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Risk Factors for Problematic Gambling: A Critical Literature Review

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Abstract This article is a critical review of risk factors for pathological gambling categorized by demographics, physiological and biological factors, cognitive distortions, comorbidity and concurrent symptoms, and personality symptoms and characteristics. There is also a varia section (availability, parents playing, sensory characteristics, schedules of reinforcement, age of onset, and playing duration). The review found very few well established risk factors for pathological gambling (i.e. more than two studies to support the conclusions). Well established risk factors included demographic variables (age, gender), cognitive distortions (erroneous perceptions, illusion of control), sensory characteristics, schedules of reinforcement, comorbid disorders (OCD, drug abuse), and delinquency/illegal acts. An understanding of risk factors for pathological gambling should enhance prevention and treatment approaches.

Keywords Pathological gambling · Risk factors · Experimental studies · Significance

Introduction

Over the last several years, there has been an increase in gambling research. Substantial breakthroughs have emerged in understanding the clinical aspects of pathological gambling as well as in the area of treatment. Research regarding risk factors, however, is sparse.

Due to the lack of rigid empirical research, preventive actions become delayed. The aim of the present study is to organize the empirical facts regarding risk factors for the

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development and maintenance of pathological gambling and thereby enable adequate preventive actions.

Review Approach

Although empirically based critical reviews are almost non-existent, there are some publications with some form of an overview. We have found seven such studies (Shaffer and Hall 1996; Spunt et al. 1998; Wardman et al. 2001; Raylu and Oei 2002; Walthers 2002; Goudriaan et al. 2004; Welfe et al. 2004), which will be described below. We have not included treatment studies in the present article as their relevance for this review is doubtful.

The following were inclusion criteria for inclusion for publications (partly based on the research synthesis by Shaffer et al. 1999): (1) it included original research on PG; (2) it was published in international scientific journals; (3) it specified the size of the sample; (4) it specified the instrument used to identify disordered gambling and comparison variables; (5) it specified the design used; (6) it included and it specified adequate statistical analyses; (7) the documentation was adequate; and (8) the results were conclusive.

In relation to standards of proof, we have partly followed the discussion of effective treatments of Nathan and Gorman (1998) by classifying the risk factors into three levels:

(1) well-established risk factors for PG: several ($\geq$3) well-performed empirically validated studies;
(2) probable risk factors for PG: one or two well-performed empirically validated studies;
(3) possible risk factors for PG (not included in table, only in text): some indications of a risk factor, but not documented as a risk factor. In our review of the literature, we have used the two first levels in the tables (well-established risk factors, and probable risk factors). In addition we have mentioned some promising studies without rigorous documentation not enabling clear conclusions.

In the following text, "gambling machines" is used in place of the terms "fruit machines," "slot machines," and "electromagnetic machines." As the concept "probable pathological gambling" (Lesieur and Blume 1987) in most texts is equivalent to pathological gambling (PG), we have used the later descriptive term. In the present review, we use the term "problematic gambling" to denote the combination of pathological gambling and problem gambling (also called "at-risk" gambling). When a study makes many comparisons, one must strive to present only clearly pertinent facts, whilst restricting other extraneous detail.

Earlier Critical Reviews

Shaffer and Hall (1996) completed a meta-analysis on nine non-duplicative adolescent gambling studies in North America ($n = 7700$) until 1993. The different studies (published and unpublished) had used the following assessment instruments: DSM-III (APA 1987), SOGS (Lesieur and Blume 1987), SOGS-RA (Winters et al. 1993a), MAGS (Shaffer et al. 1994), and GA (Gamblers Anonymous twenty questions criteria). The meta-analysis showed PG prevalence rates from 3.5% to 8.5% (with a 95% confidence interval 4.4% to 7.4%). Shaffer and Hall (1996) advised the use of a multi-level classification scheme to reconcile divergent classification methods and to facilitate interstudy comparisons.

Spunt et al. (1998) reviewed the link between PG and substance misuse, scrutinized instruments to measure PG and drug misuse, and outlined treatment strategies. Although

the review is not a systematic categorizing review, it critically analyzes a number of studies and examines the relationship between PG and substance misuse.

In another review, Wardman et al. (2001) reported nine empirical studies, for prevalence rates of problem gambling and PG and examined factors statistically associated with problem gambling behaviour in an Aboriginal population. There were four adolescence studies (three ($n = 1423$) reported the prevalence of problem gambling (from 10.1% to 21%)), while the fourth ($n = 122700$) presented data on general gambling activity. There were also five adult Aboriginal studies ($n = 3827$). The authors also examined factors important for adult PG and found that pathological gamblers compared to non-problem gamblers had greater gambling involvement, made greater financial expenditures, and were younger.

Another comprehensive review of factors implicated in the development and maintenance of PG was performed by Raylu and Oei (2002). The authors examined three categories of risk factors: familial (including learning/genetics), sociological, and individual (including personality, biological/biochemistry (hemispheric dysregulation, neurotransmitters; arousal), cognitions, and psychological states).

Walters (2002) did a meta-analysis of genetic research on gambling and problem gambling, and was able to trace 17 family studies but only two twin studies. There was a small but significant overall effect with both family and twins on the gene-gambling relationship. The familial effect was strongest for the relationship between sons and problem gambling fathers. They also included moderating variables (sex, nationality, family, severity) in their analysis.

Two other earlier reviews should also be noted. Goudriaan and colleagues (2004) reviewed the bio-behavioral studies of PG. The review notes the limitations of our current knowledge and offers suggestions for future research regarding the neuropsychology and pathophysiology of PG. In another study examining the risk factors for PG, Welte and colleagues (2004) used a telephone survey ($n = 2168$) and found that certain forms of gambling (e.g. casino gambling; pull-tabs), number of types of gambling in a year, current alcohol use disorder, low socio-economic status, and being a racial/ethnic minority all significantly predicted gambling pathology.

Specific Non-empirical and Theoretical Publications

Brown (1986) developed a theoretical model, based on both his own and others' research on arousal and sensation seeking behaviour, and presented an eclectic model, including psychophysiological arousal, cognitive variables, affective factors, behavioural reinforcement schedules, and social and institutional determinants and relationships, as well as subcultural conditions (cross-sectional factors). These factors have different levels of impact at different stages of the development of gambling behaviour and PG. Brown also outlines a longitudinal view, including different phases in the development of gambling, with induction, adoption, promotion, and addiction.

Related to the psychophysiological arousal aspect of Brown's model, Griffiths (1991) discusses the psychobiology of the "near-miss", in relation to biological substrates, arousal theories, and endorphins. His research on arousal in gambling includes excitement (both subjectively and objectively), heart rate, and psychology of the "near-miss".

Sharpe (2002) formulated a biopsychosocial perspective with a cognitive-behavioural model of problem gambling. The article highlights ADHD (Attention Deficit Hyperactivity Disorder), impulsivity, availability of gambling, and biological markers implicating neurotransmitter systems (dopamine, norepinephrine, and serotonin) as vulnerability factors.

Physiological aspects (arousal, heart rate), and cognitive factors and cognitive bias are mentioned.

Gupta and Derevensky (2000) present a useful list of risk factors, with references. They discuss how to proceed from research to treatment. Dickson et al. (2002) present a conceptual framework with a 16–page table, containing a very good review with risk and protective factors as well as corresponding prevention findings for adolescent substance use and abuse. With alcohol data as a background, the authors attempt to build a general theoretical model for the prevention of adolescent risk behaviors, applicable for potential prevention of gambling problems in youth.

The pathways model of problem and pathological gambling presented by Blaszczynski and Nower (2002) could be a series of useful tools in the analysis and treatment of pathological gambling but does not fit in the present empirical review of risk factors for PG.

Review of Risk Factors

Attempts to study risk factors not meeting our inclusion criteria merit mentioning in the text, without inclusion in the table, to give a better overview and understanding of the field. For condensed results, please see Table 1. For more information on results, please refer to the text.

To give a summary of results and provide an impression about how scarce empirical studies are, Table 2 is compiled as a summary, detailing the amount of such studies that have been performed on each risk factor. Conclusions about the well-established and probable or potential risk factors are also indicated within the Table.

Demographics

Demographic variables have often been mentioned as possible risk factors for PG but they have seldom been empirically tested.

Age

Ladouceur et al. (1999a) studied 3426 high school students in a correlational design using the SOGS (South Oaks Gambling Screen) (Lesieur and Blume 1987) and its relation to grades in school. A univariate ANOVA (Analysis of Variance) on SOGS scores and grade level showed a statistically significant main effect ($F = 7.73$, $p < .001$). Furthermore, a Sheffé test showed higher SOGS scores for younger students (8 grade compared to 10–11th grade; $p < .05$).

Bondolfi et al. (2000) used the SOGS to analyze different risk factors in a correlational designed telephone interview prevalence study ($n = 2526$) on gambling in Italy. The results showed that being younger than 29 years of age was a significant risk factor ($X^2 = 17.1$, $p = .01$).

Volberg et al. (2001) performed a large prevalence study using the SOGS-R and DSM-IV (APA 2000) in a Swedish sample ($n = 8845$). Life-time problem and pathological gamblers were compared to non-problem gamblers on different variables. Age (younger than 25) was shown to be a significant risk factor (OR = 2.51, $p = .000$).

These studies suggest that younger age (i.e. younger than 29 years old appears to be a significant risk factor for PG.

Table 1 Risk factors for pathological gambling (dependent variable: PG) [rev 2004-04-05 final]

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	p value	Study
1. Demographics							
1.1 Age							
	3426 HS stud	SOGS	School level	Younger	$F = 7.73$	<.0001	Ladouceur et al. (1999a)
	2526 calls	SOGS	Age	Younger	$X^2 = 17.81$	.01	Bondolfi et al. (2000)
	562 calls	Questionnaire	Age	Younger	$X^2 = 25.29$	≤.0001	Potenza et al. (2001)
	8845 normals	SOGS-R	Age	Younger	OR = 2.51	.000	Volberg et al. (2001)
1.2 Gender							
	220 MMTP	SOGS	Gender	Male	$r_{xy} = .12$	.04	Feigelman et al. (1995)
	3426 stud	SOGS	Gender	Male	$X^2 = 39.52$	<.001	Ladouceur et al. (1999a)
	2526 calls	SOGS	Gender	Male	$X^2 = 8.94$	.01	Bondolfi et al. (2000)
	8845 normals	SOGS-R	Gender	Male	OR = 3.71	.000	Volberg et al. (2001)
1.4 Marital status							
	2526 calls	SOGS	Marital status	Married	$X^2 = 7.52$	.02	Bondolfi et al. (2000)
	8845 normals	SOGS-R	Marital stat	Non-cohabitng	$X^2 = 121.67$	.000	Volberg et al. (2001)
1.5 Income/Finance							
	2526 calls	SOGS	Income	Higher	$X^2 = 10.88$	.01	Bondolfi et al. (2000)
	562 calls	Questionnaire	Income	Higher	$X^2 = 5.79$	<.02	Potenza et al. (2001)
	562 calls	Questionnaire	Problems	More	$X^2 = 4.21$	=.04	Potenza et al. (2001)
1.6 Employment							
	220 MMTP	SOGS	Empl status	Non-empl	$r_{xy} = .15$	.02	Feigelman et al. (1995)
	313	DSM-III-R	Unemployed	Increase	$t = 11.09$	<.001	Hall et al. (2000)
1.7 Social welfare status							
	8845 normals	SOGS-R	Soc welf stat	On welfare	$z = 2.41$	.05	Volberg et al. (2001)
1.8 Residence							
	8845 normals	SOGS-R	Residence	Big city	$z = 4.00$	.01	Volberg et al. (2001)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	*p* value	Study
1.9 Academic achievement							
	3426 HS stud	SOGS	Acad level	Lower	$F = 19.44$	<.001	Ladouceur et al. (1999a)
	702 (15-18)	SOGS-RA	Low grades	More	$X^2 = 21.7$	<.001	Winters et al. (1993)
1.10 Immigrants and ethnic groups							
	562 calls	Questionnaire	African/am	Increase	$X^2 = 3.87$	<.05	Potenza et al. (2001)
	8845 normals	SOGS-R	Birth country	Born abroad	OR = 2.08	.01	Volberg et al. (2001)
	2168 calls	DIS	African/am Hisp/Asian	Increase	IRR 1.96-4.71	.01	Wetle et al. (2004)
2. Physiological and biological factors							
2.1 Heart rate and arousal							
	12 gambl/ 12c	Polygraph	HR	Increase	$r_{xy} = .741$	<.0005	Andersson and Brown (1984)
	22 hi/22low freq	Polygraph	HR	Increase	$F = 6.523$	<.05	Leary and Dickerson (1985)
2.2 Transmittor activity							
	24 PG/20c	CSF probes	MHPG	Red plasma lev	$t = 2.9$	<.007	Roy et al. (1988)
	24 PG/20c	Urinary probes	NA	Incr urinary outp	$F = 11.6$	<.0003	Roy et al. (1988)
	27 PG/27 match	Blood probes	Platelet MAO	Decreased	Wilcoxon pairs	<.01	Blanco et al. (1996)
2.3 Genetic studies							
	10 PG/7c	CSF probes	DA	Decrease	22.1/34.3	<.01	Bergh et al. (1997)
	10 PG/7c	CSF probes	DOPAC	Increase	117.0/77.1	<.01	Bergh et al. (1997)
	10 PG/7c	CSF probes	HVA	Increase	477.1/297.6	<.01	Bergh et al. (1997)
	10 PG/7c	CSF probes	DOPAC/DA	Increase	5.7/2.4	<.01	Bergh et al. (1997)
	10 PG/7c	CSF probes	HVA/DA	Increase	23.0/8.9	<.01	Bergh et al. (1997)
	10 PG/7c	CSF probes	NA	Increase	128.8/85.7	<.01	Bergh et al. (1997)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	p value	Study
	10 PG 7c	CSF probes	MHPG	Increase	23.0/8.9	<.01	Bergh et al. (1997)
	22PG/23 c	Genotyping	D2A1 allele	Present	OR = 5.03	<.000	Comings et al. (1996)
	69 PG	SOGS/DSM-IV	DRD2 polym	Increased	$X^2 = 13.9$	.003	Ibanez et al. (2001)
3. Cognitive distortions							
3.1 Erroneous perceptions or biased evaluations							
	29 students	Experiments	Biased memory	More biased	$t = 2.33$	<.05	Gilovich (1983)
	64 students	Experiments	Selective memory	More selective	$t = 2.33$	<.05	Gilovich (1983)
	100 Ss exp/100c	Interview	Regular players	Probab of win	$X^2 = 6.94$	<.01	Savoie and Ladouceur (1995)
	100 Ss exp/100c	Interview	Regular players	Inc win chance	$X^2 = 13.66$	<.01	Savoie and Ladouceur (1995)
	44 reg players/ exp/c 22/22	Questionnaire	Reg players less confident to win		$F = 7.38$	<.025	Savoie and Ladouceur (1995)
	44 reg players/ exp/c 22/22	Questionnaire	Control group reduced playing		$t = 3.2$	<.025	Savoie and Ladouceur (1995)
3.2 Illusion of control							
	36 students	Exp:bias (con/shy)	Bias	Illusion of contr	$t = 5.46$	<.005	Langer (1975)
	47M/6F Ss	Exp:choice	Bias	Illusion of contr	$t = 4.33$	<.005	Langer (1975)
	13 Ss	Exp.stim famil	Bias	Illusion of contr	$t = 5.46$	<.005	Langer (1975)
	13F/2M Ss	Exp:involvement	Bias	Illision of contr	$F = 7.33$	<.01	Langer (1975)
	49F/73M Ss	Exp:field setting	Bias	Illusion of contr	$F = 7.58$	<.01	Langer (1975)
	31M/10F Ss	Exp:ctr field st	Bias	Illusion of contr	$X^2 = 4.19$	<.05	Langer (1975)
	90 students	Exp:sequence	Skill feeling	Winning bias	$F = 4.20$	<.05	Langer and Roth (1975)
	80 stud, 36 Ss	Exp:loose bet	Bias	Illusion of contr	$F = 10.72$	<.001	Gilovich and Douglas (1986)
	160 students	Exp:rand outc	Bias	Illusion of contr	$t = 2.56$	<.02	Gilovich and Douglas (1986)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	*p* value	Study
4.Varia							
4.1 Availability of plays							
	1002/1257	SOGS	Availability	75%	$X^2 = 19.39$	<.001	Ladouceur et al. (1999b)
4.2 Sensory characteristics							
	29 PG/31 n-PG	SOGS	Sensory features	Main effect	$F = 11.29$	<.001	Loba et al. (2001)
	29 PG/31 n-PG	SOGS	Sensory features	Game interaction	$F = 5.50$	<.01	Loba et al. (2001)
	29 PG//31n-PG	SOGS	Sensory features	Excitement	$F = 9.85$	<.001	Loba et al. (2001)
	29 PG/31 n-PG	SOGS	Sensory features	Enjoyment	$F = 7.69$	<.005	Loba et al. (2001)
	29PG//31 n-PG	SOGS	Sensory features	Tens-reduction	$F = 6.95$	<.005	Loba et al. (2001)
	29 PG/31 n-PG	SOGS	Sensory features	Easy to stop	$F = 4.62$	<.05	Loba et al. (2001)
	29 Pg/31 n-PG	SOGS	Sensory features	Desire to play	$F = 4.86$	<.01	Loba et al. (2001)
	29 PG/31n-PG	SOGS	Sensory features	Notice difference	$F = 19.68$	<.001	Loba et al. (2001)
	n-PG SOGS		Sensory features	Bothered	$F = 11.29$	<.001	Loba et al. (2001)
4.3 Schedules of reinforcement							
	Operant model	RF schedule	Intermittent RF	–	–	(see text)	Skinner (1953)
3.4 Age of onset							
	2526 calls	SOGS	Onset playing	Before 21 years	$X^2 = 10.17$	.01	Bondolfi et al. (2000)
	45 normals	SOGS-R	Gambl age	Early	$F = 52.57$	.000	Volberg et al. (2001)
4.5 Rapid onset							
	44 consec PGs	SOGS/DSM-IV	Latency (years)	Shorter latency	–	–	Breen and Zimmerman (2002)
	44 consec PGs	SOGS/DSM-IV	Latency (years)	Main effect	$F = 8.42$	<.01	Breen and Zimmerman (2002)
	44 consec PGs	SOGS/DSM-IV	Latency (years)	Longer latency	$t = 2.90$	<.01	Breen and Zimmerman (2002)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	*p* value	Study
5. Comorbidity and concurrent symptoms							
5.1 Depression							
	60 (30 GA members/ 30c)	SOGS	GA	Increase	$F = 17.43$	<.001	Getty et al. (2000)
	60 (30 GA members / 30c)	SOGS	Female	Increase	=8.16	=.006	Getty et al. (2000)
	69 pat	BDI	Comorbid dis	Increase	$t = -3.4$	=.0001	Ibanez et al. (2001)WTwTwTT
	Twin study	DIS	Comorbid depr	Increase	OR = 4.12	–	Potenza et al. (2005)
	3426 stud	SOGS	Suic. attempts	Increase	$X^2 = 60.26$	<.001	Ladouceur et al. (1999a)
	562 calls	Questionnaire	Suic. attempts	Increase	$X^2 = 4.19$	<.05	Potenza et al. (2001)
5.3 Anxiety							
	69 pat	STAI trait	Concurrsy	Increase	$t = 2.0$	=.05	Ibanez et al. (2001)
		STAI state			$t = 3.0$	=.004	
5.4 OCD							
	89(41M/48F)	SOGS/YBOCS	Obessions	Increase	$t = 3.45$	<.001	Frost et al. (2001)
	89(41M/48F)	SOGS/YBOCS/	Compulsions	Increase	$t = 2.77$	<.01	Frost et al. (2001)
	89 (36PG)	SOGS	Hoarding	More	$t = 2.71$	<.01	Frost et al. (2001)
	89 (36PG)	SOGS	Urge to play	More	$t = 36.16$	<.001	Frost et al. (2001)
	89 (36PG)	SOGS	Avoidance	More	$t = 4.30$	<.001	Frost et al. (2001)
	89 (36PG)	SOGS	Impulsivity/IES	More	$t = 3.66$	<.005	Frost et al. (2001)
5.5 Alcohol							
	220 MMTP	SOGS	Alcoholism	More	$r_{xy} = .14$	.02	Feigelman et al. (1995)
	220 MMTP	SOGS	Use of alc last month	More	$r_{xy} = .14$	.02	Feigelman et al. (1995)
	3426 stud	SOGS	Alcohol	Increase	$F = 24.71$	<.0001	Ladouceur et al. (1999a)
	Twin study	DIS	Alcohol	Increase	–	–	Slutske et al. (2000)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	p value	Study
5.6 Other drugs							
Heroin	220 MMTP	SOGS	Daily use	More	$r_{xy} = .14$	.02	Feigelman et al. (1995)
Drugs	702 (15-18)	SOGS-RA	Drugs	Increase	$X^2 = 46.2$	<.001	Winters et al. (1993)
Drugs	3426 stud	SOGS	Drugs	Increase	$F = 29.09$	<.0001	Ladouceur et al. (1999a)
Drugs	562 calls	Questionnaire	Drugs	Increase	$X^2 = 5.66$	<.02	Potenza et al. (2001)
Drug problem	220 MMTP	SOGS	Drug problem last year	More	$r_{xy} = .12$	.04	Feigelman et al. (1995)
Cigarettes	3426 stud	SOGS	Cigarettes	Increase	$F = 20.42$	<.0001	Ladouceur et al. (1999a)
5.7 Personality disorders							
	7,869M(twinp)	DSM-III-R/DIS	ASPD	Increase	OR = 6.4		Slutske et al. (2001)
	7,869M(twinp)	DSM III-R/DIS	CD	Increase	OR = 3.6		Slutske et al. (2001)
	7,869M(twinp)	DSM-III-R/DIS	AAB	Increase	OR = 6.1		Slutske et al. (2001)
	69 pat	DSM-III-R	Concurrsy	Increase	$t = -3.0$	=.004	Ibanez et al. (2001)
	69 pat	DSM-III-R	Comorbid dis	Increase	$r_{xy} = .40$	<.001	Ibanez et al. (2001)
6. Personality symptoms and characteristics							
6.1 Coping styles							
	30GAexp/30c	SOGS	Main eff gr var	Coping styles	$F = 8.35$	<.001	Getty et al. (2000)
	30GAexp/30c	SOGS	Suppressive	High	$F = 13.81$	<.001	Getty et al. (2000)
	30GAexp/30c	SOGS	Reactive	High	$F = 16.22$	<.001	Getty et al. (2000)
	30GAexp/30c	SOGS	Reflect cop style	Low	$F = 7.81$	<.007	Getty et al. (2009)
6.2 Impulsivity							
	754 adol boys	SOGS/EIS/TRIS	Impulsivity	Increase EIS	$X^2 = 30.58$	<.01	Vitaro et al. (1997)
	754 adol boys	SOGS/EIS/TRIS	Impulsivity	Increase TRIS	$X^2 = 27.95$	<.01	Vitaro et al. (1997)

Table 1 continued

Domain	Sample	Instrument	Risk factor	Increased risk	Statistics	p value	Study
6.4 Sensation seeking							
	27 PG/ 27 match	SSS	Thrill	Increase	WMP = −76	<.03	Blanco et al. (1996)
	27 PG/ 27 match	SSS	Disinhib	Increase	WMP = −88	<.01	Blanco et al. (1996)
6.5 Delinquency, criminality and illegal activity							
	702 (15–18)	SOGS-RA	Delinquency	Increase	$X^2 = 47.3$	<.001	Winters et al. (1993a, b)
	220 MMTP	SOGS	Delinquency	More	$r_{xy} = .25$	.001	Feigelman et al. (1995)
	220 MMTP	SOGS	Criminality	More	$r_{xy} = .16$	.01	Feigelman et al. (1995)
	3426 stud	SOGS/SRDS	Delinquency	Increase	$F = 176.18$	<0001	Ladouceur et al. (1999a)
	562 calls	Questionnaire	Delinquency	Increase	$X^2 = 9.53$	<.002	Potenza et al. (2001)
	313	DSM-III-R	Incarceration	Increase	$t = 16.53$	<.001	Hall et al. (2000) see ref
	313	DSM-III-R	Illegal activity	Increase	$t = 7.83$	.02	Hall et al. (2000)

Note: Some references to weak/inconclusive studies are not included in table, only in text

PG = Pathological Gambling

SOGS = South Oaks Gambling Screen

DIS = Diagnostic Interview Schedule for Pathological Gambling

DSM = Diagnostic and Statistical Manual of Mental Disorders

SSS = Sensation Seeking Scale

r_{xy} = correlation, t-test, OR = odds ratio, FA = Anova, FR = Regression

Table 2 Risk factors for PG—summary of results

Domain	Risk factor	No. studies	Level[a]	
1. Demographics				
1.1 Age	Low age	4	1	
1.2 Gender	Male	4	1	
1.3 Education	Low education	1		3
1.4 Marital status	Married/unmarried	2		
1.5 Income	Low income	2		
1.6 Employment	Unemployed	2		2
1.7 Social welfare status	On social welfare	1		2
1.8 Residence	Large city	1		2
1.9 Acad achievement	Low achievement	2		2
1.10 Immigrant, ethnic grp	Immigrant, foreign	3		2
2. Physiological/biological fact				
2.1 Heart rate and arousal	Increased during play	2		2
2.2 Transmittor activity	Increased	3		2
2.3 Genetic studies	DA rec increased	4		2
3. Cognitive distortions				
3.1 Erroneous perceptions	Bias	4		2
3.2 Illusion of control	Illusion	9	1	
4. Varia				
4.1 Availability of plays	High	3	1	
4.2 Sensory characteristics	Yes	7	1	
4.3 Schedules of reinforcement	Type	–	1	
4.4 Age of onset	Early	2		2
4.6 Rapid onset	Short latency	1		2
5. Comorbidity & concurrent symptoms				
5.1 Depression	Yes	3		2
5.3 Anxiety	Yes	1		2
5.4 OCD	Yes	6	1	
5.5 Alcohol	Yes	3	1	
5.6 Other drugs	Yes	5	1	
5.7 Personality disorders	Yes	2		2
6. Personality symptoms and characteristics				
6.1 Coping styles	Low	1		2
6.2 Impulsivity	High	1		2
6.3 Hyperactivity (ADHD)	0	0		
6.4 Sensation seeking	High	2		2
6.5 Delinquency & illegal acts	Yes	5	1	

[a] (1) well-established risk factor for PG (>2 studies); (2) probable risk factor for PG (1–2 studies); (3) not a risk factor. There are also some weaker studies mentioned in the text but not in Table 1

Gender

In a study conducted by Winters et al. (1993b), subjects aged 15–18 years ($n = 702$) were interviewed using the SOGS-RA (Winters et al. 1993a) on a targeted telephone list

(not a random digit-dial procedure). A comparison of answers on item #1 of SOGS-RA, showed that boys had higher gambling activity than girls ($t = 6.46$, $p < .001$). The researchers compared higher degrees of problem gambling (pathological gambling and at risk gambling) to different variables. There was no comparison made between gender and problematic gambling and therefore the reference is not included in the table on gender.

In a study by Ladouceur et al. (1999a) of 3426 high school students, a univariate ANOVA on SOGS scores and male gender showed a statistically significant main effect ($X^2 = 39.52$, $p < .001$).

Feigelman et al. (1995) investigated pathological gambling in 220 methadone patients in two methadone maintenance treatment programs (MMTP), finding a high rate of gambling problems in this treatment population. Pathological and problem gambling was assessed using SOGS and, based on earlier results, other important factors were assessed. A significant relationship between problem gambling and male gender was found ($r_{xy} = 0.12$, $p = .04$).

Male gender was also shown to be a risk factor ($X^2 = 8.94$, $p = .01$) in a telephone interview prevalence study of gambling conducted by Bondolfi et al. (2000) in Italy. This was also the case in the Volberg et al. (2001) study where male gender was again shown to be a risk factor for gambling problems (OR = 3.71, $p = .000$).

In four of the five gender studies where gender has been evaluated in relation to problem gambling, clear support for the notion that male gender is a significant risk factor for PG has been demonstrated. There are indications (for instance in Wardman et al. 2001) that females are at higher risk than men in aboriginals but this finding has not been replicated.

Education

In the Volberg et al. (2001) study, education level did not significantly predict risk for gambling problems. Further study on how education levels impact PG is needed.

Marital Status

In the study by Bondolfi et al. (2000), marital status (married) was shown to be a risk factor ($X^2 = 7.52$, $p = .02$). Conversely, the Volberg et al. (2001) study showed that being single was a risk factor for gambling problems ($X^2 = 121.67$, $p = .000$).

Only two studies have been directed towards marital status empirically, and with contradicting results, conclusions are not possible at this time.

Income

In the study by Bondolfi et al. (2000), higher income was shown to be a risk factor ($X^2 = 10.88$, $p = .01$) for gambling problems. The helpline study by Potenza et al. (2001) reported financial problems as a significant risk factor ($X^2 = 4.21$, $p < .04$).

Only two studies have examined income and financial problems empirically, and they have produced contradictory results.

Employment

In the study by Feigelman et al. (1995), there was a significant relationship between unemployment status within the last year and problem gambling ($r_{xy} = -0.15$, $p = .02$).

Hall et al. (2000) studied pathological gambling among 313 cocaine-dependent outpatients. They used the DIS (Diagnostic Interview Schedule) to reach a DSM-III-R diagnosis of PG, and the ASI (Addiction Severety Index) to obtain sociodemographic and other information. Of the 313 patients, 25 (8.0%) fulfilled full DSM-III-R criteria for PG. There was a significant relationship between unemployment status and PG ($t = 11.09$, $p < .001$).

Only two studies have been directed towards employment empirically and thus, we consider employment status as a probable risk factor for PG.

Social Welfare Status

In the Volberg et al. (2001) study, being on social welfare was shown to be a significant risk factor for gambling problems ($z = 2.41$, $p = .05$).

Only one study has been directed towards social welfare status empirically and thus, we consider social welfare status as a probable risk factor for PG.

Residence

In the Volberg et al. (2001) study, living in a large city was shown to be a risk factor for gambling problems ($z = 4.00$, $p < .01$).

Only one study has been directed towards residence empirically, thus, we consider residence as a probable risk factor for PG.

Academic Achievement

In the study by Ladouceur et al. (1999a), an ANOVA performed on a five question scale for academic achievement and SOGS scores showed a significant main effect ($F = 19.44$, $p < .001$).

In the study by Winters et al. (1993b), average-to-below average school grades were related to problematic gambling ($X^2 = 21.7$, $p < .001$).

Only two studies have been directed towards academic achievement empirically. We consider academic achievement as a probable risk factor for PG.

Immigrants and Ethnic Groups

In the helpline study by Potenza et al. (2001), African-American ethnicity was identified as a significant risk factor ($X^2 = 3.87$, $p < .05$). In the Welte et al. study (2004), being African-American, Hispanic, or Asian were all risk factors for problematic gambling (IRR 1.96–4.71; $p < .01$). In the Volberg et al. (2001) study, being born outside the country was shown to be a risk factor for gambling problems (OR = 2.08, $p = .01$).

Because three studies have been directed towards immigrants and ethnic groups empirically, (but only two have focused on ethnicity), we consider immigration and ethnic groups as probable risk factors for PG.

Physiological and Biological Factors

Heart Rate and Arousal

One study used a laboratory setting with an artificial casino and compared it to a real casino situation. A group of 12 experienced gamblers showed significantly higher heart rate (HR) increases ($p < .0001$) in the real casino condition. The correlation between the amount of money wagered and HR increase was significant ($r_{xy} = .741$, $p < .0005$, one-tailed). The other group consisted of students ($n = 12$) who did not differ from the experienced gamblers in their reactions to the artificial casino (Anderson and Brown 1984).

A second empirical study was performed by Leary and Dickerson (1985) who followed high- and low ($n = 22/22$) frequency players by assessing heart rate during playing. Playing was significantly associated with increases in arousal in both groups but was significantly more so by high-frequency players ($p < .05$).

Cocco et al. (1995) hypothesized that poker machine gamblers and horse race gamblers should differ in their state of arousal. Of the 12 problem poker machine players and 13 horse race gamblers assessed, the researchers were able to show that poker machine gamblers showed higher arousal avoidance and higher trait anxiety as compared to horse race gamblers (both $p < .05$). No attempt was made to predict pathological gambling.

Only two studies have been directed towards heart rate and arousal empirically. Consequently, we consider heart rate and arousal as a probable risk factor for PG.

Griffiths (1995) reviewed the literature on this field (see Griffiths 1995, Table 1.4, p. 18) and it seems the correlation of increased heart rate to increased playing among gamblers compared to non-gamblers is weak. Empirical support for such a notion seems too weak to justify further reporting.

Transmitter Activity

Bergh et al. (1997) studied monoamines and their metabolites in cerebrospinal fluid (CSF) from 10 PGs and 7 controls. There was a significant difference between the two groups (unpaired t tests): the experimental group showing a decrease in dopamine (DA) and an increase in 3,4–dihydroxyphenanylacetic acid (DOPAC) as well as in homovanillic acid (HVA). The ratio DOPAC/DA and HVA/DA was significantly different as well. Noradrenaline (NA) and its metabolite MHPG were increased, whereas 5-HT and 5-HIAA were unchanged.

Roy et al. (1988) investigated CSF levels of 3–methoxy-4-hydroxyphenolglycol (MHPG) and urinary outputs of noradrenaline (NA), in a group of PG ($n = 24$), and compared them to controls ($n = 20$). They showed that gamblers had significantly lower plasma MHPG levels than controls ($t = 2.9$, $p < .007$), and significantly greater urinary outputs of NA ($F = 11.6$, $p < .0003$). The results are discussed in a theoretical framework.

Blanco et al. (1996) studied platelet MAO activity on 27 male PGs compared to matched controls using the Jackman's procedure. They found that MAO activity was lower in the PGs than in controls ($p < .01$, Wilcoxon matched-pairs signed rank test).

Three studies have been performed to examine transmitter activity in relation to PG. Several transmitters have been studied and due to the complex interactions between these neurotransmitters, the results are difficult to interpret.

Genetic Studies

The dopamine D_2A1 allele has been connected to substance abuse. Comings et al. (1996) studied the presence of the dopamine (DA) D_2 receptor in a PG sample without drug addiction, and a control sample. The group with PG they showed a significantly different occurrence of D_2A1 allele (OR = 5.03).

Ibáñez et al. (2001) studied 69 consecutive PGs applying for treatment for their gambling. They used the SOGS to diagnosis PG as well as the full DSM-IV (APA 2000) clinical interview (both Axis I and II). They assessed allele distribution of the dopamine receptor gene (DRD_2) polymorphism. The results showed that DRD_2 polymorphism was different in gamblers with and without psychiatric comorbidity ($X^2 = 13.9$, $p = .003$) and the allele DRD_2 C4 allele was present in 42% with comorbid psychiatric disorders compared to 5% without ($X^2 = 7.0$, $p = .008$).

Other studies assessed D_2, D_4 and D_1 as well as serotonin and norepinephrine genes as possible risk factors for PG (Comings et al. 2001). A family study also reported that the D_1 receptor gene is associated with PG (da Silva Lobo et al. 2007). We consider genetic studies as probable instruments for assessment of PG risk factors.

Cognitive Distortions

The literature reports a range of gambling-related cognitive psychopathology (Toneatto 1999). For example, magnification of gambling skill results in the gambler having an exaggerated self-confidence and ignoring the severity of losses. Superstitious beliefs are characterized by the thought that the gambler has a reliable means of manipulating outcome in his or her favour. Subcategories are talismanic superstitions that a certain object increases winning probability, behavioural superstitions that some rituals can increase winning, and cognitive superstitions that certain mental states can influence winning. The gambler's fallacy means that a series of losses is expected to be compensated for by chasing and therefore becomes the means by which the gambler recovers losses.

Erroneous Perception or Biased Evaluations

Gilovich (1983) investigated factors leading to continued gambling behavior in spite of loosing more than winning. In one experiment he investigated how much time the subjects (29 students) used in their explanations for losses versus wins. It appeared that they used more time to discuss losses than for wins ($t = 2.33$, $p < .05$). In a second experiment, 64 students reported that their memories from the previous week correctly reported 47% of the losses but only 27% of wins ($t = 2.33$, $p < .05$).

Savoie and Ladouceur (1995) studied erroneous perceptions in two studies. The question examined was if it is possible to modify the erroneous perceptions through exact information about the probability of negative gains on gambling. The study aimed to revise

the erroneous concepts among the participants and to eventually modify their playing customs.

Savoie and Ladouceur's (1995) first study involved 100 subjects regularly participating in a lottery (53M/47F) and 100 (42M/58F) participating only occasionally. A short interview asked questions regarding their superstitious habits and preferences about lottery, choosing numbers, etc. The probability of winning was estimated as higher amongst the regular players than the occasional players ($X^2 = 6.94$, $p < .01$). The experimental group differed from the control group in that they believed that their strategies of choosing their numbers increased their chances to win ($X^2 = 13.66$, $p < .01$).

In their second study, 44 regular lottery players were asked about playing habits, concepts about lotteries, participation frequency, strategies involved in choosing numbers, degree of confidence, irregular preferences, and gains from the lottery. Participants were randomly assigned to experimental and control groups ($n = 22/22$). They were asked to compare the preciseness of their concepts with the actual result probabilities. A month later, they received the same questionnaire again. The results showed that the regular players had more erroneous perceptions than the control. The experimental group had become less confident to win ($F = 7.38$, $p < .025$), while the control group had reduced their playing activity during the time ($t = 3.2$, $p < .025$).

Illusion of Control

"Illusion of control" means an expectancy of a personal success probability inappropriately higher than the objective probability would warrant. It is based on the idea that factors from skill situations introduced into chance situations cause individuals to feel inappropriately confident. A series of six experiments examined the effects of competition, choice, familiarity, and active involvement on illusion of control (Langer 1975). In these experiments, a variety of factors were manipulated: bias (confidence/shyness) ($t = 5.46$, $p < .005$); choice ($t = 4.33$ $p < .005$); familiarity ($t = 5.46$, $p < .005$); involvement ($F = 7.33$, $p < .01$). Subjects did not distinguish chance from skill-determined events. The subjects acted as if they could control outcomes and they gave up the opportunity to exert real control.

In a second study, Langer and Roth (1975) performed an experiment to examine the development of illusion of control. They investigated attributions in a purely chance task (predicting coin tosses), where the task had a descending, ascending, or random sequence of outcomes. Early successes did induce a skill orientation towards the task ($F = 4.20$, $<.05$). The subjects with a descending condition rated themselves as significantly better at predicting outcomes than the other two conditions. They also selectively remembered past successes and expected more future successes.

Gilovich and Douglas (1986) also studied the development of illusion of control. They showed that evaluations of randomly determined gambling outcomes were biased ($F = 10.72$, $p < .001$). The losers appeared to use manipulated fluke events to explain away the outcome, whereas winners discounted their significance. In a second experiment, the outcomes were shown to be biased towards randomly determined gambling outcomes ($t = 2.56$, $p < .02$). Subjects were induced to perceive an "illusion of control" and the outcome affected those who had lost the first bet but had no effect on those who had won. In the no-control condition, the responses were more symmetric to the fluke manipulations by winners and losers.

There is strong evidence that illusion of control is a phenomenon that could interact with the development of PG. Nine experiments have been performed on normal subjects (students and company employers).

Varia

Availability of Plays

Countries with high level of gambling availability have among the highest prevalence rates of pathological gambling. Availability of gambling is correlated with prevalence of pathological gambling (Campell and Lester 1999; Walker 1992).

Ladouceur et al. (1999b) directly tested the effect of increased availability of gambling activities and the rate of pathological gambling in the community by conducting two prevalence studies separated by a 7-year period. The second study conducted after more gambling venues were available showed a 75% increase in the number of pathological gamblers.

Sensory Characteristics

Loba et al. (2001) studied the effect of sensory manipulations (fast speed/sound, slow speed/no sound, counter present), to examine subjective self-reported differences in reaction amongst gamblers. They used video lottery terminals (VLT), a "continuous" form of gambling, where time between wager and payout is short. Subjects included 60 (22F/38M) regular (playing at least twice a month) VLT players, recruited via advertisements. They used the SOGS to assess PG severity and used a survey of subjective reactions to VLT manipulations. The experimental condition was either a video poker game or a 20 min spinning reels game. The game versions had varied sensory characteristics (i.e. slow/no sound, fast/sound, control, counter present). The results showed that non-PG subjects were bothered by fast speed and sound, while PG subjects were bothered by slow speed and no sound. There was a significant main effect of sensory features ($F = 11.29$, $p < .001$) and a sensory feature by game interaction ($F = 5.50$, $p < .01$). The sensory features gave significant results for the following subjective variables: excitement ($F = 9.85$, $p < .001$), enjoyment ($F = 7.69$, $p < .005$), tension-reduction ($F = 6.95$, $p < .005$), easy to stop ($F = 4.62$, $p < .05$), desire to play again ($F = 4.86$, $p < .01$), notice difference ($F = 19.68$, $p < .001$), and bothered ($F = 11.29$, $p < .001$). Decreased speed and turning off the sound decreased ratings of enjoyment, excitement, and tension reduction for PG subjects compared to non-PG subjects. The study supports Griffiths (1993) notion that sensory characteristics are important in the development of PG.

This study (Loba et al. 2001) presented very robust data on seven important subjective variables. The tentative conclusion is that several sensory characteristics may play an important role in the development of PG.

Schedules of Reinforcement

In some early work, Skinner (1953, 1969) defined the schedule of reinforcement as a simple temporal order of response and consequence. The consequence, if positive, could

work as a reward for the behaviour or response emitted. If negative, it could have a punishing effect. A continuous reinforcement (reward in each trial) is easy to manipulate. Withdrawing the reinforcer will cause the behaviour to eventually cease (extinguish). An intermittent reinforcement/reward, however, is more resistant against extinction. A fixed ratio (FR10, every tenth occasion), or a variable interval (VI 1 hr, every hour), has a stronger effect, and is less amenable to extinction. A random reinforcement is the strongest conditioning, which is hardest to extinguish. Therefore, the payout interval, in games of chance may be important in the development of PG.

Early wins may induce a skill orientation (Gilovitch 1983) and reinforce the need to try again, even if the contingency schedule is rather intermittent. In the same manner, a big win in gambling can also be analyzed with the operant model. The concept of the near miss has been assessed by Griffiths (1991) and is also in accordance with the operant principles.

Skinner himself did not rely on statistical probability testing, rather he designed straightforward models, where the effect was easily visible and beyond doubt. Therefore, it is unsuitable to add statistical tests to support the importance of the operant conditioning model. The results reported are clearly clinically significant.

There is a vast body of research illustrating the usefulness and importance of the operant model. This model can improve the understanding of the mechanisms of development of PG.

Age of Onset

In a study by Bondolfi et al. (2000), age of onset (before age 21) was shown to be a significant risk factor for PG ($X^2 = 10.17$, $p = .01$).

In the Volberg et al. (2001) study, age of onset was shown to be a risk factor for gambling problems (19.9 years for non-gambling problems and 15.6 years for problem gambling) ($F = 52.57$, $p = .000$).

Only two studies examined age of onset, and therefore, we consider age of onset as a probable PG risk factor.

Rapid Onset

Breen and Zimmerman (2002) studied the latency of PG-onset (from age of regular involvement, to PG criteria; in years) in 44 consecutive PG subjects. The primary form of gambling at PG-onset was the only variable retained in a stepwise multiple regression analysis ($F = 8.42$, $p < .01$). The traditional gamblers had a longer latency period than machine gamblers (3.58 vs 1.08 years; $t = 2.90$, $p < .01$).

Only one study has been performed on rapid onset, thus, we consider rapid onset as a probable PG risk factor.

Comorbidity and Concurrent Symptoms

Depression

In a study by Getty et al. (2000), a difference in depression between experimental (mean 17.73) and control (mean 8.80) groups ($F = 17.43$, $p < .001$) was noted. In the study by Ibáñez et al. (2001), higher rates of depression, as examined by the Beck Depression

Inventory (Beck and Steer 1993), were found in the more serious pathological gamblers ($t = 3.4$, $p = .0001$).

A study by Potenza et al. (2005) found that in males with PG, 34% of the genetic variance for major depressive disorder (MDD) contributed to PG and vice versa.

With three studies performed on depression, we consider depression as a probable PG risk factor.

Anxiety

In the study by Ibáñez et al. (2001), trait anxiety (STAIT) was significantly higher amongst the more severe pathological gamblers ($t = 2.0$, =.05).

With only one study performed on anxiety, we consider anxiety as a probable PG risk factor.

OCD

Frost et al. (2001) studied the relationship between OCD symptoms and PG in 89 subjects (48F/41M). PG was diagnosed through use of the SOGS and OCD symptoms through the YBOCS (Yale-Brown Obsessive Compulsive Scale) (Goodman et al. 1989a, b) as well as a hoarding scale. Thirty-six participants met criteria for PG. The results showed higher intensity of symptoms for the PG group than for the rest, particularly for obsessions ($t = 3.45$, $p < .001$), compulsions ($t = 2.77$, $p < .01$), hoarding ($t = 2.71$, $p < .01$), urge to gamble ($t = 36–16$, $p < .001$), avoidance ($t = 4.30$, $p < .001$), and impulsivity (IES; $t = 3.66$, $p < .005$).

With six areas covered (by one paper) on the relationship of OCD to PG, we consider OCD as a probable risk factor for PG. This connection also supports the close relationship between obsessions and obsessive gambling.

Alcohol Abuse

In the study by Feigelman et al. (1995), both a lifetime alcohol problem ($r_{xy} = 0.14$, $p = .02$) and use of alcohol within the last month ($r_{xy} = 0.14$, $p = .02$) were significantly related to problem gambling.

In the Ladouceur et al. (1999a) study, statistical analyses showed a relationship between SOGS scores and alcohol use ($F = 24.71$, $p < .0001$).

In a twin study of genetics, between 12% and 20% of the genetic variation in the risk for PG was accounted for by the risk for alcohol dependence (Slutske et al. 2000).

We consider alcoholism as a probable PG risk factor.

Other Drugs

In the study by Feigelman et al. (1995), there was a significant relationship between problem gambling, having a major drug problem within the last year ($r_{xy} = 0.12$, $p = .04$), and frequency of heroin use ($r_{xy} = 0.14$, $p = .02$).

The study mentioned above (Ladouceur et al. 1999a) also showed a relationship between SOGS scores and cigarette smoking ($F = 20.42$, $p < .0001$). In the study by Winters et al. (1993b), frequent drug use was significantly connected to more problem gambling ($X^2 = 46.2, p < .001$). The study mentioned above (Ladouceur et al. 1999a) also showed a relation between SOGS and drug use ($F = 29.09, p < .0001$). The helpline study by Potenza et al. (2001) reported that drug use was identified as a significant risk factor ($X^2 = 5.66$, $p < .02$).

Because the five studies were examining different drugs, we consider drug abuse a probable PG risk factor.

Personality Disorders

In a study by Slutske et al. (2001), 7,869 men from 4,497 twin pairs (Vietnam Era Twin Registry) were diagnosed with PG using the DSM-III-R (APA 1987), with whilst using the DIS for antisocial behavior disorders. Telephone interviews were conducted to ascertain prevalence rates of PG and antisocial behavior disorders. The results showed elevated prevalence rates of antisocial personality disorder amongst individuals with a history of PG (OR = 6.4).

In a study by Ibáñez et al. (2001), there were more personality disorders in the more severe cases of PG subjects ($t = 3.0$, $p = .004$).

With only two studies performed on personality disorders, we consider personality disorder as a probable PG risk factor.

Personality Symptoms and Characteristics

Coping Styles

Getty et al. (2000) studied a group of members of Gamblers Anonymous ($n = 30$) compared to a matched control group ($n = 30$). The PG diagnosis was made using the SOGS. The study used the Problem-Focused Styles of Coping Inventory (PF-SOC). All types of coping styles, suppressive, reactive and reflective were significantly different between the experimental and control group with the experimental group being higher on suppressive ($F = 13.81$, $p < .001$) and reactive ($F = 16.22$, $p < .001$) while lower on reflective coping styles ($F = 7.81$, $p < .007$).

With only one study performed on maladaptive coping, we consider maladaptive coping as a probable PG risk factor.

Impulsivity

Vitaro et al. (1997) studied impulsivity among 754 adolescent boys using the Eysenck Impulsiveness Scale (EIS). PG severity was assessed with the SOGS. There was a clear relationship between greater PG severity and high rates of impulsivity ($X^2 = 30.58$, $p < .01$).

With only one study performed on impulsivity, we consider impulsivity as a probable PG risk factor.

Hyperactivity (ADHD)

Carlton and Manowicz (1994) found that adult pathological gamblers had a higher than average ate of childhood attention deficit hyperactivity disorder (ADHD). In a retrospective study and subsequent EEG assessments of pathological gamblers, they found that gamblers had patterns of activation similar to children with ADHD. The study does not include significance evaluations and is therefore not mentioned in the table.

Sensation Seeking

Blanco et al. (1996) investigated 27 PG and 27 matched control subjects found significant differences between the two groups (WMP = Wilcoxon Mathed Pairs) on the Sensation Seeking Scales (subscale Thrill and Adventure seeking, both $p < .03$; and Disinhibition, $p < .01$).

With only one study performed on sensation seeking, we consider sensation seeking as a probable PG risk factor.

Delinquency, Criminal and Illegal Activity

In the study by Winters et al. (1993b), delinquency status, illegal activity, or arrest were related to problem gambling ($X^2 = 47.3$, $p < .001$).

In the study by Feigelman et al. (1995), there was a significant relationship between problem gambling and criminality in general ($r_{xy} = 0.25$, $p = .001$) as well as in number of arrests for criminal offenses ($r_{xy} = 0.16$, $p = .01$).

The study conducted by Ladouceur et al. (1999a) also showed a relationship between SOGS scores and delinquency as measured by the Self-Reported Delinquency Scale ($F = 176.18$, $p < .0001$).

In the helpline study by Potenza et al. (2001), delinquency was identified as a significant risk factor ($X^2 = 9.53$, $p < .002$).

In the study by Hall et al. (2000), there was a clear relation between PG and duration of incarceration ($t = 16.53$, $p < .001$) and illegal activity ($t = 7.83$, $p = .02$).

With five studies performed on delinquency/illegal activity, we consider maladaptive delinquency/illegal activity as a well-established PG risk factor.

Discussion

The aim of the present study was to identify the existing empirical studies regarding the risk factors for the development and maintenance of pathological gambling. This is especially important for the implementation of prevention and treatment for PG. A study by Volberg et al. (1996) has illustrated the importance of obtaining such knowledge in order to enable, correct, and implement effective interventions.

We reviewed studies with sound experimental designs. Although both epidemiological and treatment studies could contribute to this knowledge, we choose not to include them for two reasons: (1) there are already several reviews published on epidemiological and treatment studies; and (2) the review task would be too heavy to take in one round.

The critical review of 35 different factors or dimensions unfolded very few "well-established risk factors for PG" (9 factors), where the conclusions could be supported by more than two studies. These were demographic variables (age, gender), cognitive distortions (erroneous perceptions, illusion of control), sensory characteristics, schedules of reinforcement, and comorbid states (OCD, drug abuse), and delinquency/illegal acts. Many categories (22) are considered to constitute probable risk factors (1–2 supporting studies).

The relationship between PG and some probable factors of importance (among them physiological and biological factors) are rather unclear at this time.

The different groups show some documentation for all groups of variables (see Table 2) as implicated in the development of PG, although not enough to give clear support for more than nine variables. A strong group of variables consists of demographics, where age and gender are clear risk factors in a number of studies.

Physiological and genetic studies give indications that these types of variables are related to PG in an important way. Detailed interpretation of the results in is difficult at this time. However, a variety of neurotransmitters may still be probable markers for PG risk factors.

The cognitive distortions studied amongst others by Gilovitch (1983) and Langer (1975) show a clear relationship between cognitive factors and PG, at least in the analogue setting. Only a few studies (i.e., Savoie and Ladouceur 1995), however, examined these variables in the natural environment of real gambling.

The main conclusion from the present critical review is, that very little is known with regard to risk factors for the development and maintenance of PG. This is the case both for the existence of risk factors, their strength and importance, as well as their mechanisms of action.

Consequently, there is a great need for empirical research on the development and maintenance of PG. This research should be in concert with epidemiological and prospective longitudinal studies to elucidate the prevalence (and change) of PG in normal populations, in risk populations, and in individuals with PG. Further actions should be directed towards interventions (prevention, treatment), and experimental studies should be directed towards mechanisms of action of risk factors.

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