participé au même traitement, les participants des trois groupes ont fait des progrès. Cependant, les joueurs du groupe CC étaient les seuls à ne plus exhiber de problèmes de JHA. Cette étude n'avait pas pour objectif de dériver les divers profils de joueurs et ne se qualifie donc pas comme une étude de validation du *Pathways Model*,

mais elle est néanmoins intéressante et pertinente par rapport aux possibles conséquences cliniques de ce modèle.

Validation auprès des adolescents

L'étude de Gupta et al. (2012) est la seule étude à notre connaissance à avoir tenté de valider le *Pathways Model* avec une population d'adolescents. À l'instar de Nower et al. (2013), leur analyse s'est faite auprès de 109 adolescents présentant trois problèmes ou plus de jeu selon le DSM-IV. Les variables suivantes ont été sélectionnées pour les analyses : auto-dégradation, affect dépressif, problèmes familiaux, utilisation problématique de drogues ou alcool, TDAH, tendance à être impulsif, insensibilité sociale, problèmes familiaux, maltraitance infantile et tendances suicidaires. Leur étude a démontré la présence de cinq profils de joueurs, dont trois qui ressemblent aux profils du *Pathways Model*. Les profils additionnels comportaient un profil d'adolescents uniquement dépressifs et un autre profil d'adolescents affichant à la fois des troubles d'intériorisation et des troubles d'extériorisation (expliqué comme correspondant à une superposition des groupes EV et BV du *Pathways Model*). Par contre, les symptômes d'hyperactivité étaient présents chez 4 des 5 profils, alors que le *Pathways Model* suggèrerait que ceux-ci devraient principalement être présents chez le profil BV.

Avantages du Pathways Model

Ni la théorie de Jacobs (1986) ni celle de Sharpe (2002) n'intègrent l'idée d'une personnalité antisociale, alors que plusieurs études ont mis en évidence l'existence d'un profil distinct marqué par cette caractéristique (Milosevic et Ledgerwood, 2010; Nower et al., 2013). Ce profil a aussi présenté un niveau de sévérité de trouble lié aux JHA plus élevé que les deux autres profils, ce qui renforcerait l'idée d'un profil distinctif.

Le trait d'impulsivité a aussi été admis par ces auteurs comme étant un facteur de risque pour le développement de problèmes de JHA, mais aucun d'entre eux n'a proposé un profil de joueurs différencié à partir de cette caractéristique. Pourtant, certaines études ont démontré que certains joueurs (semblables au profil CC) ont des niveaux d'impulsivité plus faibles que d'autres, même si cela demeure un facteur général de prédiction des problèmes de JHA. L'impulsivité devrait donc servir pour différencier les profils de joueurs.

De plus, le modèles de Jacobs (1986) et de Sharpe (2002), quoique capables de fournir une explication pour les niveaux élevés d'un trouble lié aux JHA et utiles pour orienter le traitement, ne donnent pas beaucoup d'espace de manœuvre pour la prévention. Un modèle efficace de prévention doit pouvoir identifier les facteurs de risque avant qu'un trouble ne survienne, chose que seul le *Pathways Model* permet de faire. En somme, le *Pathways Model* semble être le plus complet des trois. Ses bases empiriques demeurent toutefois fragiles et incertaines en raison des limites méthodologiques des études précédentes.

Limites des études existantes

Au final, deux études cherchant à valider le *Pathways Model* (Gupta et al., 2012; Nower et al., 2013) ont trouvé une répartition uniforme de traits d'impulsivité entre les profils EV et BV (plutôt que seulement chez les BV). Quoique les symptômes de TDAH pourraient servir à prédire la sévérité du problème de JHA (Derevensky, Pratt, Hardoon et Gupta, 2007), il semblerait que l'impulsivité ne puisse pas servir de critère de différenciation entre les EV et BV, contrairement à ce que le *Pathways Model* stipule. Toutefois, les mesures d'impulsivité étaient variées : Nower et al. (2013) ont utilisé une mesure de TDAH et Gupta et al. (2012) ont mesuré la tendance à être impulsif avec le *Millon Adolescent Clinical Inventory*. Or, Nower et Blaszczynski (2005)

recommandent d'utiliser un outil spécifiquement destiné à la mesure de l'impulsivité. Donc, avant de conclure que l'impulsivité est un critère inadéquat pour différencier les EV et BV, il faut d'abord utiliser un outil de mesure approprié.

Ledgerwood et Petry (2010) ont créé leurs profils de manière arbitraire. Ils ont ainsi imposé un nombre précis de profils et la structure de ceux-ci était prédéterminée. Ces contraintes présupposent que le *Pathways Model* est valide et n'envisagent pas la possibilité d'un nombre différent de profils ou d'une structure alternative à celle prédéfinie. Afin de valider efficacement ce modèle, il faudrait permettre aux profils de surgir librement, en spécifiant seulement les variables suggérées par le modèle théorique). En laissant la porte ouverte à l'apparition de profils alternatifs, un résultat conforme au *Pathways Model* fournirait un appui important dans la validation du modèle. Finalement, toutes les études précédentes (avec des adultes ou avec des adolescents) utilisent une approche transversale (c.-à-d., problèmes de jeu mesurés en même temps que les facteurs de risque). Une telle approche ne permet pas de mettre en valeur la dimension développementale du modèle et, surtout, de distinguer les facteurs qui précèdent de ceux qui découlent des problèmes de jeu. L'utilisation d'un seul temps de mesure masque donc la trajectoire développementale sous-jacente. Dans les rares études qui adoptent une vision développementale (Nower et al., 2013), celles-ci reposent sur des données rétrospectives sujettes à des problèmes de mémoire et de reconstruction du passé (p. ex. « âge de la première dépression »). Finalement, aucune étude ne s'est penchée sur la stabilité temporelle des problèmes de JHA à travers les divers profils de joueurs, même si le modèle des parcours multiples fait des prédictions explicites à ce sujet. Notamment, on s'attendrait à voir un taux de stabilité plus élevé pour le profil BV, à cause de leurs prédispositions biologiques endurantes; pour le profil EV, le taux de stabilité dépendrait de l'état de la vulnérabilité émotionnelle sous-jacente (c.-à-d. si la dépression se maintient); et pour

le profil CC, un taux de stabilité plus faible serait attendu surtout lorsque le contexte social subit des changements importants comme lors de la transition entre l'adolescence et l'âge adulte.

Analyses de classes latentes

Quelques-unes des études de validation susmentionnées ont utilisé une analyse de classes latentes pour faire émerger les groupes distincts de joueurs (Gupta et al., 2012; Nower et al., 2013). DiStefano (2012) explique que les analyses de classes latentes sont un ensemble d'outils statistiques permettant d'identifier des profils au sein d'un échantillon en fonction des données présentées par un ensemble d'individus; lorsque les données sont de nature continue (comme cela est le cas dans cette thèse), on parle alors d'analyses de profils latents. Le nombre de sous-groupes n'est pas connu d'avance et émergent à partir des variables en jeu en fonction de tests de maximum de vraisemblance (classification à base de probabilités) et d'autres critères quantitatifs tels que le *Bayesian information criteria*, *Entropy*, et le *Vuong-Lo-Mendell-Rubin Likelihood Ratio Test* (Nylund, Asparouhov et Muthén, 2007).

Il n'existe pas de consensus en ce qui a trait à la taille d'échantillon minimale pour réaliser ces analyses. En effet, cela dépend du nombre final de profils ou encore de la capacité des variables choisies à clairement séparer les divers profils. Des études de simulation de type *Monte Carlo* permettent, quant à elles, d'avoir une idée des tailles d'échantillon requises pour correctement identifier le nombre de profils sous-jacents dans une population donnée. Ces études sont conçues de sorte à répéter les analyses de classes latentes plusieurs fois (p. ex., 100 ou 1000 itérations) en sélectionnant au hasard des sous-échantillons de *n* participants, et en répétant cette procédure en faisant varier le nombre *n* (Muthén et Muthén, 2002). Pour cela, il faut connaître au préalable le nombre réel de profils, afin d'évaluer si les divers modèles simulés arrivent à la bonne conclusion.

Des études de simulation *Monte Carlo* ont conclu que les indicateurs d'ajustement généralement utilisés pour les analyses de profils latents peuvent démontrer une puissance statistique de plus de .90 avec un échantillon de seulement 150 participants (Dziak, Lanza et Tan, 2014).

Objectifs et structure de la thèse

Les objectifs suivants ont orienté les trois articles qui composent cette thèse; nous les présentons tel qu'ils sont regroupés au sein de chaque article. Article 1 : (1) Vérifier s'il est possible de dégager au sein de joueurs problématiques adolescents ou jeunes adultes (i.e. 16 et 23 ans) des profils compatibles avec ceux suggérés par le modèle des parcours multiples à partir de variables mesurées au début de l'adolescence (i.e. 12 et 14 ans). (2) Contraster ces profils en fonction des caractéristiques de jeu des participants qui les composent (c.-à-d., âge d'initiation, fréquence et problèmes de jeu à 16 et à 23 ans). Article 2 : (3) Utiliser les mêmes variables de risque d'ordre personnel identifiées dans l'article 1 et générer des groupes à risque à partir d'échantillons populationnels suivis depuis la pré-adolescence (i.e. 12 ans). (4) Identifier des facteurs de nature sociale susceptibles de modérer ou de compenser le risque encouru par les participants des divers profils à l'égard de l'émergence de problèmes de jeu à la fin de l'adolescence ou au début de l'âge adulte (i.e. 16 et 23 ans). (5) Voir si les résultats en lien avec les objectifs 3 et 4 s'appliquent autant aux femmes qu'aux hommes. Article 3 : (6) Proposer une approche de prévention et d'intervention différenciée adaptée aux profils de joueurs tels qu'identifiés aux points précédents et dans les autres études empiriques pertinentes.

Hypothèses

Article 1 (étude empirique 1). Nous nous attendons à voir émerger au moins trois profils de joueurs définis à partir de leurs caractéristiques personnelles à 12 et 14 ans, et jugés à

risque ou à problèmes de JHA à partir de leur pratique de jeu à la fin de l'adolescence (16 ans) et au début de l'âge adulte (23 ans); ces profils correspondraient aux trois cheminements proposés par le *Pathways Model*. Notamment, le premier profil (CC) ne présenterait pas d'indicateurs de psychopathologie significative à 12-14 ans. Il aurait aussi le degré de sévérité de jeu problématique le moins élevé à 16 et à 23 ans. En revanche, le profil EV serait caractérisé par une tendance à avoir des troubles intériorisés à 12-14 ans et un niveau moyen de sévérité de problèmes de jeu. L'âge d'initiation aux JHA pour les jeunes de ce profil serait intermédiaire par rapport à celui des jeunes du profil CC et du profil BV, dont l'âge d'initiation aux JHA serait le plus précoce. Les jeunes du profil BV seraient par ailleurs caractérisés par un niveau élevé d'impulsivité, la présence de comportements antisociaux et d'une consommation excessive de psychotropes au début de l'adolescence. Ce dernier profil présenterait le niveau de problèmes de JHA le plus élevé. En lien avec l'objectif 5, les hommes devraient avoir moins de chance de se retrouver dans le profil EV, comparés aux femmes, et plus de chances de se retrouver dans le profil BV.

Article 2 (étude empirique 2). Nous nous attendons à voir émerger les mêmes profils de joueurs, mais cette fois-ci auprès de l'ensemble des participants (c.-à-d., en incluant les non-joueurs). Le but étant de permettre ensuite, en lien avec l'objectif 4, une exploration des facteurs de compensation ou de protection de nature sociale associés de manière prédictive à l'émergence des problèmes de jeu à 16 et 23 ans. Étant donné le caractère biologique des prédispositions du profil BV, nous nous attendons à ce que le risque de problèmes de jeu encouru par ces participants ne soit pas particulièrement affecté par des facteurs sociaux. En comparaison, le profil EV devrait réagir positivement au soutien social de type affectif (p. ex., attachement aux pairs et à la famille), et le profil CC devrait bénéficier de tous les facteurs de protection ou de compensation généralement associés aux JHA.

Article 3 (recension d'écrits). Aucune hypothèse n'est formulée en lien avec le troisième article et l'objectif 6; nous suggérons une approche novatrice pour les programmes de prévention de jeu problématique.

Article 1. A Longitudinal Empirical Investigation of the Pathways Model of Problem Gambling

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A Longitudinal Empirical Investigation of the Pathways Model of Problem Gambling

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Compliance with Ethical Standards

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Abstract

Background and Aims: The Pathways Model of Problem Gambling suggests the existence of three developmental pathways to problem gambling, each differentiated by a set of predisposing biopsychosocial characteristics: Behaviorally Conditioned (BC), Emotionally Vulnerable (EV), and Biologically Vulnerable (BV) gamblers. This study examined the empirical validity of the Pathways Model among adolescents followed up to early adulthood. **Design**: A prospective-longitudinal design was used, thus overcoming limitations of past studies that used concurrent or retrospective designs. **Setting:** Two samples were used: a) a population sample of French-speaking adolescents (N = 1,033) living in low socio-economic status (SES) neighborhoods from the Greater Region of Montreal (Quebec, Canada), and b) a population sample of adolescents (N = 3,017), representative of French-speaking students in Quebec. **Participants**: Only participants with at-risk or problem gambling by mid-adolescence or early adulthood were included in the main analysis (n = 180). **Analyses:** Latent Profile Analyses were conducted to identify the optimal number of profiles, in accordance with participants' scores on a set of variables prescribed by the Pathways Model and measured during early adolescence: depression, anxiety, impulsivity, hyperactivity, antisocial/aggressive behavior, and drug problems. **Findings**: A four-profile model fit the data best. Three profiles differed from each other in ways consistent with the Pathways Model (i.e., BC, EV, and BV gamblers). A fourth profile emerged, resembling a combination of EV and BV gamblers. **Conclusions:** Four profiles of at-risk and problem gamblers were identified. Three of these profiles closely resemble those suggested by the Pathways Model.

Keywords: gambling, adolescence, early adulthood, pathways model, longitudinal design

Introduction

Problem-gambling (PG) becomes a disorder when a gambler exhibits a pre-set number of problems related to their gambling practice, from a legal, social, and self-control point of view (American Psychiatric Association 2013). The prevalence of PG among the general adult population varies between 2-5%, depending on the country studied (Williams et al. 2012; Shaffer and Hall 2001). This rate is higher among adolescents and young adults (between 4-7%; Shaffer and Hall 2001; Villella et al. 2011).

Although variable-oriented research has consistently identified the same set of risk factors for PG (e.g., impulsivity, hyperactivity, emotional problems, delinquency, or drug problems), some contradictory findings have also emerged. For example, impulsivity has been extensively documented as predicting PG (Dussault et al. 2011; Johansson et al. 2009; Nower et al. 2004). However, some researchers have failed to find a link (Dannon et al. 2010; Langewisch and Frisch 1998). Seemingly contradictory results in the literature may be due to the erroneous practice of combining all problem gamblers into a single group. There is also accumulating evidence that young problem gamblers, like their adult counterparts, are not a homogenous group (Gupta et al. 2012; Milosevic and Ledgerwood 2010; Nower et al. 2013).

The Pathways Model

Many researchers and clinicians have devised different classifications of gamblers (Blaszczynski and Nower 2002; Moran 1970; Sharpe 2002). However, in Milosevic and Ledgerwood's (2010) analysis of 17 different studies that have classified PG into different subgroups, the authors concluded that a three-group model – resembling the three pathways proposed by the Pathways Model (Blaszczynski and Nower 2002) – appeared most consistently.

The Pathways Model proposes three developmental pathways to PG, each differentiated by a set of pre-disposing risk factors and consequences from gambling: Biologically Vulnerable (BV), Emotionally Vulnerable (EV), and Behaviorally Conditioned (BC) gamblers.

BV problem gamblers display biopsychosocial vulnerabilities. These gamblers exhibit a range of behaviors typically associated with impulse-control disorders, which have been found to be partly under genetic influence and related to specific neuro-cognitive deficits (Dussault et al. 2011; Lobo and Kennedy 2009; Nower et al. 2004). They also demonstrate high levels of antisocial or aggressive behaviors unrelated to gambling, as well as multiple drug use. This subgroup exhibits the most severe levels of PG, and tends to start gambling the earliest. They also tend to persist in their gambling habits.

EV gamblers suffer from an underlying affective dysregulation. Negative family background experiences or past trauma partially contribute to their emotional vulnerability. PG develops in response to their affective state, as a means of emotional regulation. In line with this subgroup, depression has been concurrently and predictively linked to PG (Dussault et al. 2011; Jacobs 2005). Members of this subgroup may also display other maladaptive behaviors (e.g., alcohol abuse) to cope with their emotional difficulties. Their problem gambling levels are fairly stable, but less severe than BV gamblers.

Finally, BC gamblers do not show signs of any biological or affective predisposition. They start gambling for reasons of excitement and socialization (i.e., peer pressure), but fluctuate between light and heavy problem gambling because of conditioning (i.e., differential reinforcement due to gains and losses). Affective problems (e.g., depressive symptomatology) that may be associated with their gambling are understood as a consequence, rather than a precedent,

of their gambling. They experience fewer gambling problems than their EV and BV counterparts, and tend to start gambling the latest. As a group, they also tend to be the first to desist from gambling once they enter into adult roles during early adulthood, but exceptions may exist.

Empirical support for the model

A number of studies have tested the Pathways Model with adult samples (Valleur et al. 2015; Nower et al. 2013). For example, in a study among 581 problem gamblers who participated in the US National Epidemiologic Survey on Alcohol and Related Conditions, Nower et al. (2013) identified three profiles evocative of the subtypes covered by the Pathways Model. In contrast, only one study has examined the applicability of this model to an adolescent population (Gupta et al. 2012). This study identified five subgroups of problem gamblers (n = 109), three of which resembled those described by the Pathways Model. However, all validation studies so far used a cross-sectional design (i.e. all measures collected at the same time). This approach makes it impossible to distinguish co-occurring mental health conditions that precede problem gambling from those that accompany it. For example, in the case of EV gamblers, a cross-sectional approach cannot help establish whether emotional problems preceded their gambling problems. To address this shortcoming and to achieve a stronger empirical validation of the Pathways Model, a longitudinal design – that collects measures unique to each pathway prior to the emergence of PG – must be used.

Study Objectives

The first aim of this study was to identify the optimal number of PG profiles in terms of personal characteristics (i.e., impulsivity, hyperactivity, anxiety, depressive symptoms, antisocial/aggressive behavior, and drug problems) in early adolescence before the emergence of

PG. The second aim was to validate the empirically derived profiles by contrasting them with regards to age of onset of gambling behavior, and other gambling characteristics (i.e., number of gambling problems and frequency of gambling behavior) by mid adolescence (age 16) and early adulthood (age 23). Two samples from Quebec (Canada), both of which used a longitudinal design, were used. The first sample ("Sample A") consists of low socioeconomic status (SES) French-speaking adolescent males (high-risk group). The second sample ("Sample B") is representative of all Quebec students attending francophone schools. Gambling problems were measured in both samples when participants were 16 and 23 years old. Personality correlates (i.e., impulsivity, hyperactivity, depression, anxiety, and antisocial/aggressive behavior) were collected at age 12 using teacher ratings. Teacher ratings were chosen over parent ratings because the former have been found to better predict problematic behavior in children (Power et al. 1998; Verhulst et al. 1994). Drug problems were assessed using a self-report measure at age 14. Self-reports were chosen because young adolescents are likely to hide their drug use from their teachers, resulting in an underestimation of drug problems from the teacher's perspective. Moreover, surveys in Quebec have shown that adolescents generally start developing significant drug problems around the age of 14 years (Dubé et al. 2009). Given that the two samples were merged to maximize the number of problem gamblers who would be included in the analyses, we also examined whether the results varied from one sample to the other.

Although we used two population-based samples, only at-risk or problematic gamblers were selected for this study. Since the Pathways Model serves to identify developmental pathways leading to PG, it makes sense to only target participants who have developed some levels of gambling problems. Past studies that have examined the Pathways model used a similar strategy.

Hypotheses

As outlined by the Pathways Model, we expected to find one subgroup (i.e., BV) with higher levels of impulsivity, hyperactivity, drug problems, and antisocial/aggressive behavior during early adolescence relative to all other subgroups. Another subgroup (i.e., EV) was expected to display higher levels of depression and anxiety during early adolescence, relative to all other groups. Also, we expected to find a third and final subgroup (i.e., BC) with low levels on all indicator variables, compared to the other two subgroups. Finally, we expected BV gamblers to report a higher number of gambling problems both during late adolescence and early adulthood and an earlier age of onset of gambling behavior compared to EV gamblers, who in turn were expected to report more gambling problems and an earlier age of onset of gambling behavior than BC gamblers.

Method

Participants

Sample A. Participants were part of an ongoing longitudinal study that started in 1984 with 1,033 French-speaking kindergarten boys attending schools in economically disadvantaged areas in Montreal, Quebec, Canada. Participants included in the initial sample represented 87% of all boys attending the 53 schools that participated in the study. Data used in this study were collected when the participants were 12, 14, 16 and 23 years old. This non-probabilistic convenience sample represents a high-risk group (i.e., low SES males).

Sample B. Participants were part of the ongoing Québec Longitudinal Study of Kindergarten Children, a representative sample of the French-speaking children attending kindergarten in the

province of Quebec, Canada, in 1986–1987. From an initial pool of 6,397 six-year old children, 2,000 participants were selected among the children for whom both parent reports and teacher reports were available, to ensure good participation during follow-up. Out of all the parents and teachers initially contacted, 63% of parents and 68% of teachers provided complete reports. An additional 1,017 participants with elevated scores on a disruptive behavior scale (Tremblay et al. 1991) were included to provide sufficient statistical power when examining problematic behavior. The final, partly probabilistic, partly high risk, sample consisted of 3,017 participants (58% male). See Rouquette et al. (2014) for a complete explanation of sampling procedure. Data used in this study were collected when the participants were 12, 14, 16 and 23 years old.

Procedure

The study was approved by the School Board and active written consent was obtained from parents in both samples. All participants actively provided their assent to take part in the study. Participants were also informed before data collection that their answers were strictly confidential and that they could end their participation at any time. All measures were administered in the Spring towards the end of the school year. Unless otherwise stated, the same instruments were administered in both samples.

Measures

Teacher Ratings. When participants were 12 years old, teachers completed the Social Behavior Questionnaire (Tremblay et al. 1987). Each item was rated on a 3-point scale (0: Does not apply, 1: Applies sometimes, and 2: Frequently applies). From this instrument, items were selected to assess Impulsivity, Hyperactivity, Depression, Anxiety, and Antisocial/Aggressive behavior. Item scores were summed to obtain a total score for each variable.

Impulsivity. A total of 7 items (e.g. "acts without thinking") were included in this scale (Sample A: Cronbach's α = .89; Sample B: α = .88). Higher scores indicate higher levels of impulsivity. Total scores varied between 0 and 13.

Hyperactivity. A total of 3 items (e.g. "squirmy, fidgety child") were included in this scale (Sample A: α = .86; Sample B: α = .85). Higher scores indicate higher levels of hyperactivity. Total scores varied between 0 and 4.

Depression. A total of 4 items (e.g. "appears miserable, unhappy, tearful or distressed") were included in this scale (Sample A: α = .62; Sample B: α = .63). Higher scores indicate higher levels of depression. Total scores varied between 0 and 7.

Anxiety. A total of 3 items (e.g. "is worried about many things") were included in this scale (Sample A: α = .86; Sample B: α = .84). Higher scores indicate higher levels of anxiety. Total scores varied between 0 and 6.

Antisocial/Aggressive behavior. A total of 9 items (e.g. "has stolen things on one or more occasions.") were included in this scale (Sample A: α = .87; Sample B: α = .87). Higher scores indicate higher levels of antisocial/aggressive tendencies. Total scores varied between 0 and 16.

Self-Reported Ratings. Participants reported on their drug and alcohol-related problems when they were 14 years old. They also reported on their gambling-related problems when they were 16 and 23 years old.

***Drug and Alcohol Problems*.** The Personal Experience Screen Questionnaire (PESQ; Henly and Winters 1988) was used to gauge illicit drug and alcohol problems. This test examines 18 potential problems (e.g. "When you have consumed alcohol or drugs, how many times have you

seen, felt, or heard things that were not really there?"), with item scores ranging from 1 (never) to 4 (often). Higher rates indicate more severe levels of drug and alcohol problems. Total scores varied between 18 and 43 (Sample A: $\alpha = .93$; Sample B: $\alpha = .90$). The following three items were included to detect if participants were lying: "In order to pay for alcohol or drugs, how many times have you conned people with counterfeit money?", "How many times have you consumed alcohol or other drugs (including cigarettes and marijuana) while parachuting?", and "How many times have you obtained alcohol or drugs from a police officer?" If a participant responded positively to one of these questions, the overall score was deleted and treated as missing data.

Gambling Problems and Gambling Frequency. In accordance with recommendations made by Nower and Blaszczynski (2005), we used adolescent and adult versions of the South Oaks Gambling Screen to assess gambling problems (SOGS-RA at age 16 years and SOGS at age 23 years; Lesieur and Blume 1987; Winters et al. 1993). The SOGS is a self-reported questionnaire designed to assess problems related to past-year gambling practices. The SOGS-Revised for Adolescents (SOGS-RA) comprises 12 Yes/No items, whereas the adult version consists of 20 items. Both instruments share 11 items. However, the twelfth item in the adolescent version (i.e., "borrowing money to gamble or to pay gambling debts") is split into nine items in the adult version, with each source of borrowing added up independently (e.g., borrowing from family and friends would result in a score of 2 in the adult version, and a score of 1 in the adolescent version). An additional nine items were used to analyze the frequency (0: never, 1: less than once a month, 2: once a month, 3: once per week, 4: daily) at which participants engaged in a variety of gambling activities (e.g., play cards for money). A frequency score was computed by adding scores for each gambling activity.

The SOGS-RA has been validated for screening gambling problems among adolescents aged 14 to 20 years old (Chiesi et al. 2013), and the SOGS has been validated in a variety of settings (i.e., Gamblers Anonymous, university students, and hospital employees; Lesieur & Blume, 1987). In Sample A, Cronbach's α was .76 and .88 at ages 16 and 23, respectively. In Sample B, Cronbach's α was .76 and .75 at ages 16 and 23, respectively.

In Sample A, age of onset of gambling was prospectively assessed starting at age 12 years, by asking participants if they had gambled in the past year. In Sample B, age of onset was retrospectively assessed at age 23 by asking participants at what age they first gambled.

Results

Data Treatment and Preliminary Analyses

Sample A. Of the initial sample of 1,033 participants, 669 (65%) and 504 (49%) had valid data on gambling at ages 16 and 23, respectively. Among those with gambling data for at least one time-point (n = 801), 84 (10.5%) displayed at-risk levels of gambling at one or more time-points and were included in our PG classification: 55 (8.2%) had two or more problems at ages 16 and 32 (6.2%) had four or more problems at age 23. Two or more gambling problems in adolescence corresponds to Level 2 of the Levels terminology used by Shaffer and Hall (2001; i.e., at-risk or transitional gambling), whereas 4 or more gambling problems in early adulthood corresponds to one less problem than that required to be classified as a "probable pathological gambler" by the SOGS (i.e., problem gambler). These criteria were similar to those used by Gupta et al. (2012) and by Nower et al. (2013).

Sample B. Of the initial sample of 3,017 participants, 1,798 (60%) and 1,445 (48%) had valid data on gambling at ages 16 and 23, respectively. Among those with gambling data for at least one time-point (n = 2248), 114 (5%) displayed at-risk levels of gambling at one or more time-points and were included in our PG classification: 101 (5.6%) had two or more problems at age 16 and 16 (1.1%) had four or more problems at age 23.

Considering that the main goal of the study was to identify antecedents of PG, and that PG may emerge in adolescence or adulthood, problem gamblers identified at either age period (i.e., mid-adolescence or early adulthood) from both samples were combined for all analyses. This strategy also allowed us to maximize sample size and hence boost statistical power. When conducted on gambling data at ages 16 and 23 years, Little's (1988) test was not significant, χ^2 (2, N = 4050) = 4.062, p = .131, indicating that gambling data were *Missing Completely At Random* (MCAR). Therefore, cases with missing data could be safely ignored when selecting our subset of problem-gamblers without introducing significant bias.

Preliminary analyses were conducted to check the distributional properties of our data. Univariate outliers were winsorized (Tabachnick and Fidell 2012); raw scores were re-coded one unit larger than the next more extreme score in the distribution. Considering many variables showed a skewed distribution, we used a Maximum Likelihood estimation with Robust standard errors (MLR; B. Muthén and Asparouhov 2002); an estimation method specifically conceived for non-normally distributed data.

Of the 198 participants selected, 18 had missing data on all indicator variables at ages 12 or 14 and were excluded from the PG classification. Of the remaining 180 participants, 21 were female (21.4% of Sample B; 11.7% of the combined samples), 11% had missing data on the

teacher-rated scales at age 12; and 26% had missing data on the self-rated scale of drug abuse at age 14 years (including 6 participants whose scores were deleted because they failed the built-in lie-detecting items). Missing data was handled using the Full Information Maximum Likelihood method for the Latent Profile Analysis (see below), and using Multiple Imputation (10 iterations) for subsequent analyses. These methods are appropriate when data is missing completely at random, which was the case according to Little's test (Little 1988), χ^2 (6, N = 180) = 7.91, p = .245. Table 1 summarizes descriptive statistics for the main variables used in this study, after combining problem gamblers from Samples A and B.

Latent Profile Analysis

Latent Profile Analysis (LPA; Collins and Lanza 2010) is a type of Latent Class Analysis, a set of statistical tools that allow identification of profiles according to a set of continuous indicator variables (DiStefano 2012). The number of profiles is not known beforehand and the optimal solution is found by using a set of model fit indicators, combined with maximum-likelihood tests (probability-based classification). Following Celeux and Soromenho (1996) and Nylund et al. (2007), the following fit indicators were used: *Bayesian information criterion (BIC)*, *Entropy*, and *Lo-Mendell-Rubin Adjusted Likelihood Ratio Test (LMR-ALRT)*. The empirically-derived profiles were further validated by testing for differences between profiles across all indicator variables using a MANOVA. LPA was performed, using a step-wise mixture modeling technique with MPlus 6.12 (L. K. Muthén and Muthén 2010), to identify the optimal number of PG profiles based on the following indicator variables suggested by the Pathways Model: teacher-rated impulsivity, depression, anxiety, hyperactivity, and antisocial/aggressive behavior at age 12; self-reported drug problems at age 14.

The LPA was conducted with the 180 participants who displayed at-risk or problematic levels of gambling either at 16 or at 23 years of age. Model fit indices are reported in Table 2. Two- and three-class models separated groups based on their degree of severity across all variables (i.e., high on all vs. low on all). Furthermore, although the five- and six-class models had lower BIC scores, this indicator kept decreasing no matter how many classes were added. The tendency for the BIC to consistently decrease has been observed across many other studies that have used continuous indicators, rather than categorical, to determine the optimal number of classes (Nower et al. 2013; Marsh et al. 2009; Geiser et al. 2014). Therefore, in accordance with Marsh et al.'s (2009) recommendations, the best-fitting solution was selected based on qualitative and quantitative changes between the classes. A four-class model turned out to be optimal, as demonstrated by the statistically significant *LMR-ALRT*, higher entropy, and profiles that were qualitatively meaningful (i.e., not simply separated into high, medium, low levels across all variables).

Three of the four profiles closely resembled the BC (n = 105; 58%), EV (n = 31; 17%), and BV (n = 36; 20%) pathways. The fourth profile (n = 8; 5%) had high levels on all indicator variables, and resembled an overlap between EV and BV profiles, hereafter dubbed Bio-Emotionally Vulnerable (BEV) gamblers; a similar profile was also found by Gupta et al., 2012).

Confirming PG Profiles

All subsequent analyses were conducted with SPSS version 22 (IBM 2013). Participants were classified into one of the four profiles identified, based on their most likely class membership. Participants classified into the fourth pathway were not included in these analyses because of the low participant count (n = 8), resulting in a subset of 172 problem gamblers. First, a chi-square

test of independence was conducted to test equality of gender distribution across gambling groups. The test was not statistically significant, χ^2 (2, N = 172) = 2.04, p = .360. Second, a MANOVA was conducted to validate the distinctiveness of all profiles identified. All six indicator variables (i.e., Impulsivity, Depression, Anxiety, Hyperactivity, Antisocial/Aggressive Behavior, and Drug Problems) were included as dependent variables. Drug Problems was the only variable to show excessively high skewness and kurtosis levels, and was therefore subjected to a logarithmic transformation prior to analyses.

Profiles served as a fixed factor (i.e., BC, EV, and BV). The results from the MANOVA indicated a significant overall difference between groups across indicator variables: average Pillai's Trace = 1.13, F (12, 330) = 35.61, p < .001, partial eta squared = .56, power to detect the effect was 1.00. The results remained significant after controlling for Gender and SES. Priority was given to Pillai's Trace because it has been shown to be very robust to violations of MANOVA assumptions (Scheiner 2001), such as heteroscedasticity. Levene's test of equality of error variances yielded satisfying results for all variables except Antisocial/Aggressive, F (2, 169) = 7.32, p < .001, Hyperactivity, F (2, 169) = 3.07, p = .049, and Drug Problems, F (2, 169) = 4.19, p = .017. Univariate F tests for between-subject effects revealed a significant difference between groups for: Impulsivity, F (2, 169) = 106.05, p < .001; Hyperactivity, F (2, 169) = 108.50, p < .001; Depression, F (2, 169) = 71.01, p < .001; Anxiety, F (2, 169) = 38.44, p < .001; Antisocial/Aggressive Behavior, F (2, 169) = 36.62, p < .001; and Drug Problems, F (2, 169) = 5.35, p = .006. A second MANOVA also included Sample (A or B) as a second factor in order to test for a Pathway * Sample interaction, which was not significant, Pillai's Trace = 0.09, F (12, 324) = 1.32, p = .205. Therefore, the differences between pathways were not due to belonging to one sample or another.

Post hoc tests were conducted to examine specific differences between groups. The Student-Newman-Keuls test (appropriate for unequal group sizes) was used for this purpose. Table 3 summarizes mean scores of all variables for each group. Some significant differences ($\alpha = .05$), consistent with the Pathways Model, are particularly worth noting:

- EV gamblers were rated by their teacher as manifesting more depressive symptoms than BV gamblers, who in turn had higher levels than BC gamblers;
- EV gamblers were rated by their teachers as manifesting more anxiety symptoms than BV gamblers, who in turn had higher levels than BC gamblers;
- BV gamblers were rated by their teachers as manifesting more impulsive symptoms than EV and BC gamblers;
- BV gamblers were rated by their teachers as manifesting more hyperactivity symptoms than EV and BC gamblers;
- BV gamblers were rated by their teachers as manifesting more antisocial/aggressive behaviors symptoms than EV and BC gamblers; and
- BV gamblers reported more drug problems compared to EV and BC gamblers.

Validating PG Profiles

Gambler profiles (i.e., BC, EV, and BV) were then compared in terms of their gambling practices (Age of Onset, Frequency, and Problems; mean scores are reported in Table 3). First, a series of ANOVAs were conducted. No difference between groups appeared with regards to Age of Onset, $F(2, 169) = .76$, $p = .469$. Likewise, no differences were found with regards to Gambling

Frequency at both time-points: age 16, F (2, 169) = 1.40, p = .249; age 23, F (2, 169) = 0.92, p = .400.

The comparison for gambling problems required a different analytical strategy since gambling problems are *count* data. A choice had to be made between a Poisson and a negative binomial regression model. As there was a high level of overdispersion in the data (i.e. variance 2 to 3 times greater than the mean, for both time-points), a negative binomial regression model was estimated (Gardner et al. 1995). BV gamblers were set as a reference group, and served as a basis for comparison with the two other groups (i.e., BV vs. BC, BV vs. EV). SES and gender were included as covariates.

At age 16 years, the omnibus likelihood ratio χ^2 test revealed no effects of the independent variables: χ^2 (4, N = 172) = 2.31, p = .679, indicating that there was no difference between pathways. At age 23, however, a significant effect was found: χ^2 (4, N = 172) = 21.49, p < .001. The gender covariate was significant (p = .001), indicating that males had approximately 1.27 more problems than females. Above and beyond the significant effect of the gender covariate, BV gamblers had approximately .48 (p = .045) more gambling problems compared to their BC counterparts, and .66 more gambling problems compared to EV gamblers (p = .053). When controlling for gambling problems at age 16, the only remaining difference was between BV and BC gamblers (p = .033)

Finally, considering that participants were selected based on whether or not they displayed PG at either age 16 or 23 years (i.e., could have displayed PG at age 16 but not 23, or vice-versa), another series of chi-square tests of independence were conducted to test whether PG prevalence differed between pathways at each time-point. The test was not significant at age 16, χ^2 (2, N =

172) = 1.87, p = .393. However, results were significant at age 23, χ^2 (2, N = 172) = 6.78, p = .034. In other words, among problem-gamblers at age 16, the proportion belonging to each profile was similar to the initial percentages identified through the LPA. However, at age 23 years, the proportion belonging to the BV subgroup increased (i.e., they were over-represented), the proportion of BC gamblers decreased (i.e., they were under-represented), and the proportion of EV gamblers remained stable.

Discussion

The Pathways Model is a developmental etiological model with a set of predisposing factors that can lead at-risk gamblers to follow one pathway over another. The current study is the first to examine the model's empirical validity for adolescents using a longitudinal design that can disentangle the chronological ordering between predisposing factors and gambling behavior.

In line with the first study objective, four profiles of problem gamblers were identified based on personal characteristics measured in early adolescence, three of which closely resemble the subtypes described by the Pathways Model: Behaviourally Conditioned (58%), Emotionally Vulnerable (17%), and Biologically Vulnerable problem gamblers (20%), and a fourth sub-type displaying an overlap of Biologically and Emotionally Vulnerable (BEV) characteristics (5%). These findings are partially concordant with those reported by Gupta et al. (2012) and Nower et al. (2013). BC, EV, and BV profiles were also identified in both of these studies. The BEV profile was only identified in the Gupta et al. (2012) study, which examined adolescent gambling, suggesting that this profile may be specific to this developmental period.

As hypothesized, EV gamblers displayed higher levels of anxiety and depressive symptoms compared to all other groups. Also, BV gamblers were more impulsive and hyperactive than their

EV and BC counterparts. However, contrary to the validation studies by Gupta et al. (2012) and Nower et al. (2013), a hyperactivity component was not present in EV gamblers. Nonetheless, this result is concordant with the Pathways model. With regards to drug problems, our results are also consistent with the Pathways Model: BV gamblers displayed higher scores compared to BC and EV gamblers, who did not differ from each other.

With regards to the second study objective (i.e., comparing profiles in terms of gambling behavior), the three pathways identified in this study did not differ with regards to age of onset of gambling behavior. This conflicts with theoretical predictions, considering that BV gamblers are hypothesized to start gambling earlier and to gamble more frequently than their EV and BC counterparts. Results with respect of age of onset, however, could have been different had we started assessing gambling behavior before the age of 12, as some children may have started gambling at an earlier age (Vitaro and Wanner 2011). Moreover, no differences were found between groups at age 16 with regards to gambling problems on the SOGS-RA, possibly because age 16 corresponds to the peak of gambling problems for all the groups (Wanner et al. 2006). However, at age 23, BV gamblers reported more gambling problems compared to their BC counterparts, which is in line with theoretical predictions. Also, BV gamblers' problems seemed to persist, whereas EV and BC gamblers seemed to desist, as evidenced by BV gamblers' higher rate of PG at age 23.

By showing that characteristics correlate differently with specific pathways, the present results also help reconcile contradictory findings found in past studies. For instance, Dannon et al.'s (2010) finding that PG was not associated with impulsivity could be due to the fact that their sample consisted mainly of BC and EV gamblers. Furthermore, McCormick et al.'s (1984) finding that depression does not always precede gambling problems might be explained by the qualitative

difference between BC and EV gamblers. This does not mean, however, that personal characteristics are the only risk factors associated with problem gambling. Other risk factors include environmental as well as structural factors (Johansson et al. 2009).

Our findings support the need to adopt a differentiated view with respect to etiological models of adolescent PG. More specifically, our findings suggest that different personality characteristics may combine to put some individuals at risk for problem gambling, although the origins of these characteristics and their possible interplay with socio-environmental factors remain unknown. Our findings also point to the need to shift toward a differential targeted prevention approach in regard to PG in adolescents. Considering that disordered gambling has recently been classified among other substance use disorders (American Psychiatric Association 2013), prevention program developers should find inspiration from existing targeted prevention programs that have focused on substance use by targeting the specific personal and socio-environmental risk factors experienced by different subgroups (Conrod et al. 2013; Allami and Vitaro 2015).

Strengths and Limitations

This study has several strengths. First, adopting a longitudinal perspective allowed us to disentangle personal characteristics generally associated with PG from the specific gambling profiles they are linked to. Second, we used different raters to assess participants' characteristics and gambling problems, thus reducing bias that could result from shared method variance. Third, we included longitudinal measures of gambling characteristics to further validate the gambling profiles. Despite these assets, some limitations must be noted. First, even with our large combined sample, the relatively small sample size of problem gamblers may have limited our ability to

effectively identify differences between groups. For example, the fourth profile of PG could have been included in subsequent analyses had a larger sample been used. Statistical significance might also have been achieved on comparisons of gambling frequency, problems and gender. Second, there was a relatively large amount of missing data. Although handled with state of the art statistical techniques, this may limit the generalizability of our results. Third, the instruments used to assess personality predispositions (i.e., impulsivity, hyperactivity, depression, anxiety, and antisocial/aggressive behavior) were relatively short, with the number of items per scale ranging from 2 to 7, and may only be considered as screening tools, rather than thorough diagnostic assessments. Finally, although most measures were collected prospectively, some were collected using a retrospective strategy (i.e., age of onset for Sample B) and may thus have been affected by recall bias.

Despite these limitations, the current study showed that four profiles of adolescent at-risk and problem gamblers can be empirically derived, three of which closely resemble those suggested by the Pathways Model, thereby contributing to the accumulating evidence supporting the validity of this theoretical model.

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Tables

Table 1

Descriptive Statistics (Problem Gamblers, N=180)

Variable (Age)	*M*	*SD*	Skewness	Kurtosis
Antisocial/Aggressive behavior (12)	3.21	3.83	1.30	0.97
Impulsivity (12)	3.39	3.60	0.92	-0.25
Hyperactivity (12)	1.30	1.60	1.07	-0.02
Anxiety (12)	2.03	1.60	0.40	-0.68
Depression (12)	1.45	1.66	1.25	1.32
Drug problems (14)	20.31	5.20	3.56	14.89
Log(Drug Problems)	0.61	0.93	1.37	0.75
Gambling problems (16)	2.86	2.06	1.52	4.14
Gambling frequency (16)	6.52	4.39	1.13	2.36
Gambling problems (23)	1.81	3.09	1.80	2.25
Gambling frequency (23)	3. 82	3.54	1.72	3.54
Age of Gambling Onset	12.50	3.31	0.38	-0.50

Note. M: Mean, SD: Standard deviation.

Table 2

Model Fit Indicators for Latent Profile Analysis (N=180)

Model	BIC[a]	Entropy	LMR-ALRT[b] (*p* value)
2-class	4347	.847	.000
3-class	4260	.864	.063
4-class	**4168**	**.874**	**.037**
5-class	4142	.863	.523
6-class	4111	.868	.438

Note. Best-fitting model is shown in bold.

[a] Bayesian Information Criterion

[b] Lo-Mendell-Rubin Adjusted Likelihood Ratio Test

Table 3

Group Means (M) and Standard Deviations (SD) for Behaviorally Conditioned (BC), Emotionally Vulnerable (EV), and Biologically Vulnerable (BV) Gamblers, Averaged Across 10 Imputations

	BC (n = 105)		EV (n = 31)		BV (n = 36)	
Variable (Age)	*M*	*SD*	*M*	*SD*	*M*	*SD*
Antisocial/Aggressive behavior (12)	2.02 [c]	2.77	2.42 [c]	2.36	6.91 [a,b]	3.98
Impulsivity (12)	2.07 [c]	2.26	1.81 [c]	1.77	8.25 [a,b]	2.52
Hyperactivity (12)	0.75 [c]	0.99	0.52 [c]	0.76	3.40 [a,b]	1.15
Anxiety (12)	1.33 [b,c]	1.24	3.42 [a,c]	1.48	2.65 [a,b]	1.13
Depression (12)	0.75 [b,c]	1.06	3.29 [a,c]	0.81	1.36 [a,b]	1.08
Drug Problems (14)	20.23 [c]	3.78	19.85 [c]	2.98	23.31 [a,b]	7.69
Gambling Problems (16)	2.63	1.97	3.45	2.25	2.78	2.12
Gambling Frequency (16)	6.21	4.30	7.58	4.36	6.99	4.77
Gambling Problems (23)	1.73 [c]	3.05	1.31	2.19	2.93 [a]	3.35
Gambling Frequency (23)	3.81	3.20	3.96	3.20	3.70	3.09
Age of gambling onset	12.45	3.58	12.32	3.01	12.75	2.95

[a] Different from BC group at the α = .05 level.

[b] Different from EV group at the α = .05 level.

[c] Different from BV group at the α = .05 level.

Article 2. Identifying At-Risk Profiles and Protective Factors for Problem Gambling: A Longitudinal Study Across Adolescence and Early Adulthood

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Identifying At-Risk Profiles and Protective Factors for Problem Gambling: A Longitudinal Study Across Adolescence and Early Adulthood

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Abstract

Past studies identified various risk and protective factors for Problem Gambling (PG). However, no study examined the interplay between these factors using a combination of person-centered and variable-centered approaches embedded within a longitudinal design. The present study aimed to: 1) identify distinct profiles in early adolescence based on a set of risk factors commonly associated with PG (impulsivity, depression, anxiety, drug/alcohol use, aggressiveness, and antisociality), 2) explore the difference in reported gambling problems between these profiles during mid-adolescence and early adulthood, and 3) identify family and peer-related variables that could operate as protective or compensatory factors in this context. Two samples were used: a) a population sample (N = 1,033) living in low socio-economic status neighborhoods, and b) a population sample (N = 3,017) representative of students attending Quebec schools. Latent Profile Analyses were conducted to identify at-risk profiles based on individual risk factors measured at age 12 years. Negative binomial regression models were estimated to compare profiles in terms of their reported gambling problems at ages 16 and 23 years. Finally, family- and peer-related variables measured at age 14 years were included to test their protective or compensatory role with respect to the link between at-risk profiles and gambling problems. Four profiles were identified : Well-Adjusted, Internalizing, Externalizing, and Comorbid. Compared to the Well-Adjusted profile, Externalizing and Comorbid profiles reported more gambling problems at ages 16 and 23 years, but the Internalizing profile did not differ significantly. Various protective and compensatory factors emerged for each profile at both time-points.

Keywords: gambling, latent profile analysis, protective factor, adolescence, longitudinal design.

Introduction

Gambling is a widespread activity; between 50 and 80% of adolescents and adults report at least some gambling over a previous year (Williams, Volberg, & Stevens, 2012). However, only a minority develop gambling problems (i.e., problems directly related to their gambling behavior). The term "problem gambling" (PG) is generally defined as gambling behavior that leads to any number of negative consequences for the gambler, his or her social network, and the community at large (Ferris & Wynne, 2001; Neal, Delfabbro, & O'Neil 2005). Clinical assessment tools, such as the *Diagnostic and Statistical Manual of Mental Disorders: Fifth Edition* (DSM-5; American Psychiatric Association, 2013), use the term "disordered gambling" to describe PG that reaches a pre-set number of legal, social, or self-regulatory problems due to their gambling activities. The prevalence of disordered gambling among the general adult population varies between 2-5%, and between 4-7% for adolescents and young adults, depending on the population studied and the scoring criteria used (Shaffer & Hall, 2001; Williams et al., 2012). Moreover, early-onset gamblers tend to develop more severe gambling-related problems compared to their later-onset counterparts (Burge, Pietrzak, & Petry, 2006; Lynch, Maciejewski, & Potenza, 2004). As such, in order to refine current etiological models of PG and improve prevention programs, there is an urgent need to identify young adolescents who are at risk for gambling problems before they develop.

A variety of PG risk factors first become apparent in childhood and adolescence (Dowling et al., 2017; Gupta & Derevensky, 1998); many of which pertain to individual characteristics. Given their number and their nature, these factors may combine additively or interactively (Milosevic & Ledgerwood, 2010). In either case, variable-centered approaches (i.e., regression based models) are at a disadvantage when accounting for the effects of the numerous interactions which may result. For example, a regression model using six risk factors (i.e., predictors) would yield only six

main effects, but could potentially include 36 two-way interactions alone, not counting for three- and four-way interactions. Given the need to simplify the identification of risk factors and their interactive effects, a person-centred approach such as Latent Profile Analysis (LPA; Collins & Lanza, 2010) should be employed. LPA is a statistical tool that examines participants' scores on a number of continuous indicator variables in order to identify qualitatively distinct participant subgroups (DiStefano, 2012). For example, participants who score highly on variables A and B, but low on variables C and D, are grouped together and separated from participants who display low scores on all variables. Therefore, the first goal of the present study was to use LPA to identify profiles of individuals in the general population that are differently at risk for gambling problems by early adolescence, before the emergence of such problems.

Individual Risk Factors

Many aspects of an individual's personality, whether behavioral, cognitive, or affective, have been linked to gambling problems in adolescence or adulthood. For example, impulsivity, whether based on paper-and-pencil instruments or on laboratory tasks, has been repeatedly linked to PG (Johansson, Grant, Kim, Odlaug, & Gotestam, 2009; Vitaro, Arseneault, & Tremblay, 1999). In these studies, the predictive power of impulsivity remained significant after accounting for influences such as socio-demographic variables, early gambling behavior, and other personality variables (e.g., aggressiveness and anxiety). Other problematic externalizing behaviors can also come into play. For instance, PG has been linked to antisocial (Jacobs, 2000; Stinchfield, 2000; Vitaro, Brendgen, Ladouceur, & Tremblay, 2001) and aggressive behavior (Martins et al., 2013).

Behavioral disorders such as substance use are also repeatedly associated with PG (Maccallum & Blaszczynski, 2002; Petry, Stinson, & Grant, 2005). Notably, substance use and PG appear to have much in common at the physiological and phenotypic level (Wareham & Potenza, 2010).

Moreover, drug and alcohol problems predict an increase in gambling problems over time (Vitaro et al., 2001). It is therefore not surprising that disordered gambling has been reclassified as an addiction, alongside substance abuse, in the DSM-5.

Problem gamblers generally show higher than average levels of internalizing symptoms (e.g., anxiety and depression; Becona, Del Carmen Lorenzo, & Fuentes, 1996; Petry et al., 2005). However, longitudinal studies reveal subtle distinctions. For example, while higher depressive symptom-count predicts increases in gambling problems from mid-adolescence to young adulthood, even when other risk factors are accounted for, this relationship is no longer evident across early- to mid-adolescence (Dussault, Brendgen, Vitaro, Wanner, & Tremblay, 2011).

Whatever their possible additive or interactive combination, the presence of any or all of these risk factors does not guarantee the emergence of gambling problems (Lussier, Derevensky, Gupta, & Vitaro, 2014). This is highlighted by the fact that effect sizes are generally small to moderate (Dowling et al., 2017) and that, when predicting the emergence of PG, the effect of important risk factors such as impulsivity or alcohol consumption may be counteracted or mitigated by social factors (Barnes, Welte, Hoffman, & Dintcheff, 2005; Scholes-Balog, Hemphill, Dowling, & Toumbourou, 2014). This suggests that compensatory or protective factors are most likely at play. The former is identified by an additive (i.e., countering main) effect which decreases the probability of PG emergence (akin to a risk factor, but in the opposite direction), whereas the latter is suggested when an interactive (i.e., a multiplicative/mitigating) effect is present. Either can derive from social agencies such as family or the peer group. Given these possibilities, another goal of the present study was to examine a series of social factors that may compensate or moderate the link between at-risk personality-based profiles and problem gambling.

Compensatory and Protective Factors for Gambling Problems

Social support from parents and friends positively affects multiple physical and mental health outcomes in youth (Abazari, Haghdoost, & Abbaszadeh, 2014; King, Tergerson, & Wilson, 2008). With regards to gambling, support from family, but not from friends, appears to minimize harmful gambling behavior (Rasanen, Lintonen, Tolvanen, & Konu, 2016). In turn, evidence indicates that peer conformity (i..e, peers with normative values and no deviant behaviors) is a consistent compensatory factor against adolescent gambling problems (Lussier et al., 2014; Vitaro et al., 2001). Positive parenting practices hold sway as well. Lee, Stuart, Ialongo, and Martins (2014) demonstrated that higher parental monitoring from 11 to 14 years of age was associated with fewer gambling problems in late-adolescence and young adulthood.

Sociodemographic factors also contribute to the emergence of PG in youths and adults. The incidence of PG is higher among men and ethnic minorities living in underprivileged neighborhoods (Jacobs, 2005). Moreover, Socioeconomic Status (SES) appears to moderate the link between early impulsivity and PG, as impulsivity has been associated with earlier-onset gambling mainly in low-SES participants (Auger, Lo, Cantinotti, & O'Loughlin, 2010). In summation, while the individual risk factors associated with PG are numerous and varied, their effects may fluctuate depending on a host of compensatory or protective factors, as well as participants' age.

Study Objectives

The current study aimed to identify subtypes of young adolescents that are differentially at risk for gambling problems. Subtypes were determined according to individual risk factors most often associated to gambling (i.e., impulsivity, depression, anxiety, substance use, aggressiveness, and antisociality). A second objective was to explore the difference in reported gambling problems

between these profiles by middle adolescence or by early adulthood, as well as the temporal stability between the two time-points. A third and final objective was to explore the compensatory or protective effects of various social factors. The compensatory or protective factors retained for the present study represent two domains: peers and family. For each of these socializing agents, a distinction was made between their behavior (i.e., what parents and peers do that could compensate or protect), and adolescents' connectedness to them (i.e., a form of social support).

Using a longitudinal design, we followed two Quebec (Canada) samples. The first sample ("Sample A") consists of low socioeconomic status (SES) French-speaking adolescent males. The second sample ("Sample B") is representative of all Quebec students attending francophone schools. Personality variables (i.e., impulsive, depressive, anxious, antisocial, and aggressive behaviors) were reported by teachers when participants were 12 years old, an age where most have not yet gambled for the first time (Traoré et al., 2015). Teacher ratings were chosen over parent ratings because the former better predict problem behavior in children (Power et al. 1998; Verhulst et al. 1994). Substance use was assessed using a self-report measure at age 14. Self-reports were chosen because young adolescents are likely to hide their substance use from their teachers, thereby resulting in an underestimation from the teacher's perspective. Moreover, surveys in Quebec have shown that most adolescents start using drugs or alcohol around the age of 14 years (Dubé et al. 2009). Putative compensatory/protectivefactors (parental involvement, parent-child connectedness, peer conformity, and peer connectedness) were assessed through self-reports at age 14 years. Gambling problems were measured in both samples when participants were 16 and 23 years old. Given that both samples were merged, we also examined whether results varied from one sample to another.

Material and Methods

Participants

Sample A. Participants were part of a longitudinal study started in 1984 with 1,033 French-speaking kindergarten boys attending schools in economically disadvantaged areas in Montreal, Quebec, Canada. Participants included in the initial sample represented 87% of all boys attending the 53 schools that participated in the study. Data used in this study were collected when the participants were 12, 14, 16 and 23 years old.

Sample B. Participants were part of the Quebec Longitudinal Study of Kindergarten Children, a representative, random sample of the French-speaking children attending kindergarten in the province of Quebec, Canada, in 1986–1987. From an initial pool of 6,397 families with six-year old children who were contacted, 4,360 (68%) agreed to participate. Of these, 2,000 participants were randomly selected in order to ensure good participation during follow-up and due to budget limitations. An additional 1,142 participants from the initial pool who had elevated scores on a disruptive behavior scale (Tremblay et al., 1991), were included to provide statistical power in studies dealing with problematic behavior. The final sample consisted of 3,142 participants (58% males). See Rouquette et al. (2014) for a complete explanation of sampling procedure. Data used in this study were collected when the participants were 12, 14, 16 and 23 years old.

Procedure

The study was approved by the University of Montreal Institutional Review Board and by the respective school boards. Active written consent was obtained from parents in both samples. All participants actively provided their assent to take part in the study. Participants were also informed before data collection that their answers were strictly confidential and that they could end their

participation at any time. All measures were administered in the Spring towards the end of the school year. The same instruments were administered in both samples. All instruments were administered in French. Instruments that were written originally in English were translated into French and then translated back into English. English-speaking judges verified the semantic similarity between the back-translated items and the original items in the questionnaire. Descriptive statistics (i.e., correlations, means, and standard deviations) are reported in Table 1.

Measures: At-risk Personal Characteristics Used for LPA

With the exception of drug/alcohol use that was self-reported when participants were 14 years old, all measures used to assess personal characteristics were teacher-rated using the Social Behavior Questionnaire (SBQ; Tremblay, Desmarais-Gervais, Gagnon, & Charlebois, 1987) when participants were 12 years old. From the SBQ, items were selected to assess impulsivity, depression, anxiety, aggressiveness, and antisociality. Each item was rated on a 3-point scale (0: Does not apply, 1: Applies sometimes, and 2: Frequently applies). Item scores were summed to obtain a total score for each variable. Therefore, higher scores indicate higher levels on each scale.

Impulsivity. A total of seven items (e.g. "acts without thinking") were included in this scale (Sample A: Cronbach's α = .89; Sample B: α = .88). Total scores varied between 0 and 13. ***Depression***. A total of four items (e.g. "appears miserable, unhappy, tearful or distressed") were included in this scale (Sample A: α = .62; Sample B: α = .63). Total scores varied between 0 and 7. ***Anxiety***. A total of three items (e.g. "is worried about many things") were included in this scale (Sample A: mean r = .43; Sample B: mean r = .44; for very short scales, Clark & Watson, 1995, recommend using the mean of inter-item correlation instead of Cronbach's alpha). Total scores varied between 0 and 6. ***Antisociality***. A total of three items (e.g. "has stolen things on one or more occasions.") were included in this scale (Sample A: mean r = .25; Sample B: mean r = .25). Total

scores varied between 0 and 6. ***Aggressiveness.*** A total of six items (e.g. "uses physical force, or threatens to use force, in order to dominate other children.") were included in this scale (Sample A: α = .89; Sample B: α = .88). Total scores varied between 0 and 12. ***Drug Consumption at Age 14 Years.*** The Personal Experience Screen Questionnaire (PESQ; Henly & Winters, 1988) was used to gauge illicit drug and alcohol consumption. The frequency scale was derived by asking the following question: "During the last 12 months, how many times have you consumed (blank)?". The blank was replaced by "marijuana or haschich", "alcohol", "cigarettes", and "other drugs", resulting in a total of 4 questions, which were then averaged into a composite frequency score. Answers were reported on a 7-point Likert scale, ranging from "never" to "40+ times". Average scores varied between 1 and 7 (Sample A: α = .69; Sample B: α = .68). The following three items were included to detect if participants were lying: "In order to pay for alcohol or drugs, how many times have you conned people with counterfeit money?", "How many times have you consumed alcohol or other drugs (including cigarettes and marijuana) while parachuting?", and "How many times have you obtained alcohol or drugs from a police officer?" If a participant responded positively to one of these questions, the overall score was deleted and treated as missing data.

Measures: Compensatory and Protective Factors

All instruments used to assess compensatory or protective factors were self-reported when participants were 14 years old.

Parental Involvement. Two items ("Do your parents know who you are with when you are out of the house?", "Do your parents know where you are when you are out of the house?") were answered on a scale from 1 ("never") to 4 ("always"). Total scores varied between 2 and 8. Correlation between items for Samples A and B were .69 and .59, respectively. ***Parent-Child Connectedness.*** A total of seven items (e.g., "Do you talk with your parents about your thoughts

and about your feelings?") were included in this scale (Sample A: α = .74; Sample B: α = .78). Items were rated from 1 ("always") to 4 ("never"). Total scores varied between 7 and 28. To facilitate interpretation of analysis results, final scores were reverse coded such that higher scores to indicate high parent-child connectedness. ***Peer Conformity*.** Three items (e.g., "Has the boy/girl who is your best friend already been arrested by the police?", reverse-coded) were used to create a Peer Conformity scale. Items were scored 1 ("no") or 2 ("yes"), yielding total scores between 3 and 6. Mean inter-item correlations were .45 and .38 for Samples A and B, respectively. ***Peer Connectedness*.** A total of 5 items (e.g., "Do you talk about personal things with the girl/boy who is your best friend?") were included in this scale (Sample A: α = .65; Sample B: α = .60). Items were rated from 1 ("often") to 4 ("never"). Total scores varied between 5 and 20. To facilitate interpretation of analysis results, final scores were reverse coded such that higher scores to indicate high peer connectedness.

Outcome Variables: Gambling Problems at Ages 16 and 23 Years. Adolescent and adult versions of the South Oaks Gambling Screen (SOGS) were used to assess gambling problems (Lesieur & Blume, 1987; Winters, Stinchfield, & Fulkerson, 1993). The SOGS is a self-reported questionnaire designed to assess problems related to past-year gambling practices. The SOGS-Revised for Adolescents (SOGS-RA) comprises 12 Yes/No items, whereas the adult version consists of 20 items (both versions share 11 items). The adolescent questionnaire only has one Yes/No question indicating whether the participant has borrowed money for gambling or to pay gambling debts. In contrast, the adult questionnaire splits the "borrowing money to gamble or to pay gambling debts" indicator into nine items listing various money loaning sources (e.g., credit cards, loan sharks, etc.). For the purpose of the current study, the nine items in the adult version were collapsed into one Yes/No item indicating whether the participant has borrowed money to

gamble. This resulted in a consistent set of items for both time-points and allowed a longitudinal (i.e., repeated-measures) comparison of gambling problems between adolescence and young-adulthood.

The SOGS-RA has been validated for screening gambling problems among adolescents aged 14 to 20 years old (Chiesi, Donati, Galli, & Primi, 2013), and the SOGS has been validated in a variety of settings (i.e., Gamblers Anonymous, university students, and hospital employees; Lesieur & Blume, 1987). In Sample A, Cronbach's α was .76 and .88 at ages 16 and 23, respectively. In Sample B, Cronbach's α was .76 and .75 at ages 16 and 23, respectively.

Control Variable: Socioeconomic Status. When the participants were 12 years old, parents answered Blishen, Carroll, and Moore (1987) Occupational Prestige Scale. This scale, specific to Canada, classifies occupations according to income and education. A composite score was created by summing up both parents' scores.

Data Analysis Plan

Latent Profile Analysis (LPA; Collins & Lanza, 2010) was used for the first set of analyses. LPA is a type of Latent Class Analysis, which allows identification of profiles according to a set of indicator variables (DiStefano, 2012). The number of profiles is not known beforehand and the optimal solution is found by using a set of model fit indicators, combined with maximum-likelihood tests (probability-based classification). Following Celeux and Soromenho (1996) and Nylund, Asparouhov, and Muthén (2007), the *Bayesian information criterion (BIC)*, *Entropy*, and *Lo-Mendell-Rubin Adjusted Likelihood Ratio Test (LMR-ALRT)* were used to evaluate model fit. LPA was performed using a step-wise mixture modeling technique with MPlus 6.12 (Muthén & Muthén, 2010) to identify the optimal number of PG profiles based on the following indicator

variables: impulsivity, depression, anxiety, antisociality, and aggressiveness at age 12 as well as drug/alcohol use at age 14 years.

All the analyses predicting gambling problems at ages 16 and 23 years statistically controlled for Gender, SES, and Sample and were also conducted with MPlus 6.12 (Muthén & Muthén, 2010). Analyses predicting gambling problems at age 23 years also controlled for gambling problems reported at age 16 years. Participants were classified into one of the four profiles identified by the LPA, based on their most likely class membership. The Well-Adjusted profile was set as the reference group, because it comprised the vast majority of participants and was considered a low risk group. For each time point (i.e., age 16 and age 23 years), a hierarchical two-step analysis was conducted. First, compensatory (i.e. main) effects were examined by using the profiles (dummy-coded) to predict gambling problems. Second, protective (i.e., interactive) effects were examined by computing interaction terms between each putative compensatory/protective factor and the dummy-coded variable for each profile. Considering that SOGS and SOGS-RA data are count data (i.e., number of problems), analyses of compensatory and protective factors were conducted by estimating regression models using a Negative Binomial distribution. A Negative Binomial regression model was preferred over a Poisson model because SOGS and SOGS-RA data were both over-dispersed (i.e., variance 3 to 4 times greater than the mean, for both time points) (Gardner, Mulvey, & Shaw, 1995).

Finally, a linear growth model for a count outcome using a negative binomial model was estimated to examine changes in gambling problems over time. Intercept and slope (i.e., rate of change) were estimated by fitting our data using two time-points. The slope was then regressed, by controlling for the intercept, on dummy-coded variables indicating each at-risk profile (once again, with the Well-Adjusted profile serving as a reference group).

Results

Data Treatment and Preliminary Analyses

Missing Data. Both samples had missing data. Little's (1988) Missing Completely At Random (MCAR) test was conducted using all variables included in the LPA (listed in the next section) and was not significant: χ^2 (40, N = 4,175) = 39.03, p = .514), supporting the hypothesis that data are MCAR. Out of the 4,175 initial participants, 555 participants had missing data on all indicator variables and were therefore excluded from the LPA, resulting in 3,620 classified participants. Participants with partially missing data were included in the LPA and all subsequent analyses (i.e., negative binomial regression estimations and linear grwoth model), because they use a Full Information Maximum Likelihood estimation method, thus overcoming any limitations introduced by missing data (as long as data is not Missing Not At Random).

Sample A. After removing participants with missing data, 939 participants remained out of the initial 1,033. When participants were 16 years old, 5% reported 3 or more gambling problems, and 1.4% reported 5 or more gambling problems. At age 23 years, 6.7% had 3 or more gambling problems, and 3.3% had 5 or more gambling problems. These rates are higher than what is usually found in population studies, but are explained by the high-risk nature of this sample (i.e., low SES males).

Sample B. After removing participants with missing data, 2,681 participants remained out of the initial 3,142. When participants were 16 years old, 3.2% reported 3 or more gambling problems, and 1.1% reported 5 or more gambling problems. At age 23 years, 1.8% had 3 or more gambling problems, and 0.5% had 5 or more gambling problems. These proportions are consistent with population studies conducted in various regions across the world (Williams et al., 2012).

Latent Profile Analysis

Model fit indices are reported in Table 2. Two- and three-class models separated groups based on their degree of severity across all variables (i.e., high on all vs. low on all). Furthermore, although the five- and six-class models had lower BIC scores, this indicator kept decreasing no matter how many classes were added. The tendency for the BIC to consistently decrease has been observed across many other studies that have used continuous, rather than categorical, indicators to determine the optimal number of classes (Geiser, Okun, & Grano, 2014; Marsh, Lüdtke, Trautwein, & Morin, 2009; Nower, Martins, Lin, & Blanco, 2013). The five-class model also had a profile with less than 5% participants. As such, and in accordance with Marsh et al.'s (2009) recommendations, the best-fitting solution was selected based on qualitative and quantitative changes between the classes. A four-class model was the most optimal, as demonstrated by the statistically significant *LMR-ALRT* and profiles that were qualitatively meaningful (i.e., not simply separated into high, medium, and low levels across all variables).

The most common profile (n = 2,498; 69.01%; "Well-Adjusted") displayed low levels on all indicator variables. The second-largest profile (n = 576; 15.91%; "Externalizing") had high levels of impulsivity, aggressiveness, antisocial behavior, and substance use. The "Internalizing" profile (n = 331; 9.14%) scored highly on anxiety and depression symptoms, and low on everything else. Finally, the "Comorbid" profile (n = 215; 5.94%) scored highly on all variables. Table 3 summarizes each profile's scores on all indicator variables. Notably, similar profiles were found in both samples, albeit in different proportions.

To confirm differences between profiles on indicator variables, a multivariate Analysis of Variance (ANOVA) was conducted, with profiles serving as a fixed factor. The multivariate result was significant, indicating a difference between groups across all indicator variables: Pillai's Trace

= 1.34, F (18, 5,835) = 262.15, $p < .001$. All univariate F tests for between-subjects effects were significant (see Table 3). A chi-square test of independence was conducted next to test gender distribution across profiles. The test was statistically significant, χ^2 (3, N = 3,620) = 231.47, $p < .001$. Specifically, women were more likely to have a Well-Adjusted profile, and were underrepresented in the Externalizing and Comorbid profiles. An ANOVA was conducted to examine SES differences between profiles. There was a significant effect of profiles on SES, F(3, 1978) = 12.61, $p < .001$. SES was higher for Well-Adjusted participants, compared to the other three profiles. Finally, a chi-square test of independence was conducted to test whether profile distribution was different across samples. The test was also statistically significant, χ^2 (3, N = 3,620) = 129.38, $p < .001$. Well-Adjusted participants were more likely to come from Sample B, whereas Internalizing, Externalizing and Comorbid profiles were more likely to come from Sample A. To account for these significant effects, subsequent analyses included Gender and Sample as covariates, in addition to parents' occupational prestige scores.

Comparison of Gambling Problems Between Profiles

As shown in Table 4, significant differences in gambling problems emerged between profiles at age 16 years. More specifically, Externalizing ($M = 0.52$; $SD = 1.39$; $\beta = 0.56$; $p < .001$) and Comorbid ($M = 0.60$; $SD = 1.30$; $\beta = 0.70$; $p = .001$) profiles both reported more gambling problems at age 16 years compared to the Well-Adjusted profile ($M = 0.25$; $SD = 0.85$), but the Internalizing ($M = 0.30$; $SD = 0.95$; $\beta = 0.16$; $p = .486$) profile did not. Gender and SES were significant covariates, but Sample was not, indicating that males and low-SES participants reported more gambling problems at age 16.

At age 23, Gender and Sample were significant predictors, indicating that males and participants from Sample A reported more gambling problems. Regarding at-risk profiles, Externalizing ($M =$

0.58; *SD* = 1.46; β = 0.45; *p* = .026) and Comorbid (*M* = 0.99; *SD* = 2.32; β = 0.81; *p* = .004) profiles both reported more gambling problems at age 23 years compared to the Well-Adjusted profile (*M* = 0.24; *SD* = 0.92), but the Internalizing (*M* = 0.31; *SD* = 0.95; β = 0.04; *p* = .904) profile did not.

When examining changes over time in terms of gambling problems (from ages 16 to 23 years) the mean slope across all participants was not significantly different from zero (β = -0.10; *p* = .764), indicating a lack of evidence for an increase or decrease in gambling problems over time. However, when including participants' profiles as predictor variables (with the Well-Adjusted profile serving as a reference point), results indicated that Externalizing (β = 0.47; *p* = .018) and Comorbid profiles (β = 0.83; *p* = .005) both reported an increase in gambling problems from ages 16 to 23 years. The predictive effect of the Internalizing profile on the slope of gambling problems was not significant (β = 0.06; *p* = .842).

Compensatory and Protective Factors

The next section examines the social factors related to the family and peers that could operate as compensatory or protective factors with respect to the link between at-risk profiles and gambling problems either at age 16 or at age 23 years. Regression coefficients for main (i.e., compensatory) and interaction (i.e., protective) effects for both time-points are reported in Table 5. It is important to note that all the putative compensatory or protective factors were badly correlated with the profiles (i.e., Spearman ρ ranging from .06 to .11).

Parental Involvement. At age 16 years, higher parental involvement significantly predicted fewer gambling problems for all participants. No interaction effects emerged, indicating that all the profiles equally benefitted from parent involvement. At age 23 years, again, higher parental involvement significantly predicted fewer gambling problems for all participants. However, when

participant profiles were included in the analysis, only Internalizing participants reported fewer gambling problems when parental involvement was high, indicating that the compensatory effect observed previously might be unique to this profile (and possibly the Comorbid profile, which was close to significance at $p = .056$)..

Parent-Child Connectedness. At age 16 years, higher parent-child connectedness significantly predicted fewer gambling problems for all participants. No interaction effects emerged, indicating that all the profiles equally benefitted from parent involvement. At age 23 years, there was no main and no interaction effect of parent-child connectedness on gambling problems.

Peer Conformity. At age 16 years, participants with higher levels of peer conformity reported fewer gambling problems. No interaction effect was found. Similar results emerged at age 23 years.

Peer Connectedness. At age 16 years, there were no main and no interaction effects of peer connectedness on gambling problems. At age 23 years, higher peer connectedness significantly predicted fewer gambling problems for all participants. No interaction effects emerged, indicating that all the profiles equally benefitted from parent involvement.

Discussion

The present study aimed (a) to identify distinct profiles of individuals at-risk of developing gambling problems on the basis of individual risk factors measured during early adolescence and (b) to explore whether certain social factors linked to family and peers could exert a compensatory or a protective role. The findings support the notion that some individuals are more at risk of developing gambling problems by virtue of their behavioral profile (and more so over time) compared to individuals with a different behavioral profile. At the same time, not all individuals sharing the same at-risk behavioral profile develop gambling problems, suggesting

protective/compensatory factors. The impact of these protective/compensatory factors, however, seems to vary depending on participants' profile and age.

At-Risk Profiles

Regarding the first and second study objectives, we identified four distinct profiles based on the behavioral patterns of early adolescents. Three represented "at risk" profiles: (1) an Internalizing profile (i.e., participants with high scores on anxiety and depression scales), (2) an Externalizing profile (i.e., participants with high scores on antisociality, aggressiveness, and impulsivity scales, and medium scores on drug/alcohol consumption scales), and (3) a Comorbid profile (i.e., participants with high levels on all scales). The fourth was a Well-Adjusted profile, characterized by its low scores on all individual personality risk factors (although drug use scores were higher than the Internalizing profile). Compared to the Well-Adjusted profile, the Externalizing and the Comorbid profiles manifested more gambling problems when participants were 16 and 23 years old. These two profiles were also the only ones to report an increase in gambling problems between both time-points.. These results deserve a number of comments.

First, the prospective links between risk profiles based on individual factors and gambling problems found in the current study are consistent with Dowling et al.'s (2017) systematic review of early risk factors. Particularly, profiles reporting high levels of impulsivity, substance use, and antisociality (i.e., Externalizing and Comorbid profiles) reported the most gambling problems at ages 16 and 23. Current results are also consistent with certain etiological models (e.g., Multiple Pathways Model to Problem and Pathological Gambling; Blaszczynski & Nower, 2002), which claim that gamblers with impulsive and antisocial traits display the most severe levels of gambling problems.

Second, it is important to note that the Well-Adjusted profile still reported some gambling problems in adolescence and young adulthood, albeit to a smaller extent than the other three profiles. Our findings are based on two samples: one was a high-risk sample, whereas the other was derived from an oversampling of high-risk individuals. As such, the descriptive term of "Well-Adjusted" used to identify their constituent participants refers to their initial profile configuration relative to the other profiles. These individuals were not totally exempt of any personal or environmental risk factors. Indeed, youths with this profile reported more substance use when compared to those with the Internalizing profile. Considering the significant links between substance use and gambling problems (Dowling et al., 2017; Vitaro et al., 2001), it is possible that Well-Adjusted participants' gambling problems emerged as a corollary of their drug/alcohol consumption habits, which in turn flow from the high-risk nature of our combined sample. Thus, whatever the reason for Well-Adjusted participants' gambling problems, findings from the at-risk profile comparisons reported in the current study are conservative.

Third, the present results indicate that young adolescents with only internalizing problems are not necessarily more at risk for later gambling problems than well-adjusted individuals. This may suggest that internalizing symptoms alone, even though generally linked to PG, may exert a weaker effect than externalizing symptoms on the emergence of PG. Considering that past studies have found clear links between internalizing symptoms and gambling problems, a few explanations may be offered to account for this unexpected finding (other than the high-risk nature of our Well-Adjusted profile and therefore the conservative results). One possibility may be that the links identified in the literature are accounted for by subgroups of gamblers analogous to our Comorbid profile (who reported high levels of internalizing symtpoms and gambling problems). The age at which internalizing symptoms were assessed might also play a role. For example, Walther,

Morgenstern, & Hanewinkel (2012), who have also used a sample of 12 years old participants, found no association between depression or social anxiety and PG, whereas those who did used older participants (e.g., Dussault et al., 2011).

Finally, stability of gambling problems depended on profile membership. Externalizing and Comorbid participants were the only profiles to report an increase in gambling problems. Both profiles reported high levels of impulsive, antisocial, and aggressive behaviors. Hence, these types of behaviors may be particularly responsible for an increase in gambling problems, possibly by motivating individuals to engage in delinquent activities to fund gambling behavior and debts.

Protective and Compensatory Factors for PG

In line with the third study objective, social compensatory and protective factors relevant to the risk of PG emerged. Once more, a developmental dimension seems to be involved as the role and the nature of the compensatory and protective factors differed depending on whether gambling problems were assessed during adolescence (i.e., age 16) or during early adulthood (i.e., age 23). Finally, the role of some compensatory or protective factors also varied depending on participants' profile.

Regarding parental influences, parent-child connectedness served as a compensatory factor against gambling problems at age 16, but not at age 23. Results at age 16 years are consistent with previous studies (e.g., Rasanen et al., 2016), but the literature is lacking information regarding the link between parent-child connectedness assessed in adolescence and PG in young adulthood. An explanation for the present results may be that, as offspring transition into adulthood, they tend to spend less time with their parents and become closer to their peer group and romantic partner, thus leading parents to play a smaller role in their lives.

Yet, parent involvement emerged as a compensatory factor both at age 16 and at age 23, suggesting that parents' awareness of their children's activities may have an enduring sheltering effect against developing PG. However, when participants' at-risk profile was accounted for, only Internalizing participants benefited from parent involvement at age 23 years (although the effect for Comorbid participants was close to statistical significance), suggesting that participants with internalizing symptoms (e.g., anxiety and depression) are most sensitive to the enduring beneficial effect of parent involvement on PG. This is in stark contrast to findings by Scholes-Balog, Hemphill, Toumbourou, & Dowling (2015), which showed no link between various indicators of parent-child relationships (family concord, attachment, interaction, and prosocial behavior) and PG and no interaction between Internalizing symptoms and parent-child indicators when predicting PG. However, it should be noted that, unlike the present study, all potential protective factors were assessed when participants were between 17 and 24 years of age. Also, and perhaps more importantly, PG was dichotomized based on whether participants answered positively to at least one of two screening questions. Therefore, findings from the Scholes-Balog et al. (2015) study must be interpreted with great caution.

With regards to peer influences, we found little evidence for a compensatory or interactive effect of peer connectedness (i.e., a form of social support) on the risk of PG at age 16, which is consistent with previous findings (Rasanen et al., 2016). Studies that have found a positive association between peer connectedness and PG in adolescence have assessed both variables concurrently (Rasanen et al., 2016). As adolescents grow older, peers become their main gambling partners (Gupta & Derevensky, 1998). Peer connectedness, in this case, may give adolescents some access to gambling partners in an unsupervised setting (compared to gambling with parents). Thus, peer connectedness may have an effect on PG, but that effect likely depends on peer gambling

behavior. In the present study, most participants likely had nongambling peers, which may explain the lack of association between peer connectedness and PG at age 16. However, unexpectedly, the present study has uncovered a delayed compensatory effect from peer-connectedness in early adolescence on PG at age 23 years. Developing close emotional bonds with peers during adolescence may have allowed individuals to develop adequate social skills to properly fit into society once they reach adulthood. Finally, all participants who reported having conformist peers also reported fewer gambling problems at ages 16 and 23. This supports findings generally found in the literature regarding peer conformity (Lussier et al., 2014).

Strength, Limitations and Conclusions

Several strengths of this study should be noted. This is the first prospective cohort study to a) identify profiles of adolescents among the general population at risk for gambling problems and b) examine compensatory and protective factors for gambling problems among these profiles. In comparison to cross-sectional studies, the results of our longitudinal analyses help reinforce the directionality (albeit not the causality) from risk or compensatory factors to gambling problems. Next, the use of various sources of information (i.e., teachers and participants) minimizes the risk of shared-method variance in our data. Finally, at-risk profiles were determined when participants were 12 years old, before gambling problems generally appear. It is therefore highly unlikely that profile characteristics were affected by gambling problems.

On the other hand, some limitations remain. First, a number of variables assessed in this study were derived from scales that consisted of only two or three items. Second, the model fit indicators used to determine the optimal LPA model did not consistently converge toward the same number of profiles; a qualitative analysis of results was needed to address this issue. Third, it would have been useful to gather additional variables about peers and family, especially in terms of specific

gambling practices, in order to determine participants' specific sources of gambling exposure, and explore any moderating effects with regards to peer and family connectedness. Finally, although the missing data analysis allowed us to safely remove a small portion of our participants, it should be noted that it is always preferable to have complete data. Despite these limitations, the longitudinal and profiling analyses conducted in the current study provide additional and innovative insights into the current state of knowledge of individuals at risk for developing gambling problems and about compensatory/protective factors for PG, thus allowing for a refining of etiological models and prevention efforts. Finally, the current study showed that, depending on when the problematic behavior is measured, differences between profiles may vary and the same compensatory/protective factors may have different effects. Together, these findings call for careful analyses when deducing any given variable's effect on a problem gambling.

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Tables

Table 1

Correlations (with Pairwise Deletion; N Between 1129 and 3338) and Descriptive Statistics for all Variables

Variable (age in years)	1	2	3	4	5	6	7	8	9	10	11	12
1. Impulsivity (12)	1											
2. Anxiety (12)	.42**	1										
3. Depression (12)	.26**	.58**	1									
4. Antisociality (12)	.52**	.30**	.25**	1								
5. Aggression (12)	.69**	.29**	.24**	.60**	1							
6. Drug use (14)	.07**	-.03	-.06**	.09**	.10**	1						
7. Parental Monitoring (14)	-.10**	-.03	-.01	-.10**	-.12**	-.29**	1					
8. Peer Bonding (14)	-.06*	-.10**	-.10**	-.04	-.05*	.20**	.02	1				
9. Parent-child Connectedness (14)	-.05*	-.03	-.04	-.05*	-.06**	-.18**	.39**	.12**	1			
10. Peer Conformity (14)	-.17**	-.06**	-.01	-.19**	-.22**	-.44**	.31**	-.06**	.16**	1		
11. Gambling Problems (16)	.11**	.08**	.07**	.08**	.14**	.09**	-.12**	-.03	-.08**	-.13**	1	
12. Gambling Problems (23)	.16**	.07**	.09**	.18**	.16**	.10**	-.08**	-.12**	-.02	-.16**	.22**	1

Table 1

Continued

Variable	1	2	3	4	5	6	7	8	9	10	11	12
M	2.39	1.74	1.09	0.40	1.66	1.72	6.34	12.99	17.54	5.61	0.31	0.33
SD	3.25	1.56	1.44	0.75	2.62	1.09	1.43	2.88	4.24	0.69	0.97	1.16

* $p < .05$. ** $p < .01$

Table 2

Model Fit Indicators for Latent Profile Analysis

Model	BIC[a]	Entropy	LMR-ALRT[b] (*p* value)
2-class	66530	.890	.000
3-class	64945	.902	.000
4-class	**63915**	**.868**	**.000**
5-class	60520	.934	.000
6-class	59548	.919	.698

Note. Best-fitting model is shown in bold.

[a] Bayesian Information Criterion

[b] Lo-Mendell-Rubin Adjusted Likelihood Ratio Test

Table 3

Mean Scores for Latent Profile Analysis Indicator Variables, According to Profile

Variables	Well-Adjusted	Internalizing	Externalizing	Comorbid	*F*
Anxiety	0.94*	2.31*	5.68*	8.62*	413.48
Depression	1.11*	3.90*	2.49*	2.81*	493.05
Impulsivity	0.54*	3.57*	1.39*	2.22*	842.37
Aggressiveness	0.12*	0.41*	0.90*	1.87*	3710.82
Antisocial behavior	0.38*	0.78*	4.38*	8.96*	453.55
Drug/Alcohol use	1.70	1.48*	1.83	2.01*	8.28

* Statistically different from all other profiles ($p < .05$)

Table 4

Negative Binomial Regression of Profiles Predicting Gambling Problems (GP) at Age 16 and 23 years

	Age 16		*Age 23*	
Variable	*β*	*p*	*β*	*p*
Sample (0 = A; 1 = B)	0.21	.156	-0.45	.017
SES	-0.01	.000	0.00	.965
Gender (0 = M; 1 = F)	-1.09	.000	-1.04	.000
Internalizing	0.16	.486	0.04	.904
Externalizing	0.56	.001	0.45	.026
Comorbid	0.70	.002	0.81	.004
Well-Adjusted	1		1	
GP at age 16 years	(not applicable)		0.34	.000

Table 5

Main (i.e., Compensatory) and Interaction (i.e., Moderating) Effects of Social Factors on Gambling Problems at Age 23 Years

Age	Variable	Main effect	Interaction effect			
		β	Well-Adjusted (Reference group)	Internalizing	Externalizing	Comorbid
16 years	Parental Involvement	**-0.20****	**-0.20****	0.08	0.05	0.04
	Parent-Child Connectedness	**-0.06****	**-0.08****	0.04	0.03	0.04
	Peer Conformity	**-0.41****	**-0.38****	-0.03	0.05	-0.03
	Peer Connectedness	0.02	0.00	0.00	0.04	0.07
23 years	Parental Involvement	**-0.15***	-0.09	**-0.17****	-0.06	-0.16
	Parent-Child Connectedness	-0.03	-0.01	-0.04	-0.02	-0.05
	Peer Conformity	**-0.36****	**-0.30****	-0.16	-0.01	-0.03
	Peer Connectedness	**-0.08****	**-0.07****	-0.07	-0.02	-0.02

Note: Significant effects shown in bold.

*$p < .05$; **$p < .01$

Article 3. Pathways Model to Problem Gambling: Clinical Implications for Treatment and Prevention Among Adolescents

Allami, Y., & Vitaro, F. (2015). Pathways model to Problem Gambling: Clinical Implications for Treatment and Prevention Among Adolescents. *Canadian Journal of Addiction*, *6*(2), 13-19.

Pathways model to problem gambling and its clinical implications for prevention

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Word count: 3,941

Abstract (EN)

The Pathways Model is the most promising etiological model of problem gambling. It suggests the existence of three developmental pathways to problem gambling, each differentiated by a set of predisposing biopsychosocial characteristics. In this paper, we provide empirical evidence in support of this etiological model in relation to youth gambling problems. We also propose a differential approach to prevention that is tailored to young gamblers' profile.

Abstract word count: 66

Abstract (FR)

Le modèle des parcours multiples est le plus prometteur des modèles étiologiques pour les problèmes de jeux de hasard et d'argent (JHA). Il met de l'avant trois parcours développementaux, chacun défini par un ensemble de prédispositions biopsychosociales, menant à l'émergence de problèmes de JHA. Dans cet article, nous fournissons des données probantes soutenant ce modèle étiologique en lien avec les problèmes de JHA chez les adolescents. Nous proposons aussi une approche de prévention différenciée adaptée au profil d'appartenance de jeunes joueurs.

Abstract word count : 81

Keywords: adolescent, gambling, prevention, pathways model

Introduction

Definition and Prevalence

According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013), a Gambling Disorder (GD) is evident when there is a presence of at least four gambling-related problems. Other instruments, such as the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987; Winters, Stinchfield, & Fulkerson, 1993), use different criteria and identify different types of problem gamblers (e.g., score 1-4: Some problems with Gambling; score 5 or more: Probable Pathological Gambler). Regardless of whether one uses a dimensional or a categorical approach to understanding problem gambling, there is a general agreement that a number of individuals experience several forms of gambling-related issues. The prevalence of GD among the general adult population in Canada, when assessed during the previous year, is around 2-3% (Cox, Yu, Afifi, & Ladouceur, 2005; Williams, Volberg, & Stevens, 2012).

Adolescents (12 to 17 years of age) present higher rates of problem-gambling compared to their adult counter-parts (Jacobs, 2005; Shaffer & Hall, 2001). Although some authors suggest that prevalence estimates among adolescents may be overestimated (Derevensky, Gupta, & Winters, 2003), most adults suffering from a gambling disorder assert having started gambling before or during their adolescent years (Gupta & Pinzon, 2012). Although not all adolescent gamblers experience gambling related problems (e.g. stealing money to fund gambling activities), a significant portion may be at-risk due to their level of involvement in gambling activities. In fact, Gupta and Derevensky (1998) reported that 28,2% of high-school students claimed to gamble at least once a week. Furthermore, there is empirical evidence to support that gambling frequency within the adolescent population is a predictor for later gambling problems, independent of current

problems (Vitaro, Brendgen, Ladouceur, & Tremblay, 2001). It is therefore crucial to establish a paradigm which identifies problem or at-risk gamblers within the adolescent population in order to better direct prevention efforts and avoid the negative personal and social consequences that can follow.

Subtyping of problem-gamblers

In order to effectively prevent problems that might arise from or precede disordered gambling, it is important to understand the factors which might have led to the emergence of this disorder. Moran (1970) was the first to suggest different causes for the emergence of problem gambling and identified five types of gamblers: subcultural, neurotic, impulsive, psychopathic, and symptomatic. According to the author, these types may not be mutually exclusive. However, there was very little empirical evidence to support Moran's theory at the time. Since then, numerous studies have supported the idea that there are different profiles of gamblers, each with a defining set of personal and environmental characteristics (Gonzalez-Ibanez et al., 2003; Gupta et al., 2012; Ledgerwood & Petry, 2010; Nower, Martins, Lin, & Blanco, 2013).

Three theoretical models stand out with respect to the notion of different profiles of problem gamblers: The General Theory of Addictions, founded by Jacobs (1986); the Biopsychosocial Perspective of Problem Gambling formulated by Sharpe (2002); and the Pathways Model of Problem and Pathological Gambling theorized by Blaszczynski and Nower (2002).

The Pathways Model holds significant advantages over the other two models. First, neither Jacobs nor Sharpe include the notion of an antisocial personality, despite the fact that many studies have uncovered the existence of this distinct subgroup (Milosevic & Ledgerwood, 2010; Nower et al., 2013). Also, this subgroup tends to exhibit remarkably severe levels of gambling problems.

Secondly, Milosevic and Ledgerwood (2010) reviewed 17 empirical studies that classified problem-gamblers according to various biopsychosocial characteristics and they concluded that a 3-group model consistently emerged. This 3-group model closely resembled the three pathways described by Blaszczynski and Nower (2002). Particularly, a subgroup of gamblers with relatively low impulsivity levels was identified (Milosevic & Ledgerwood, 2010). Although all three models recognize impulsivity as a major contributor to problem gambling, only the Pathways Model splits problem gamblers into non-impulsive and impulsive subgroups. Furthermore, the existence of a distinct subgroup for impulsive gamblers could partly explain why there are conflicting results in the literature regarding the relation between impulsivity and gambling problems (Dannon, Shoenfeld, Rosenberg, Kertzman, & Kotler, 2010).

Finally, although the General Theory of Addictions and the Biopsychosocial Perspective of Problem Gambling are both capable of offering reasonable explanations for GD development and have proven useful in orienting treatment practices, they are limited in their ability to identify opportunities for prevention. An effective prevention model must be able to target risk factors before a clinical disorder develops; a condition that is only fulfilled by the Pathways Model by virtue of its unique developmental perspective. Therefore, we believe the approach presented in the Pathways Model to be the most comprehensive.

Pathways Model of Problem and Pathological Gambling

The Pathways Model (Blaszczynski & Nower, 2002; Nower & Blaszczynski, 2005) puts forward three developmental profiles to problem gambling, each of which is characterized by a set of predisposing risk factors and consequences that result from gambling. The first profile (Pathway #1) relates to “Behaviorally Conditioned” gamblers who do not exhibit any biological or affective predisposition. These individuals start to gamble for reasons connected to excitement and

socialization, and fluctuate between regular and excessive gambling depending on effects of conditioning. Affective problems (e.g. depressive symptoms) associated with their gambling are understood as a consequence of their gambling, rather than a precursor. This subgroup is the most responsive to treatment and should be the easiest to prevent from experiencing gambling-related problems.

The second pathway identified by Blaszczynski and Nower refers to "Emotionally Vulnerable" (Pathway #2) gamblers, who suffer from an underlying affective dysregulation. Although they are vulnerable to the same conditioning processes and ecological risk factors as the "Behaviorally Conditioned" subgroup, the "Emotionally Vulnerable" gamblers engage in problem gambling in response to their affective state, as a means of emotional regulation. These gamblers can also adopt other maladaptive behaviors (e.g., drug use) to cope with their emotional difficulties. Their problem gambling levels are fairly steady and more severe than Pathway #1 gamblers. They are also more resistant to treatment, as the reasons underlying their gambling (i.e. emotional regulation) also need to be addressed. "Emotionally Vulnerable" gamblers therefore require a different form of preventive or curative treatment compared to "Behaviorally Conditioned" gamblers.

The third and final subgroup of problem gamblers, i.e. the "Antisocial-Impulsivist[1]" (Pathway #3) gamblers, display psychosocial vulnerabilities similar to those of Pathway #2 gamblers. However, they are distinguished by their biological, and possibly genetic, vulnerabilities; critical brain regions involved in impulse-control are affected. These gamblers can manifest a range of behaviors typically associated with impulse-control disorders. For example, they may exhibit

[1] This group has been renamed "Biologically Vulnerable" since the original publication of the Pathways Model (A. Blaszczynski, personal communication, July 12, 2014)

higher percentages of Attention Deficit Hyperactivity Disorder (ADHD), non-gambling related antisocial or delinquent behaviors, multiple drug use, and a family history of antisocial behavior. The most severe levels of problem gambling are found within this subgroup. Its members are also the most resistant to treatment, as well as to prevention efforts.

The Pathways Model will have tangible implications for the prevention of problem gambling, provided it is accurate. Its assumption is that each pathway develops gambling problems for distinct reasons, and therefore, each pathway must be addressed in a unique way.

Empirical Validation among Adults

Out of the 17 studies identified by Milosevic and Ledgerwood (2010), only one used a longitudinal design (Ledgerwood & Petry, 2010), allowing the authors to disentangle factors that predict from factors that result from problem gambling. More specifically, Ledgerwood and Petry (2010) divided their participants into three arbitrary groups based on the Pathways Model. Segmentation was done according to participants' self-reported levels of impulsivity, depression and anxiety (i.e., Pathway #1: low levels for each variable; Pathway #2: high levels for anxiety and depression; Pathway #3: high levels on all 3 variables). They then compared the occurrence of other factors (e.g. antisocial personality, drug abuse, gambling severity) among these three groups and observed results that were consistent with the Pathways Model (with the exception of illegal acts related to gambling, which was uniformly spread across groups).

Since then, additional evidence for the Pathways Model has been provided in a cross sectional study using a nationally representative sample of American adults (Nower et al., 2013). In their study, Nower and her colleagues selected a sub-sample comprised of participants at risk of developing clinical levels of GD (n = 581): all participants had gambled at least 5 times in the past year and exhibited at least 3 diagnostic criteria out of 10 (from the DSM-IV-TR, at the time). They

used a wide range of variables (17 total) that reflect in one way or another the three groups described by the Pathways Model. The authors conducted Latent Class Analyses (LCA) to identify the optimal number of underlying groups. In this case too, the 3-class solution emerged as the best-fitting model with all three groups corresponding to those presented in the Pathways Model. However, unlike the theory, this study found that the presence of ADHD was not limited to participants belonging to the Pathway #3 group but was also observed among participants in the Pathway #2 group. On the other hand, the authors found a higher level of Antisocial Personality Disorder (ASPD) and drug abuse prevalence among participants who fell under the Pathway #3 group.

Empirical Validation among Adolescents

Gupta et al. (2012) were the first to attempt the empirical validation of the Pathways Model within an adolescent population. Their analysis (also LCA) was conducted among a sample of 109 adolescents who showed significant gambling problems (3 or more diagnostic criteria from the DSM-IV), similarly to Nower et al.'s (2013) study. They selected a total of 11 variables listed in the Pathways Model. The study revealed the presence of five subgroups of gamblers, including three which were concordant with Nower and Blaszczynski's theoretical model. The additional two groups consisted of one group that only showed symptoms of depression and another exhibiting both internalizing and externalizing symptoms (this last group was deemed to represent an overlap of pathways #2 and #3). Hyperactivity symptoms were found to be present in four out of the five groups, whereas the Pathways Model expects them to only be present among Pathway #3 gamblers.

Allami, Vitaro, Brendgen, Tremblay, and Lacourse (2014) were the first to use a longitudinal design to empirically validate the Pathways Model among adolescents[2]. Using an at-risk sample of 17-year-old male problem gamblers (n = 149), the authors conducted LCA on a set of risk factors that were measured when the participants were 14 years old (impulsivity, delinquency, symptoms of depression, and drug use) and concluded that a 3-class model fit the data best. These three classes closely resemble the different profiles described in the Pathways Model. By assessing these same variables when participants reached 17 and 23 years of age (i.e., once gambling problems had already emerged), the authors observed a worsening of depressive symptoms, general delinquency, and drug use. This longitudinal comparison helped the authors determine which personality characteristics and behaviors preceded the emergence of gambling problems and which ones resulted from it.

In sum, there is growing empirical evidence to support the notion that there are three major pathways to problem gambling in adolescents, much in the same way as for adults. A differential prevention approach is thus required.

Prevention of Problem Gambling

Prevention programs are divided into two main categories (Gordon, 1983; Mrazek & Haggerty, 1994). Universal prevention programs target the general population without considering the individual characteristics of the people receiving the program. An example would be a prevention program administered to all high school students. Targeted prevention programs aim at a subset of the population that displays risk factors known to be related to a specific problem. Targeted

[2] À noter qu'il s'agit de résultats préliminaires présentés dans le cadre d'un congrès, et non des résultats présentés dans les études empiriques de cette thèse

prevention programs are called “indicated” if the risk factors used to screen the participants refer to personal characteristics (i.e., impulsive individuals or daily gamblers). They are called “selective” if the risk factors used to screen the participants refer to environmental features (i.e., adolescents with problem gambler parents or exposed to internet gaming).

Current Practices in Reference to the Prevention of Youth Problem Gambling

Ladouceur, Goulet, and Vitaro (2013) concluded from their review of prevention programs aimed at youth populations that all available programs were done at a universal level, despite increasing knowledge about the impact of different risk factors. Many of these programs seek to transmit accurate information about gambling activities, such as helping children better understand the actual probabilities of winning (Ladouceur, Ferland, & Fournier, 2003). Other programs focus on educating adolescents on the possible consequences of problem-gambling (e.g., by providing an accurate portrayal of individuals suffering from GD), and informing them of the resources available should they, or someone they know, need help (Ladouceur, Ferland, Vitaro, & Pelletier, 2005).

Unfortunately, most of these prevention programs have led to disappointing results. Although most programs are effective in educating youth populations about gambling issues and their associated risks, gambling behavior and gambling problems remained unchanged (Ladouceur et al., 2013). Moreover, none of the programs adopted a differential perspective in accordance to participants’ characteristics. Therefore, a universal “one-size fits all” approach might not be sufficient in preventing at-risk youths from developing gambling problems.

The Future of Prevention for At-risk Youth in Light of the Pathway Model

Universal programs

On a universal level, programs should address characteristics common to all pathways (e.g., biased cognitions related to gambling, resisting peer pressure). On a macro level, universal programs may include the use of government laws or guidelines that limit accessibility to gambling, as is already the case in all Canadian provinces (Campbell & Smith, 1998). On a local level, school-based programs may be the easiest way to reach a large number of youths. This method is especially effective in including Pathway #1 individuals who may not be considered eligible for targeted prevention since they do not manifest any observable predispositions, except perhaps the lack of coping skills and awareness of gambling risks. The content of a universal program should include information about winning probabilities and common misconceptions (i.e. gambler's fallacy), as well as lessons on how to develop resistance skills. These elements can be integrated into an already established program designed to address issues such as bullying or substance use.

Derevensky, Gupta, and Baboushkin (2007) provided evidence in support of this form of approach. They conducted an experiment in which they attempted to modify children's cognitions about their gambling behavior. Ten and 12-year-old children were randomly put under one of three experimental conditions involving a computer gambling task. The three experimental conditions consisted of winning rates of either 90%, 50% or 10%. All children in the 10% winning rate condition were less likely to attribute the gambling outcome to personal skills after the experimental manipulation. Children in the 50% and 90% conditions showed a tendency toward similar results, but only the 10% winning rate group maintained their beliefs over time (as recorded in a four-week follow-up). The results also indicated that younger children (10 vs. 12 years old)

were more likely to adjust their beliefs (i.e. believe that luck is a major component of gambling outcomes). These protective beliefs were extended to other gambling activities, but not to non-gambling related activities (i.e., such as those that are skill-driven). Put together, these results demonstrate that a universal prevention program addressing faulty cognitions should be conducted before the end of elementary school, when children are around 10 years old (or in grade five). Pfau and Bockern (1994) also found children in this developmental period (i.e. transition from elementary to middle school) to be the most responsive to smoking prevention programs, thus lending support to the notion that a universal program for addictive behaviors should be implemented no later than grade five.

With regards to resisting the effects of peer pressure, Inoculation Theory (McGuire, 1961) has effectively guided prevention programs that have yielded encouraging results with regards to alcohol use (Duryea, 1983) and smoking behavior (Pfau & Bockern, 1994). Such programs rely on the principle that when an individual is confronted with a persuasive message which goes against their initial beliefs (e.g., a peer encouraging another to engage in a risky behavior), their position is strengthened if counter-arguments to the persuasive message are prepared in advance.

The fact that previous efforts in validating universal prevention programs have not yielded consistent results may be due to the possibility that only a subset of at-risk gamblers benefit from them; hence resulting in a low (or null) effect size. Since only Pathway #1 individuals are expected to benefit from universal programs, results from these programs should be analyzed using the participants' personality profile as a moderating factor.

Our perspective furthermore suggests that universal programs may not be sufficient in preventing the emergence of gambling problems among youths who fit a Pathway #2 or Pathway #3 profile. As pointed out in the Pathways Model, these adolescents seek out gambling activities

as a way of coping with underlying affective disorders or expressing their delinquent orientations. Complementary prevention programs, which account for these subsets of at-risk adolescents, should be introduced to address these situations.

Targeted programs

In Canada, targeted (either selective or indicated) prevention programs for problem gambling have yet to be implemented among adolescents (i.e. 17 years old and younger; Ladouceur et al., 2013). It is critical for such programs to be able to correctly identify individuals at risk of developing gambling problems in order to adjust their targets and contents accordingly. A trial prevention program called *Pre-Venture* has already been conducted to thwart substance use and misuse (Conrod, Castellanos-Ryan, & Mackie, 2011; Conrod, O'Leary-Barrett, & Newton, 2013; Conrod, Stewart, Comeau, & Maclean, 2006; Conrod et al., 2000) using this tailored approach. Participants are split into four groups according to their personality-types (anxiety-sensitivity, hopelessness, impulsivity, and sensation-seeking) and receive cognitive restructuring interventions tailored to their profiles. Remarkably, studies measuring the efficacy of the program found that only two 90-minute group workshops were sufficient to have a positive impact on adolescents' drinking behavior (Conrod et al., 2013; Conrod et al., 2006; Conrod et al., 2000). This influence lasted for up to two years after these programs were implemented (Conrod et al., 2011).

The Pathways Model framework should serve as a stepping stone for identifying individuals who are at risk of developing gambling problems, since each person expresses a different set of risk factors depending on their pathway. Individuals belonging to the Emotionally Vulnerable and Biologically Vulnerable pathways especially should be distinguished and be subject to different prevention programs.

Emotionally Vulnerable. Individuals at risk of following the second pathway to problem gambling first develop internalized disturbances (such as anxiety or depression) which can generate maladaptive coping behaviors to handle their distress. Gambling behavior emerges as one form of coping mechanism to escape this distress (Dussault, Brendgen, Vitaro, Wanner, & Tremblay, 2011). It is therefore necessary for these individuals to acquire useful coping skills to help resist from succumbing to the temptation of choosing instantly gratifying, yet maladaptive, solutions.

The *Pre-Venture* trial described above may provide useful insights for implementing a prevention program for gambling problems even though the study related to substance use. By targeting the anxiety-sensitivity and hopelessness personality types, this prevention program follows the same rationale as a program designed in accordance with the Pathways Model when determining ways to provide help for adolescents following Pathway #2. In the *Pre-Venture* trial, participants with these two personality types were taught to recognize and modify cognitive distortions, which were taken from previously developed cognitive restructuring interventions for anxious and depressed individuals (Conrod et al., 2000).

Emotionally Vulnerable youth should therefore benefit from a prevention program that specifically addresses their emotional vulnerabilities in conjunction with a universal program. This statement is concordant with the recommendations put forward by Offord, Kraemer, Kazdin, Jensen, and Harrington (1998), who suggest that targeted preventions are most effective if universal programs are concurrently addressing other risk factors (such as faulty cognitions related to gambling in our case). Other prevention programs designed specifically for internalized disorders may be connected to problem gambling by shedding light on how gambling may be used as a tempting, but maladaptive, form of coping with undesirable feelings.

Biologically Vulnerable. The high-risk individuals from Pathway #3 display a propensity to gravitate toward immediately-gratifying activities. Although this group may also exhibit emotional disturbances, their impulse-control deficiency is seen as being biologically determined (i.e. neurological abnormalities), rather than as a consequence of their emotional turmoil. These individuals may benefit from learning adaptive emotional coping skills, but the primary concern with regards to their gambling behavior is at the impulse-control level. Individuals who fit this profile also exhibit delinquent behaviors independent of their gambling. Therefore, members of this group should strengthen their capacity to delay gratification and inhibit impulses, as well as diminish their co-occurring risky behaviors; all of which can have adverse consequences on their well-being. The *Pre-Venture* trial has demonstrated the efficacy of targeting cognitive distortions to prevent substance abuse among impulsive and sensation-seeking individuals.

The Montreal Experimental Program (MEP) is a promising prevention program that has proven itself to be effective in reducing general delinquency (Vitaro, Brendgen, & Tremblay, 2001) and substance use (Castellanos-Ryan, Séguin, Vitaro, Parent, & Tremblay, 2013) among male adolescents. The MEP is a 2-year program aimed at highly disruptive children who have been screened before they completed kindergarten. The disruptiveness scale used in this program evaluates aspects of hyperactivity–aggressiveness and opposition-related behaviors; two dimensions that are reminiscent of the Biologically Vulnerable pathway.

A notable strength of this program is the fact that prosocial boys were included in the intervention group. This was done for two purposes. First, members of this particular group serve as positive models and reinforcement agents. Second, this integration encouraged targeted children to participate without being stigmatized by their peers. As demonstrated by Dishion, McCord, and Poulin (1999), prevention programs, which regroup deviant individuals, may inadvertently

promote deviancy among these same individuals through a process labelled “deviancy training”, by providing a social group in which antisocial behavior is normalized and taught amongst peers. Although it is unclear whether the *Pre-Venture* program takes this phenomenon into consideration, we believe that the deviancy training effect is of critical importance when assigning participants in pairs, especially since part of the program consists of role-playing situations where risk factors are present with peers, and challenging each other’s cognitions. The MEP also includes a parent-training component. Dretzke et al. (2009) conducted a meta-analysis of 57 studies examining the impact of parenting programmes for children displaying conduct problems and concluded that parenting modules contribute significantly to the desirable effects of prevention programs. In addition, some authors have found that proper parent supervision successfully prevents delinquency and substance use among adolescents (Branstetter & Furman, 2013; Dishion & McMahon, 1998; Kiesner, Poulin, & Dishion, 2010; Stattin & Kerr, 2000).

Once Pathway #3 individuals develop a gambling disorder, they are the most resistant to treatment. Therefore, they may also require more resources in terms of prevention. In order to effectively target this group of gamblers, a substantial multi-modal approach would be necessary. We therefore recommend that programs should consider implicating parents, school boards, peers, as well as the at-risk individuals in order to be effective. Such programs should also target environmental risk factors that might exacerbate the development of problem gambling in at-risk individuals, such as exposure to pro-gambling norms, either within or outside the family. Finally, they should be implemented at an early stage of a person’s life (i.e. by the end of elementary school) before attitudes and behaviors become ingrained.

Conclusion

Ever since GD has been integrated to Substance Use Disorders (SUD) in the DSM-5, it has become increasingly clear that prevention practices for gambling problems can find inspiration in programs that have been developed and validated for SUD. Universal prevention programs aimed at addressing gambling problems are doomed to fail for a large proportion of at-risk problem gamblers if they are administered alone. It is therefore crucial for universal programs to be complemented with targeted programs that seek out groups which fit into the Emotionally or Biologically Vulnerable pathways to problem gambling. The use of a differential approach to prevention has shown to be successful in addressing SUD, which is why we believe it is now time to apply the same method to GD.

Researchers and other professionals who wish to implement and evaluate a prevention program structured around the Pathways Model may not only increase their probability of effectively preventing gambling problems, but also generate additional empirical validation of the model by testing its basic tenets. For instance, targeted prevention methods addressing impulsive and delinquent characteristics must demonstrate greater effect sizes among the Biologically Vulnerable subset of youth (i.e. screened for existing impulsive and delinquent traits), compared to the Emotionally Vulnerable or Behaviorally Conditioned subset. The same goes for targeted prevention methods directed at internalized symptoms, and their effect on the Emotionally Vulnerable subset of participants. Including and testing for relevant mediating factors that are specific to each pathway would reinforce the case for differentiated prevention approach, and by extension, the Pathways Model in which it finds its roots.

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Discussion Générale

Rappel des objectifs et des principaux résultats

Les résultats de cette thèse contribuent à l'image générale que les joueurs problématiques ne forment pas un groupe homogène. À partir d'expériences cliniques et de documentation scientifique, Blaszczynski et Nower (2002) ont élaboré le modèle des parcours multiples, afin de mettre en place un modèle cohérent pouvant expliquer diverses observations apparemment contradictoires. Au fil des années, plusieurs chercheurs ont contribué à bâtir une bibliographie soutenant la validité du modèle théorique. Toutefois, des limites méthodologiques des études antérieures n'ont pas permis de capturer l'aspect développemental du modèle théorique. Après avoir identifié et comparé divers profils de joueurs, nous avons proposé une approche de prévention et d'intervention différenciée adaptée aux profils de joueurs à risque.

Identification des profils

Les études empiriques menées dans le cadre de cette thèse ont permis d'identifier quatre profils d'individus, à partir de caractéristiques personnelles minutieusement sélectionnées pour distinguer les profils suggérés par le modèle des parcours multiples, et mesurées au tout début de l'adolescence (objectif 1). Ces profils se distinguent sur deux dimensions principales : symptômes d'internalisation et d'externalisation. Le profil des Bien Ajustés, caractérisés par des niveaux bas sur les échelles d'internalisation et externalisation, correspond au parcours CC du cadre théorique. Le profil des Internalisés est caractérisé, comme le nom l'indique, par des niveaux élevés sur les échelles d'internalisation et des niveaux bas sur les échelles d'externalisation; celui-ci correspond au parcours EV. De manière analogue, le profil des externalisés correspond au parcours des BV.

Quoique la première étude empirique ait ciblé des joueurs avec au moins deux problèmes à l'adolescence ou au moins quatre problèmes au début de l'âge adulte, il ne faudrait pas en déduire qu'il s'agit d'un échantillon de joueurs ayant atteint le seuil clinique du trouble lié aux JHA; il s'agirait plutôt de ce que l'on appelle des joueurs problématiques.

À priori, nous nous attendions à observer seulement les trois profils susmentionnés. Toutefois, l'émergence d'un quatrième profil ne contrevient pas à la validité du modèle théorique, qui n'exclut pas la possibilité de profils additionnels. Le dernier profil, caractérisé à la fois par des niveaux élevés sur les échelles d'internalisation et d'externalisation, est dûment appelé Comorbide, et correspond à un profil hybride ressemblant à une superposition des parcours EV et BV. Un commentaire supplémentaire est de mise. Quoique les auteurs du modèle des parcours multiples admettent que les joueurs BV peuvent aussi démontrer des symptômes d'internalisation, il est précisé que ceux-ci se distinguent principalement par leurs signes d'externalisation. Ainsi, les participants du profil Comorbide, qui rapportent les niveaux les plus élevés à la fois de symptômes d'internalisation et d'externalisation, ne sont pas uniquement caractérisés par leurs traits d'externalisation. Pour cette raison, le profil Comorbide est considéré comme un quatrième parcours hybride, qui ne fait pas directement référence au parcours BV.

Comparaison des profils

Les quatre profils ont été comparés en termes de problèmes de JHA auto-rapportés à 16 et 23 ans (objectif 2), et au niveau de leur répartition homme/femme (objectif 5). Dans nos deux études, et conformément à nos attentes, les hommes ont été surreprésentés au sein des profils ayant une forte composante d'externalisation (c.-à-d., Externalisé et Comorbide). Ces mêmes deux

profils ont aussi eu tendance à rapporter davantage de problèmes de jeu que les deux autres profils (c.-à-d., Bien Ajusté et Internalisé).

Grâce à l'identification au sein de l'ensemble de nos échantillons de profils analogues à ceux du modèle des parcours multiples, nous avons pu poursuivre d'autres analyses afin d'identifier des facteurs de protection ou de compensation pouvant prédire la non-émergence des problèmes de jeu à 16 et 23 ans (objectif 4). Il a été intéressant de constater que certaines variables exercent un effet compensatoire ou protecteur dépendamment de l'âge auquel les problèmes de JHA ont été mesurés, suggérant un effet développemental de ces variables et soulignant la valeur ajoutée des devis longitudinaux.

Des effets compensatoires sur les problèmes de JHA à 16 ans ont pu être observés pour tous les facteurs mesurés, à l'exception du soutien des pairs. Ce résultat est conforme aux résultats d'études antérieures, et, contrairement à nos attentes, une classification par profil n'aura pas permis de dégager un effet. Plus tard, à 23 ans, nous observons encore des effets compensatoires pour tous les facteurs mesurés, à l'exception du soutien parental cette fois-ci. Toutefois, il appert que l'effet compensatoire observé au niveau de la supervision parentale serait principalement dû au groupe des internalisés, qui sont les seuls à bénéficier de cet aspect une fois que les joueurs sont classifiés en profils.

Retombées théoriques

Les études présentées dans cette thèse sont les premières à identifier des profils analogues à ceux du modèle des parcours multiples, avant que les problèmes de JHA n'apparaissent, permettant ainsi de statuer sur la directionnalité de l'influence entre les facteurs à risque et le jeu problématique. En effet, lorsque des facteurs de risque sont mesurés en même temps qu'un

comportement cible (en l'occurrence, les JHA), il est impossible de déterminer laquelle de ces deux parties exerce une influence sur l'autre. Ainsi, l'étude initiale présentée dans cette thèse est la première à pouvoir suggérer que les facteurs de risque proposés par le modèle des parcours multiples peuvent mener à l'un ou l'autre des parcours énoncés. Il faut toutefois demeurer prudent quant aux interprétations de causalité en raison de la présence possible de tierces variables confondantes associées à la fois avec les problèmes de JHA et les facteurs de risque mis en jeu dans cette thèse.

La découverte d'un quatrième profil de joueurs (c.-à-d., « comorbide » ou « emotivo-biologiquement vulnérable ») vient supplémenter la formulation actuelle du modèle théorique. En effet, les études ayant utilisé des analyses de classes latentes pour tenter de valider le modèle théorique ont trouvé un nombre variable de profils (de trois à cinq; Gupta et al., 2012; Nower et al., 2013). Par la nature-même de ce type d'analyses, le nombre final de profils est dégagé librement en fonction des données empiriques, plutôt qu'a priori et de manière arbitraire, tel qu'effectué par d'autres chercheurs (Ledgerwood et Petry, 2010; Valleur et al., 2015). À la lumière des informations colligées, il apparaît non seulement que les trois profils suggérés par le modèle des parcours multiples existent réellement, mais qu'un profil supplémentaire pourrait aussi exister, particulièrement lorsque les profils sont dégagés à partir de caractéristiques mesurées à l'adolescence (tel qu'observé dans nos deux études empiriques et celle de Gupta et al., 2012). Quand les échantillons sont relativement petits, comme cela est souvent le cas lors d'études de joueurs présentant des niveaux élevés de jeu problématique, ce quatrième profil pourrait passer inaperçu, ou se fondre avec les profils émotionnellement ou biologiquement vulnérables.

Retombées pratiques

Les données probantes présentées dans cette thèse apportent un soutien empirique majeur au modèle des parcours multiples, malgré que les facteurs de risque mesurés dans nos études ne représentent qu'une partie de l'ensemble des caractéristiques de chaque profil. Une définition exhaustive de chaque parcours serait allée jusque dans les motivations sous-jacentes poussant les joueurs de chaque profil à poursuivre leur pratique de JHA. Par exemple, le profil EV jouerait pour moduler ses affects négatifs, alors que le profil BV serait avide de sensations fortes issues de situations à risque. En adoptant une perspective différenciée de l'étiologie du jeu problématique, les cliniciens ont tout à gagner en ciblant les motivations caractérisant le profil de la personne devant eux.

Par ailleurs, la deuxième étude a permis d'observer ces mêmes profils auprès d'une population générale, même de non-joueurs, ce qui ouvre la porte vers la mise en place de programmes de prévention auprès de la population générale, tout en gardant les mêmes profils applicables aux joueurs problématiques. La deuxième étude a aussi pu capturer un aspect particulièrement utile découlant de la nature développementale du modèle théorique. En mesurant les facteurs de risque plusieurs années avant les problèmes de JHA (les premiers étant évalués au début de l'adolescence, alors que les derniers en fin d'adolescence ou au début de l'âge adulte), il a été possible d'ajouter un temps de mesure intermédiaire, au milieu de l'adolescence, afin d'évaluer et proposer des cibles potentielles à intégrer aux programmes de prévention, permettant de minimiser l'impact des facteurs de risque sur l'émergence de problèmes de jeu. De plus, l'analyse différenciée des facteurs de protection en fonction de profils de personnalité à risque est une première pour le domaine des JHA. Ainsi, les programmes de prévention pourront, une fois avoir identifié les profils à risque, agir avant l'émergence des problèmes de JHA.

Tel que présenté dans le troisième article (c.-à-d., la recension des écrits sur la prévention), les programmes de prévention pour le jeu problématique sont généralement inefficaces, du moins pour ceux qui intègrent adéquatement une période de suivi à long-terme (Ladouceur et al., 2013). Cela pourrait être dû à l'habitude erronée de considérer les joueurs comme un groupe homogène. Considérant l'accumulation de données probantes soutenant l'hétérogénéité des joueurs de JHA, une approche de prévention différenciée devrait être préconisée (tel que démontré avec les problèmes de consommation chez les adolescents). Il s'agirait de placer les participants dans des ateliers spécifiques à leur problématique sous-jacente, évaluée préalablement. Par exemple, les joueurs présentant une symptomatologie d'internalisation participeraient aux ateliers traitant spécifiquement de ce sujet. Toutefois, aucun programme adoptant cette approche n'a encore été mis en place et évalué pour les JHA.

Forces et limites des articles

Les forces et limites des études présentées dans cette thèse ont été abordées dans les sections correspondantes pour chacune des études empiriques. Néanmoins, certains aspects méthodologiques communs aux deux études méritent d'être réitérés. Premièrement, nos questions de recherche ont été abordées en faisant appel à des méthodes statistiques à la fine pointe des connaissances actuelles. Par exemple, alors que la plupart des études antérieures analysent les problèmes de JHA à l'aide de régressions linéaires, nous avons opté pour la régression binomiale négative (utilisant une distribution de Poisson) spécialement conçue pour analyser des données de dénombrement (*count data*) ; chose que sont les mesures de problèmes de JHA tel que le SOGS. De plus, contrairement aux études ayant imposé un nombre précis de profils avec une structure prédéterminée, nous avons utilisé des analyses de profils latents qui ont permis de dériver librement les profils en spécifiant seulement quelles variables seraient importantes à observer. Tel

que présenté en introduction, cette approche est nécessaire pour fournir un appui crédible dans la validation du modèle, tout en laissant place à l'émergence de profils imprévus. Un deuxième atout de nos études repose sur l'adoption d'un devis longitudinal, allant du début de l'adolescence au début de l'âge adulte, pour valider le modèle des parcours multiples. En effet, étant donné l'aspect développemental du modèle, il est impératif d'utiliser des données longitudinales afin de clarifier la directionnalité des liens entre les facteurs prédicteurs (néfastes ou bénéfiques) et le jeu problématique.

Malgré ces forces méthodologiques, quelques failles demeurent. Premièrement, même si les facteurs de risque et de protection ont été mesurés avant les problèmes de JHA, la méthodologie utilisée ne permet pas de statuer hors de tout doute que ces facteurs causent effectivement l'émergence des problèmes de JHA. Il aurait fallu, entre autres, avoir une mesure de JHA concomitante aux facteurs de risque et de protection afin d'établir une base de référence. Néanmoins, quelques indices nous permettent d'envisager cette possibilité. Premièrement, la moyenne d'âge d'initiation aux JHA dans nos échantillons est de 12,5 ans, ce qui correspond approximativement à l'âge de collecte des facteurs de risque ; par conséquent, nous pouvons supposer que ce serait effectivement le cas pour une partie de notre échantillon. Deuxièmement, en ce qui concerne les problèmes de JHA à 23 ans, l'intégration d'une variable contrôle de JHA à 16 ans renforce l'argument que les différences entre groupes découlent des profils plutôt que de problèmes de JHA préexistants.

Quelques nuances s'imposent aussi suite à notre choix d'échantillons. Même si ceux-ci sont de relativement grande taille, les généralisations au grand public doivent se faire avec prudence. Un premier échantillon est composé d'une population particulièrement à risque (garçons issus de milieux défavorisés de la Grande Région de Montréal), alors que le deuxième découle aussi en

partie d'un suréchantillonnage de personnes à risque. Cela pourrait avoir engendré quelques anomalies dans nos résultats, telle qu'une estimation conservatrice des différences entre profils, ou l'identification d'un profil « Bien Ajusté » (ou CC) présentant des comportements à risque plus élevés que la normale. De plus, les données de JHA ayant été collectées il y a plus de 20 ans, il serait légitime de se questionner quant à la pertinence actuelle des résultats. Les deux échantillons, qui ont été combinés, sont décalés d'environ trois ans. Cela permet de contrer en partie les effets de cohorte. Il est certain toutefois qu'aujourd'hui, avec la popularisation de l'Internet et des appareils mobiles, ainsi que la diversification de l'offre de JHA, les pratiques des joueurs ne sont plus nécessairement les mêmes. En termes de facteurs de risque socio-culturels, nous pourrions donc supposer des changements. Toutefois, il n'y a pas de raison de croire que cela affecterait les facteurs de risque d'ordre personnel.

Ensuite, les échelles utilisées à 12 et 14 ans ont parfois été dérivées à partir de seulement deux ou trois items. Par conséquent, elles devraient être considérées davantage comme des mesures de dépistage que comme des indicateurs de trouble clinique. Par ailleurs, même si nous avons mis à profit diverses sources d'information (p. ex., enseignants et jeunes eux-mêmes), il n'en demeure pas moins que chaque mesure repose sur une seule source. L'utilisation d'un devis longitudinal nous permet d'apporter un soutien empirique quant à la direction des influences entre facteurs de risque/protection et problèmes de JHA. Toutefois, la directionnalité sous-entendue ne permet pas de statuer sur quelconque lien de causalité entre ces variables, car cela aurait nécessité un devis expérimental. Il aurait fallu, pour la dépression par exemple, répartir aléatoirement des joueurs avec des symptômes dépressifs dans un programme spécifique portant sur la réduction de la dépression (c.-à-d., un groupe expérimental), ou un groupe-témoin. Après avoir confirmé la diminution de symptômes dépressifs chez le groupe expérimental, ces deux groupes auraient

ensuite été comparés au niveau de leurs problèmes de JHA. Nous pourrions alors attribuer une part de responsabilité aux symptômes dépressifs sur les variations au niveau du nombre de problèmes de JHA chez ce groupe (comparativement au groupe contrôle). Finalement, étant donné la non-convergence des divers indicateurs d'ajustement utilisés pour les analyses de profils latents (plus particulièrement le BIC), nous avons dû opter pour une interprétation à la fois quantitative et qualitative de nos divers profils. Quoique certains auteurs recommandent de toujours concilier ces deux méthodes d'interprétation (Marsh, Hau et Wen, 2004), il aurait été préférable d'avoir des indicateurs statistiques cohérents.

Directions futures

Les études présentées dans cette thèse n'ont utilisé qu'une partie des variables caractérisant chacun des profils définis par le modèle des parcours multiple. Nower et Blaszczynski (2017), auteurs du modèle théorique, ont publié un outil de dépistage à 48 questions permettant d'évaluer divers aspects centraux à la différentiation des profils. Les facteurs suivants sont inclus : humeur avant et après l'émergence des problèmes de jeu ; abus et négligence pendant l'enfance ; jouer pour gérer le stress ; impulsivité ; le jeu donne un sens à la vie ; prise de risques ; prise de risques sexuels ; traits et comportements antisociaux. Par conséquent, les études désirant valider ou utiliser le modèle des parcours multiples en tant que cadre théorique devraient dorénavant utiliser cet instrument.

De plus, les études cherchant à explorer les effets bénéfiques de certains facteurs contre les problèmes de JHA devront distinguer les effets compensatoires des effets protecteurs, en fonction des caractéristiques à risque des profils des individus. Malgré le peu d'effets protecteurs identifiés par notre deuxième étude, celle-ci trace le chemin pour les études futures en démontrant la

possibilité d'identifier des facteurs de protection propres aux divers profils du modèle des parcours multiples. Étant donné que nous n'avons utilisé qu'une partie de l'éventail de variables compensatoires ou protectrices généralement associées au jeu, la recherche aura tout à gagner en adoptant cette méthodologie à d'autres variables, telles que la performance ou l'assiduité scolaire.

Afin de bonifier la documentation concernant les analyses de profil latents, nous recommandons que les statisticiens identifient des indicateurs d'ajustement spécifiques aux analyses de profils latents (utilisant des variables continues), par opposition aux analyse de classes latentes (aux variables catégorielles), car le BIC, quoique prisé dans la sphère des variables catégorielles (Nylund et al., 2007), ne semble pas s'appliquer aux variables continues ; plusieurs études ont trouvé qu'il ne cesse de diminuer au fur et mesure que le nombre de profils augmente (Geiser, Okun et Grano, 2014; Marsh, Lüdtke, Trautwein et Morin, 2009).

Considérant l'accumulation de soutien empirique pour le modèle, il est temps que des programmes de prévention contre les JHA soient développés et mis en place en adoptant une approche différenciée, selon les profils des individus. Cette approche pourrait être intégrée à des programmes existants sur les JHA, ou bien les JHA pourraient être intégrés à des programmes existants contre l'abus de substance utilisant une approche différenciée. Aucune étude n'a jusque-là évalué l'efficacité d'un tel programme pour les JHA, laissant ainsi libre cours aux futurs pionniers.

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