General Risk Factors for Gambling Problems and the Prevalence of Pathological Gambling in Norway

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Summary


Pathological Gambling (PG) has not until recently been scientifically studied. In a series of epidemiological studies prevalences of Pathological gambling were assessed for both men and women, adults and adolescents, and also for games without monetary rewards, and for internet. In Norway the condition affects below one percent of the adult population (higher in men than in women), close to 2% percent among adolescents, and close to 3% for games without monetary reward. For “internet addiction” (as defined in paper III) the prevalence is about 2%. In addition comes playing via the internet, which occurs in quite high figures, and with indications that this problem is rapidly increasing throughout the world.

There are several attempts to build a theory that can explain both the etiology of gambling, its development and alleviation. There are few controlled studies of the effect of treatment of pathological gambling. In an attempt to increase the understanding of pathological gambling, a critical literature review was performed of the risk factors for developing pathological gambling. The results showed that there are a few empirically documented risk factors established. We could show that quite few of our thirty-five studied risk factors have been empirically documented to enable valid conclusions: Gender, cognitive distortions, availability, sensory characteristics, schedules of reinforcement and psychiatric comorbidity, personality deviations.
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List of papers

The present thesis is based on the following papers (cited with their Roman numerals):


IV Agneta Johansson, K Gunnar Götestam: Problems with computer games without monetary reward: similarity to pathological gambling. Psychological Reports, 95, 641-650, 2004b

1. INTRODUCTION

Although “gambling plays”, and “plays of chance” have been well-known concepts for at least hundred years, gambling as a psychiatric problem has not been acknowledged until recently. In the major textbook by Mayer-Gross, Slater and Roth (1960, first mentioned in edition 1954) gambling as a psychiatric problem is not mentioned. The same is the case with the Scandinavian textbook by Strømgren (1954). Later on, gambling has been considered to be a problem, mainly in relation to horse betting. A merely compulsive gambling behaviour has been described as a specific psychiatric problem since the 70’s (Strømgren, 1974). In a study quantifying citations during the period 1964-1999, the first reference found was printed in 1964, first followed by a low level and then by a dramatic increase from 1984 (Eber & Shaffer, 2000).

A more extensive review of the field is made in the paper on risk factors (V).

With the new diagnostic practice (DSM, ICD) the severe and pathological forms of gambling have been categorized as an impulsive behaviour deviation, called Pathological Gambling (PG, coded as number 312.31 in DSM-IV). The impulsive character of the gambling traits is evident in the last version of DSM (1994). The diagnostic criteria for pathological gambling are indicated below (section 2.2.2).

Categorized under the same heading Impulse-Control Disorders, are also kleptomania, pyromania, and trichotillomania (compulsive hair pulling).

Different forms of the substance-induced disorders and dependence, include intoxication, withdrawal, dependence, and abuse, and with the drug groups of alcohol, amphetamines, cannabis, cocaine, hallucinogens, inhalants, nicotine, and opioids.

The PG and substance dependence have several similar characteristics as withdrawal (restlessness, “craving”, other physiological or psychological symptoms), increasing amount and time, unsuccessful efforts to cut down, important activities given up,
continuation despite full understanding of the severe problem. They often also cover their problem behaviour for family and friends. Pathological gambling has been called the only “true” addictive disorder, as it is an addiction with no substance implicated. It has been mentioned as a potential model for studying the mechanisms of the development and treatment of gambling.

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 study is not a systematic categorizing review, it goes through a couple of studies, and they show some new angles of the interface between PG and substance misuse.

It is difficult to get an overview of the problems with pathological gambling, but such an overview is of great importance when it comes to prevention and intervention with those who have problems with gambling. That is the main reason why we were interested in establishing the general level of the problem in the community (a more in depth literature survey is made in paper V). Most of the few studies performed at that time, were with the SOGS (Lesieur & Blume, 1987). This scale overestimates the prevalence figures, while DSM-IV with its 10 questions gives a more conservative estimate of pathological gambling and fewer false-positives. SOGS, on the other hand has more items and gives high false-positive rates in studies of the general population (roughly doubled; Ladouceur et al., 2000). With the conservative measure of DSM-IV, we expected to receive a level of pathological and at-risk gambling more easily comparable between different countries. We therefore made an epidemiological study of pathological gambling and at-risk gambling in the general adult population, using DSM-IV. Our results indicate that Norway has a level of pathological and at-risk gambling at approximately the same level as most other countries (around 1%).

We found more pathological gambling in men than women. Thus, sex is a risk factor. In addition to the establishment of level of gambling problems, we discovered that there was a much higher prevalence in our youngest part of the sample (18-30 years), so we continued to make another study of adolescents. In establishment of the prevalence of gambling problems among adolescents, we found higher prevalences among the adolescents than among adults. Thus, age is also a risk factor. In addition,
we recognized that there were other problem areas than the ordinary gambling plays. So we studied the use of internet and the possible prevalence of “Internet addiction”. In this study we got rather high prevalences of problems around internet use. When we studied internet use, we did not focus on the monetary rewarding factors, as most of the internet use was void of monetary reinforcers. We then investigated in a more general way, the effects of plays without monetary reward, which was almost never approached in research. There, as well as with the internet, we found quite high prevalences of “pathological playing” without monetary reward.

Our results generally confirm results from other studies, although the exact prevalence rates were somewhat lower than in other countries (the use of SOGS instead of DSM explained some of these differences; see Ladouceur et al, 2000).

While working with this material, revealing some risk factors, we recognized that there were very few empirically validated risk factors for Pathological gambling. So, we made a critical review of risk factors. We have not been able to find such an extensive review of risk factors in the literature.

1.1 Risk factors

Some risk factors for pathological gambling are well-known, although most of them have not been scrutinized in well-controlled studies.

1.1.1 Age and gender
Age, as well as gender are well-established risk factors for pathological gambling. Individuals aged 15-24 having a 151% higher risk than individuals aged 25 according to Volberg et al. (2001). There is also a clear relation to gender, with a doubling of female prevalence when it comes to men. The evidence has however not always been clearly proven.

1.1.2 Availability of plays
The gaming business has increased during the last years. Looking at Norwegian reports on gambling, there is a steep increase in the availability and prevalence of gambling behaviour over the last 20 years (Götestam,1993).
Countries with high level of gambling availability also have among the highest prevalence rates of pathological gambling. Availability of gambling is correlated with prevalence of pathological gambling (Campell & Lester, 1999; Walker, 1992). Ladouceur et al. (1999b) 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-years period. The prevalence in the second study showed an increase by 75% in the number of pathological gamblers.

1.1.3 Vulnerability
Among vulnerability factors it has been pointed at alcohol and drug abuse, depression, anxiety disorders, and personality disorders. Quite logically, impulsivity has also been a factor pointed at, according to the classification of PG into impulsive disorders. Any kind of risk factor could at the same time represent a certain vulnerability.

1.1.4 Physiological and biological factors
Physiological and biological factors have been studied by Anderson and Brown (1984) in a study of the correlation between bet staked and heart rate (HR). They showed increases (P<0.0001) in a real casino condition, and a significant correlation between bet staked and HR increase (rxy = 0.741, P<0.0005, one-tailed).

1.1.5 Cognitive factors
Cognitive distortions may affect gambling behaviour, both in quality and amount. Magnification of skill means that the gambler has an exaggerated self-confidence in contrast to his losses. Superstitious beliefs mean that the gambler believes that he reliably can manipulate outcomes in his or her favor. The “Gambler’s fallacy” means that a series of losses is expected to be compensated for. Selective memory, affects the recalling of wins, especially large ones, and difficulty recalling losses (Toneatto 1999).

1.1.6 Comorbidity
Comorbidity between alcohol and drug abuse and gambling has often been stated in the literature (Ladouceur et al., 1999a). The same is the case with other drugs
(Feigelman et al., 1995), although there are more views on the comorbidity, than there is clear evidence.

1.1.7 Type of plays

Some of the types of plays seem to have a more serious risk than others, while some seem to be more safe than others, statements which are often heard in the debate. This is, however, running to conclusions too quick: In the study with adults (I), the most popular play with highest usage percent is Lotto (with 76.0%), and the highest rate on problematic gambling is with slot machines (with 5.7%). With adolescents (II) it is the other way round, with the highest frequency with slot machines (81.8%) and the highest rate for problematic gambling is with Lotto (9.1%). To speak about “high risk plays” or “safe plays” is to overlook that these relations are more complicated than so.

It is fair to suppose that a skills oriented play, will give more real confidence in the situation, and thus increase the risk for playing. Bingo and horse betting are the two extreme points on a continuum from very cheep to very expensive bets. No empirical test has shown that this dimension also affects the strength of PG. The largest “personal economic catastrophes”, however, probably are to be found among the horse betters.

1.2. Prevalences of PG

The level of occurrence of pathological gambling is around 1-2% in the US, Canada, and Europe, around 3% in New Zealand, and around 7% in Australia (Ladouceur & Walker, 1996).

Research into these problems started about 20 years ago, and during the last three years there has been a healthy development of gambling research, into many areas of gambling problems, and with many researchers involved. No specific breakthrough seems to have emerged, but substantial confirmations of earlier knowledge has been ascertained. Still there is a lack of prospective studies, and more causally directed research. The research has mainly been correlational in character.

In lack of rigid empirical research, many opinions prevail, and preventive actions become delayed. The aim with the present study, is to organize the empirical facts on
risk factors for the development and maintenance of pathological gambling, to enable adequate preventive actions, to reduce the extent and seriousness of the problem.

In research on mechanisms and risk factors for development of pathological gambling, researchers have studied both risk-populations and normal populations to investigate the relationships between variables. The search for one main mechanism or risk factor will be futile. What we need is a more collaborative approach, to this manyfolded area, to be able to make more focused preventive actions, and develop treatment programs (see for instance Volberg et al., 1996).

Other researchers have focused on gamblers from the clinical settings. Simultaneous investigations of both gaming frequency and clinical problems and pathological gambling has been relatively rare. Other studies have investigated subgroups of gamblers.

There was no international publication from Norway on the prevalence of pathological gambling, until our publications in 2003. There have been a series of unpublished preliminary reports discussing possible pathological gambling and at-risk gambling. There one could find indications on probable prevalence figures.

2. THE EMPIRICAL STUDY

The empirical study consists of a series of prevalence studies on adults and adolescents, and with focus on different problems in the area of gambling and gambling behaviour. This includes the concept of Pathological gambling (PG); “At-risk gambling” (at-risk for PG), “Internet addition” and gambling or playing with plays without monetary rewards. The last study is a critical review of literature describing risk factors for pathological gambling.
2.1. Study objectives

2.1.1. Major goals
The major goal of the study, was to gather some solid knowledge of the extent of gaming and gambling activities, and potentially harmful consequences, such as Pathological gambling (PG) and “At-risk gambling”. The study should include both adults and adolescents, men and women, and different types of plays (plays with money rewards, plays without money rewards, as well as newer activities, like the use of the internet).

2.1.2. Sub-goals
The aim of the first paper (I) was to get a descriptive overview of money gambling behaviour in Norway in general, and specifically to establish prevalence rates of pathological gambling, and of “at-risk gambling”.

We have chosen DSM-IV (APA, 1994) as we are mainly interested in identifying individuals with pathological gambling and individuals potentially at risk for developing such pathological gambling (DSM-IV exists in an authorized translation to Norwegian).

The aim of the second paper (II) was to investigate a sample of adolescents (12-18 years) to establish how many of them were pathological gamblers, and to get comparable figures for adults and adolescents.

The aim of the third paper (III) was to investigate the pattern of use of the internet among adolescents, in relation to different internet activities, and to excessive use of internet. We also wanted to investigate possible ”Internet addiction”, and predictors for internet addiction. We have chosen to make use of Young’s Diagnostic Questionnaire (YDQ) for Internet Addiction (Young, 1998), translated into Norwegian. It may be said that at the time of data collection (in 1999) there was not much playing and gambling available on internet, as the system merely was used as a practical tool for different activities (reading & mailing, chatting discussion groups, etc), and only 11% using gambling facilities.
In the fourth paper (IV) the aim was to investigate (a) gambling-like problems with computer games without monetary rewards, in relation to (b) different types of gaming (TV/Video, Data/CD-rom, other computer games), and (c) excessive use of computer games, and (d) to find predictors for such gambling-like problems. We wanted to cover the whole spectrum of machine based games, without monetary rewards (not: reading, listening, ancient children games, chess, dame, ludo, etc).

The aim of the fifth paper (V) was to review and organize the empirical facts on risk factors for the development and maintenance of pathological gambling, to enable adequate preventive actions and to reduce the extent and seriousness of the problem.

The hypotheses were that (1) excessive gaming occurs in a normal adult and a normal youth population; (2) ”pathological playing” occurs in the studied populations, and (3) the prevalence of ”pathological playing” without monetary reward occurs to at least the same degree as that of pathological gambling with monetary reward.

We expected that “established” risk factors for Pathological gambling would appear in the search, but very few such factors were possible to find, due to lack of empirical support (no studies, badly performed studies, inconclusive results). Thus speculations about risk would be more adequate than conclusive risk factor computations.

2.2 Material and methods

2.2.1. Subjects
In Paper I a total of 2014 adults were obtained by random-digit telephone dialling of residential dwellings, including both sexes. This covered the entire country. If the answerer was 18 years old or older, the telephone interview was performed.

In papers II-IV a community sample of 3237 individuals (12-18 years of age), was collected for the survey. One part of the subjects were interviewed by telephone (n=1913) and another part completed a postal survey questionnaire (n=1324).
2.2.2. Procedures

2.2.2.1 Assessment of prevalences

Pathological gambling (PG) is defined as an impulse control disorder that does not involve an intoxicant. In papers I and II we used the questionnaire based on the DSM-IV criteria (APA, 1994) for pathological gambling.

**DSM Criteria for Pathological Gambling**

The diagnostic criteria for Pathological gambling read as follows (312.31):

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:

1. Is preoccupied with gambling (e.g., preoccupied with reliving past gambling experience, handicapping or planning the next venture or thinking of ways to get money with which to gamble)
2. Needs to gamble with increasing amounts of money in order to achieve the desired excitement
3. Has repeated unsuccessful efforts to control, cut back or stop gambling
4. Is restless or irritable when attempting to cut down or stop gambling
5. Gambles as a way of escaping from problems or relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)
6. After losing money gambling, often returns another day to get even (“chasing” one’s losses)
7. Lies to family members, therapist or others to conceal the extent of involvement with gambling
8. Has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling
9. Has jeopardized or lost a significant relationship, job or educational or career opportunity because of gambling
10. Relies on others to provide money to relieve a desperate financial situation caused by gambling.

B. The gambling behavior is not better accounted for by a Manic Episode.
The response categories are yes or no. Respondents who answered yes to five or more of the 10 criteria were classified as pathological gamblers, and those who answered yes to 3-4 criteria, were classified as "at-risk gamblers". We have used the designation "problematic gambling" for categorization of the group of pathological gambling plus "at-risk gambling". We assessed the frequency of gambling for money, if they played never or seldom, 1-3 days per week, 4-5 days per week or 6-7 days per week.

In the data collection, only players using gambling for money on a weekly basis (one or more days per week) were given the DSM-IV criteria questions. In Paper I and II we also assessed the frequency of gambling for money, if they played never, sometimes or often (weekly), and the yearly amount of money bet on gambling and which types of plays they used.

**Young’s “Diagnostic Questionnaire for Internet Addiction (YDQ)”**

In Paper III we choose “Diagnostic Questionnaire for Internet Addiction” (YDQ; Young, 1998) as we were interested to obtain diagnostic categories for the phenomenon of internet addiction, or pathological and excessive use of internet. Young (1998) has described internet addiction as an impulse-control disorder which does not involve an intoxicant. Young’s DQ (1998) was based on the DSM-IV criteria for pathological gambling (APA, 1994). This questionnaire consists of eight yes/no questions:

1) Do you feel preoccupied with the internet (think about previous on-line activity or anticipate next on-line session)?
2) Do you feel the need to use the internet with increasing amounts of time in order to achieve satisfaction?
3) Have you repeatedly made unsuccessful efforts to control, cut back, or stop internet use?
4) Do you feel restless, moody, depressed, or irritable when attempting to cut down or stop internet use?
5) Do you stay on-line longer than originally intended?
6) Have you jeopardized or risked to loss of significant relationship, job, educational or career opportunity because of the internet?
7) Have you lied to family members, therapist, or others to conceal the extent of involvement with the internet?
8) Do you use the internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)?

Respondents who answered yes to five or more of the eight criteria were classified as "addicted internet users" and the remainder were classified as normal internet users. DSM-IV also uses the cut off score of five for pathological gambling, although DSM-IV has 10 and not only 8 criteria. The term internet use denotes all types of on-line activity (Young, 1998). In this evaluation of internet addiction on a population, we have found it helpful to use 3-4 criteria of YDQ to denote "at-risk internet use", to indicate that these young persons are at risk to develop an "internet addiction". In some data presentations we have also used the designation "Problematic internet use" for internet addiction plus at-risk internet use. In data collection only individuals using internet on a weekly basis (one or more days per week) were asked to fill out Young’s Diagnostic Questionnaire (Young, 1998). The DQ by Young (1998), was validated on our sample (Johansson & Götestam, 2003, and below).

In paper IV we also choose Young’s (1998) "Diagnostic Questionnaire for internet addiction" (YDQ) as we were interested to get diagnostic categories for the phenomenon of "pathological playing" with plays without monetary rewards. With this special angle the YDQ appeared more appropriate than DSM. Respondents who answered yes to five or more of the eight YDQ criteria were classified as "pathological players". We also use 3-4 criteria of YDQ to denote "at-risk” playing. We have also used the designation "problematic playing" for pathological playing plus "at-risk” playing on plays without monetary rewards. In the data collection only players who played on a weekly basis (one or more days per week) were asked to fill out the YDQ.

For DSM-IV, the questions are not asked with a specific time reference, but they are worded in the present tense, and considered to yield a point prevalence figure. However, some of the questions, as the one about “chasing” (6), and about committed illegal acts (8), are rather representing lifetime prevalence. On these grounds, in the present studies, the prevalence rates are mainly interpreted as state prevalence figures.
For YDQ, most of the questions appear quite clear to refer to in present tense (partly with the exception of the question about lying to the family) (7). Likewise are the prevalence rates from YDQ mainly interpreted as state prevalence figures.

2.2.2.2. Literature review of risk factors

For the review of literature on risk factors for PG, we based our inclusion criteria partly on the research synthesis made by Shaffer et al. (1999). In the present review, thus, we included (1) original research on PG; (2) which was published in international scientific journals; (3) specified the size of the sample; (4) with specification of the instrument used to identify disordered gambling and comparison variables; (5) specified the design used; (6) included and specified adequate statistical analyses; (7) and if the documentation was adequate; and (8) the results were conclusive. A fuller description of literature background and the review procedure could be found in V.

2.2.3 Statistical Analyses

In Paper I a weight index was used from Age x Sex x Geography to give the present sample a representativity in relation to the general population.

In Papers II-IV a similar weight index was not used, as the relatively small numbers in the central entities in the study (Pathological Gambling and At-Risk Gambling) became too much skewed, changing the values too much.

Product-moment correlations were computed between degree of gambling and some established risk factors as sex (male) 0.142 (P<0.01), age (low) 0.094 (P<0.05), education (low) 0.081 (P<0.05). Employment (unemployed) showed a negative correlation, -0.095 (P<0.01), while civil status was not correlated. A chi-square test was performed for the relation between smoking and gambling, showing a clear relation between smoking and gambling ($\chi^2=41.01$, P<0.001).

In Paper II forward regression analyses were performed with "pathological gambling" as dependent variable and the following predictor variables: demographic variables (gender, age, geography), further demographic variables (school, work, domicile type), habits of use (frequency, numbers of hours per week), and type of
plays played (slot machines, football betting, Lotto, lotteries, bingo, poker & casino, toto, other).

In Paper III the scale characteristics were investigated in relation to reliability and consistency for the YDQ (Young 1998; Johansson & Götestam, 2003; SPSS, 1999). The calculation of a Spearman-Brown coefficient resulted in a split-half reliability of 0.729. The consistency of the YDQ was tested with Cronbach’s alpha (0.713, with standardized item alpha 0.759). A Principal Component Analysis (PCA) was computed. The first component had an eigenvalue of 2.921, while the three following components were all below 1.0. Thus, the first component was selected. This component explained 36.5% of the variance, and the eight items gave correlations of 0.541 to 0.657. Spearman’s rho correlations, calculated between the eight items, show highly significant correlations (p<0.01). Thus, the YDQ has a good reliability, consistency and unidimensionality.

Forward regression analyses were performed with internet addiction as dependent variable and the following predictor variables emerged: demographic variables (gender, age, geography), further demographic variables (school, work, domicile type), different places of internet access (no access, home, school) habits of use (frequency, number of hours per week), and types of activities on internet (reading news, etc.).

In Paper IV forward regression analyses were performed with "problematic playing" as dependent variable and the following predictor variables: demographic variables (gender, age, geography), further demographic variables (school, work, domicile type), habits of use (frequency, number of hours per week) and type of plays (simulator plays, sport plays, strategy plays, shooting/war plays, fighting plays, adventure plays, flipper plays).

In paper V, the critical literature review, we examined investigations using correlation, t-test, odds ratio, ANOVA (analyses of variance) and regression in their analyses.
2.2.3 **Potential conflict of interest**

The study has received financial support from Norsk Tipping (Norwegian State Lottery), and Norsk Lotterdrift (Norwegian Lottery Co). The authors of the scientific articles take complete responsibility for the integrity of the data and the accuracy of the data analyses, and have full control over the decisions about publication.

2.3. **Results**

**Paper I. Characteristics of gambling and problematic gambling in the Norwegian context. A DSM-IV-based telephone interview study** *(Addictive Behaviors, 28, 189-197, 2003).*

In Paper I we investigated the prevalence of pathological gambling and at-risk gambling. An epidemiological study was performed in a representative sample of the Norwegian population (n=2014, response rate 47.8%). The study sample contained 48.9% men and 51.2% women. Age distribution was rather balanced, and was divided into three age groups, 18-30, 31-50 and 50 and above. Those who never gambled amounted to 31.2% and gambling sometimes 47.2% and often gambling 21.0%. Men (25.5%) gambled more often than women (17.7%). The most popular and used games were Lotto (76%), football betting (10.8%), slot machines (5.1%) and lotteries (4.9%). There was a discrepancy between most used and popular plays and rank of problematic gambling. Slot machines gave higher rank for problematic playing. The four most used plays were all connected to problematic gambling. Slot machines were used with a frequency (5.1%), comparably to the frequency of problematic gambling (5.7%), while the most frequent play, Lotto (76%) gave a very low problematic gambling frequency (0.4%). Problematic gambling (pathological gambling plus at-risk gambling) was 0.60%. Men 18-30 had much higher prevalence (2.83%), compared to men over 30 (0.28%) and females 18-30 (0.84%) and over 30 (0.12%). There was none with problematic gambling over 50 years in the sample.

The DSM-IV gives a conservative estimate of pathological gambling. The first criterion in DSM-IV (preoccupied with gambling) was most frequently seen (in 3.1% of the total population). Number two (needs to gamble with increasing amounts;
2.4%), number 5 (a way of escaping from problems; 2.1%) and number 6 (returning and “chasing” 2.1%).

There were rather low but significant correlations between gambling and most of the established risk factors: sex (male), 0.142 (P<0.01), age (low) 0.094 (P<0.05), education low) 0.081 (P<0.05). There was also a clear relation between smoking and gambling ($\chi^2=41.01$, P=.001).

**Table 1.** Prevalence of pathological gambling, at-risk gambling, and total problematic gambling in adult (18-years), men and women. Assessments by DSM-IV (Götestam & Johansson, 2003).

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathol gambling</td>
<td>0.15</td>
<td>0.21</td>
<td>0.09</td>
</tr>
<tr>
<td>At-risk gambling</td>
<td>0.45</td>
<td>0.74</td>
<td>0.19</td>
</tr>
<tr>
<td>Problematic gambling</td>
<td>0.60</td>
<td>0.95</td>
<td>0.28</td>
</tr>
</tbody>
</table>

To easily compare the different main result figures for the four epidemiological studies, a simple table is extracted from each of the different studies (see Table 6).


In Paper II an epidemiological study was performed on a representative sample of the Norwegian youth population (12-18 years, $n = 3237$, response rate 45.2%). Those who never gambled were 17.6%, those who seldom gambled were 57.5%, whereas 24.9% were weekly gamblers (36.2 % of the males and 13.1 % of the females). According to problematic gambling, the results showed that 1.76 % had pathological gambling (2.79 % in men and 0.69 % in women) and 3.46 % "at-risk gambling”. Problematic gambling (pathological gambling plus "at-risk gambling") was 5.22% (7.82% of the men and 2.52% of the women).

Of the group weekly gambling 7.08% had pathological gambling (7.69% of males and 5.31% of females) and 13.91% had "at-risk gambling”. The DSM-IV gives a conservative estimate of pathological gambling.
The most popular game was slot-machines with 81.8%, followed by football betting (70.8%), Lotto (68.7%) and lotteries (39.4%). Lotto ranked highest in problematic ranking, compared to other plays (slot machines and football betting) that were used slightly more frequently. Those who played weekly was 24.9% (n=805). Among those 49.6% had no symptoms, 29.4% had one or two symptoms, while 13.9% had three to four symptoms and 7.08% had five or more symptoms. The sixth criterion (returning and "chasing") was most frequent, both total and in the group weekly gamblers. The mean debut age was 9 years (range 4-18), mean playing time per week was 1.77 hours (range 0-30h) and the mean amount spent was 79.02 Norwegian Kroner (range 1-2500). The correlation between degree of pathological gambling and money spent was 0.348 (P<0.01) and used hours 0.220 (P<0.01), while the correlation between degree and debut age was although significant, only -0.051 (P<0.01).

Table 2. Prevalence of pathological gambling, and at-risk gambling, and total problematic gambling in youths (12-18-years), men and women. Assessments by DSM-IV (Johansson & Götestam, 2003).

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathol gambling</td>
<td>1.76</td>
<td>2.79</td>
<td>0.69</td>
</tr>
<tr>
<td>At-risk gambling</td>
<td>3.36</td>
<td>5.03</td>
<td>1.83</td>
</tr>
<tr>
<td>Problematic gambling</td>
<td>5.12</td>
<td>7.82</td>
<td>2.52</td>
</tr>
</tbody>
</table>


In Paper III an epidemiological study was performed on a representative sample of the Norwegian youth population (12-18 years, n = 3237, response rate 45.2%). There were only 4.9% not using internet at all, while 35.8% were non-frequent users, and 49.6% were weekly users. The internet was used on the mean 4.3 hours a week. A mean of 1.98% (men 2.42%, women 1.51%) could be described to have internet addiction according to the criteria in the Diagnostic Questionnaire of Young (1998) and an additional 8.68% were considered to have an at-risk internet use (men 9.21%, women 8.13%). Among individuals who used internet weekly 4.02% fulfilled 5 criteria, and an additional 17.66% 3-4 criteria. The different diagnostic criteria gave a broad range of affirmative answers from 0.4% to 27.9%.
Only 9.48% of the adolescents did not have access to internet. Most of them (64.6%) used internet 1-3 days per week, and usually for 2-3 hours. On the mean, 1-2 symptoms were most frequent (49.7%), and the mean number of symptoms was 2.5. Problematic internet use was defined as internet addiction plus at-risk internet use.

There were significant sex differences both for internet addiction and at-risk Internet use (P<0.01). There were 27.9% who stayed online longer than intended, 14.5% were preoccupied with internet and 0.4% had lost relationships with significant others. The correlation between frequency of use and internet addiction was relatively low (rxy=0.284, P<0.01, which contradicts the notion that internet in itself would represent a risk factor.

**Table 3.** Prevalence of “internet addiction”: pathological gambling, at-risk gambling, and total problematic gambling in adolescents (12-18 years) boys and girls. Assessments by Young Questionnaire (Johansson & Götestam, 2004a).

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathological gambling</td>
<td>1.98</td>
<td>2.42</td>
<td>1.51</td>
</tr>
<tr>
<td>At-risk gambling</td>
<td>8.68</td>
<td>9.21</td>
<td>8.13</td>
</tr>
<tr>
<td>Problematic gambling</td>
<td>10.66</td>
<td>11.63</td>
<td>9.64</td>
</tr>
</tbody>
</table>

**Paper IV. Problems with computer games without monetary reward**

*Psychological Reports, 95, 641-650, 2004*.

In Paper IV an epidemiological study was performed on a representative sample of the Norwegian youth population (12-18 years, n = 3237, response rate 45.2%). Weekly players of different computer games was 63.3% (n=2050) and non frequent users was 36.7%. A mean of 2.7% (male 4.2%, female 1.1%) could be described to be pathological playing according to the criteria in the Diagnostic Questionnaire of Young (1998) and an additional 9.82% were considered to be "at-risk" playing (male 14.5%, female 5.0%). Of weekly gamblers 4.2% fulfilled 5 criteria for pathological playing and an additional 15.5% 3-4 criteria at-risk playing. Mean time playing per week on TV/Video games was 6.05 hours (range 0-65), on Data/CD games 6.36 hours (range 0-60) and on other plays 2.1 hours (range 0-20). The diagnostic criterion number 5 (do you gamble longer than intended) and number 1 (do you feel preoccupied with gambling) gave the highest frequencies of affirmative answers.
Correlations between frequent use and problematic playing gives TV/Video $r_{xy} = 0.345$, Data/CD-rom $r_{xy} = 0.418$, computer games $r_{xy} = 0.297$, (all p’s <0.01) which indicates a relation between frequent playing and problematic playing.

**Table 4.** Prevalence of plays with no monetary reward: pathological gambling, at risk gambling, and total problematic gambling in adolescents (12-18 years) boys and girls. Assessments by Young Questionnaire (Johansson & Götestam, 2004b)

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathological gambling</td>
<td>2.70</td>
<td>4.20</td>
<td>1.10</td>
</tr>
<tr>
<td>At-risk gambling</td>
<td>9.82</td>
<td>14.50</td>
<td>5.00</td>
</tr>
<tr>
<td>Problematic gambling</td>
<td>12.52</td>
<td>18.70</td>
<td>6.10</td>
</tr>
</tbody>
</table>

**Paper V. Risk factors for problematic gambling: a review** (*Research Report, Section for Psychiatry and Behavioural Medicine, NTNU, No 95, Vol 25, 2004c*)

The aim of this study was to organize the existing empirical facts on risk factors for the development and maintenance of pathological gambling. We have reviewed the international research literature and found very few publications with some form of overview, earlier critical reviews or specifically non-empirical and theoretical publications. The main part is a critical review of risk factors found in the literature, which can be described as well-established risk factors, probable risk factors or potential risk factor for pathological gambling. The risk factors are categorized with demographics variables (age, gender), physiological and biological factors, cognitive distortions (erroneous perceptions, illusion of control), sensory characteristics, schedules of reinforcement, comorbid states (OCD, drug abuse) and delinquency/illegal acts, concurrent symptoms and personality symptoms and characteristics.

Demographic variables as age and gender are well-known risk factors. Availability of plays is also an important and well-known risk factor. With increased availability both the playing and the pathological gambling increase. This could be observed both in relation to availability of plays over time, and different availability of different plays.
Table 5. Risk factors for PG – summary of results

<table>
<thead>
<tr>
<th>Domain</th>
<th>Risk factor</th>
<th># of studies</th>
<th>Level*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Demographics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.1 Age</td>
<td>low age</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Gender</td>
<td>male</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>2. Cognitive distortions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.1 Illusion of control</td>
<td>illusion</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>3. Varia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.1 Availability of plays</td>
<td>high</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>3.2 Sensory characteristics</td>
<td>yes</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>3.3 Schedules of reinforcement</td>
<td>type</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>4. Comorbidity &amp; concurrent symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.1 OCD</td>
<td>yes</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>4.2 Other drugs</td>
<td>yes</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>5. Personality symptoms and characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.1 Delinquency &amp; illegal act</td>
<td>yes</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

* 1 = well-established risk factor for PG

Other important variables were sensory characteristics of data plays (sound, light) and the schedules with random rewards as the strongest reinforcer.

From clinical work we know that there is a comorbidity between PG and OCD (obsessive-compulsive disorder) and drugs. PG is also closely related to criminality, as shown by several studies.

3. Discussion

The main results show that the prevalence of Pathological gambling and at-risk gambling are at the same level as other European countries, we usually compare ourselves with.

We have found that the level of gambling problems is higher in young adults (about ten times as much), and adolescents, than in older adults, and above the age of 50 there are relatively few gambling (so far). With Internet there is more than ten times more (in 1999; today is it probably manifold more frequent). Plays without monetary
rewards are highly addictive, with twenty-fold the prevalence in adolescents compared to normal adults.

To compare the different prevalence figures for different types of problematic gambling, we have put them together into one table (Table 6).

Table 6. Prevalence of pathological gambling of different types, divided on gender in the different prevalence studies (I-IV).

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults</td>
<td>0.15</td>
<td>0.21</td>
<td>0.09</td>
</tr>
<tr>
<td>Adolescents</td>
<td>1.76</td>
<td>2.79</td>
<td>0.69</td>
</tr>
<tr>
<td>Internet</td>
<td>1.98</td>
<td>2.42</td>
<td>1.59</td>
</tr>
<tr>
<td>Without money</td>
<td>2.70</td>
<td>4.20</td>
<td>1.10</td>
</tr>
</tbody>
</table>

We conclude that our hypotheses have been supported, and our results have lately been confirmed by some reports in Norwegian, have investigated the problem area (SIRUS, NOVA). Important implications from our results is the recognition that problematic gambling is an important problem. We are now also able to estimate the size of the problem, and its possible consequences. This will enable the government to prepare for potential increases in the problem, stop the development of a fast-running development (specifically with the Internet), and take control (cp Volberg et al, 1996).

Still it is also possible to perform prospective studies, which are still lacking. The needs for intervention and prevention will become important governmental tasks, as treatment offers available.

Prevalences by phone or postal questionnaire

Both phone and postal questionnaire was used in the second study (II). For PG the prevalence was higher for phone method (2.24%) against postal questionnaire (1.06%). Similarly for at-risk gambling the prevalence for phone method gave higher values than postal questionnaire (3.97% vs 2.72%). Chi square analyses showed significant differences between the two interviewing procedures, for PG ($X^2=8.34$, $p<0.004$), for at-risk gambling ($X^2=7.86$), and when both problematic gambling groups were put into the analysis ($X^2=12.58$, $<0.001$). Although the prevalence differences are highly significant, the numeral difference are quite small.
Scandinavian data compared

There exist in Scandinavia prevalence data from Sweden and Norway (with Danish data to arrive in June 2006), which could be compared to our published data. There are differences in instruments used, which affects the comparability. The differences in age span in the groups investigated, also makes the comparison difficult as the age factor has great impact in gambling problems.

As earlier indicated, DSM is a conservative measure, while for instance the extensively used SOGS is quite liberal, and usually gives prevalences about double that for DSM (Ladouceur et al., 2000). Also, most studies have mixed adolescents and adults in the same sample, which is also reducing the comparability.

DSM (Diagnostic and Statistical Manual for Mental Disorders) is the "original instrument" used (latest version DSM-IV, 1994). SOGS (South Oaks Gambling Screen), and SOGS-R, which is the most used instrument, is based on DSM. NODS (NORC DSM Screen for Gambling Problems), is also based on DSM. CPGI (Canadian Problem Gambling Index), is built on 3 questions from DSM, and Lie/Bet Screen (2 items from DSM) was by Rossow and Hansen (2003) supplemented with the "chasing" item in DSM.

Table 7. Comparison between Norway & Sweden

<table>
<thead>
<tr>
<th>Reference</th>
<th>Instrument</th>
<th>Pathol Gambl (age) (N)</th>
<th>Probl /At risk (age) (N)</th>
<th>Yr data/publ ¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Götestam &amp; Johansson</td>
<td>DSM-IV</td>
<td>0.15%</td>
<td>0.45%</td>
<td>1997 / 2003</td>
</tr>
<tr>
<td>Johansson &amp; Götestam</td>
<td>DSM-IV</td>
<td>1.76%</td>
<td>3.46%</td>
<td>1999 / 2003</td>
</tr>
<tr>
<td>Volberg, Rönnberg</td>
<td>SOGS-R DSM-IV ²</td>
<td>1.2%</td>
<td>2.7%</td>
<td>1999 / 2001</td>
</tr>
<tr>
<td>Rossow, Hansen, NOVA</td>
<td>“Lie/Bet”</td>
<td>3.2%</td>
<td>6.0%</td>
<td>2003 /</td>
</tr>
<tr>
<td>Lund, Nordlund, SIRUS</td>
<td>SOGS NODS</td>
<td>0.6%</td>
<td>0.8%</td>
<td>2003 /</td>
</tr>
<tr>
<td>MMI</td>
<td>CPGI</td>
<td>1.9%</td>
<td>3.6%</td>
<td>2005 /</td>
</tr>
</tbody>
</table>

¹ Year for the data collection, and for international publication ² Used, but data not separately reported

The first study of ours (Götestam & Johansson, 2003) is on an adult population with prevalence of PG on 0.15% and at-risk gambling 0.45. Lund and Nordlund (2003) give roughly the same values on NODS (0.3% and 0.4%), on a mainly adult population. Our second study (Johansson and Götestam (2003) is on an adolescent...
population. Comparatively, Rossow and Hansen (2003) gives as expected around the double values on SOGS (0.6 and 0.8). Volberg (et al. 2001) with Rönnberg on a mixed age Swedish population gets values somewhat different, but in the same ballpark. In comparison the Lie/Bet Screen data (2 LBS items and the “chasing” item from DSM) gives Rossow and Hansen (2003) on adolescents high prevalences of gambling problems. In comparison we have data on Lie/Bet Screen (Götestam et al., 2004) giving a quite similar prevalence of 5.5% for adolescents.

Why was it important to perform the present study? As long as we were ignorant towards the gambling problem, we were not expected to be able to reduce or prevent the problem. We need exact knowledge of the prevalence of gambling problems, and its changes over time. For this, we still lack good prospective studies. They are important for the prediction and assessment of the development over time. This enables intervention and treatment for people with gambling problems, and the prediction and prevention of a malignant development and spreading of the problem. We have an impression that gambling problems today are very steeply increasing. Maybe on an individual basis, this is correct. However, to conclude that this is also the case on a societal basis is premature. It may well be that there are waves, and their extension goes up and down. A clear upward trend, however, will indicate a serious development of the problem. During the last years, several epidemiological studies have been performed, and we certainly have better control of the development today (see Volberg et al., 1996), in Norway as well as in other countries. But with the rapid technical development we may also expect the development of other and newer problem behaviours related to pathological gambling (as for instance ”e-mail addiction”).


Another problem is the treatment of people with pathological gambling. The availability of good and effective treatment is very far from satisfactory, as few health professionals are trained to give good treatment for gambling. Furthermore, the treatment methods supplying effective treatment is even fewer.

We should therefore design future applications from this knowledge, to develop better treatment and prevention studies. Today there exist few developed treatment strategies. In a Cochran analysis, Oakley-Browne et al. (2003) found 17 treatment studies, of which only four fulfilled RCT criteria. Mainly, they found some support for cognitive therapy as an effective treatment for pathological gambling, but there is also a need for better interventions, and broader applications of them.

There are some weaknesses with this research. The most serious are the relatively low response rates (although they are in the expected range, which is lower than earlier). This we have tried to overcome by triangulation, and the use of both phone interviewing and paper survey. The other studies in the area confirms also the validity of our studies.

We have used a valid and conservative measure for the diagnosis of pathological gambling, the DSM-IV (APA, 1994), which also makes it comparable to other studies.

The results could also be of theoretical interest in several ways. One is the evaluation of risk factors for PG. This approach has several facets, which could stimulate research and collaboration. Furthermore, the close links between PG and the abuse of drugs, may be a potential for collaboration, and a clearer understanding of this “drug addiction disorder without a drug implicated”.

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We definitely believe that our results are valid, taken the results from other countries, and the reports in Norwegian performed after our studies, with results in the same range.

The results from the literature review are also discussed in the last paper (V).

4. Main Conclusions

From the present study, it is possible to derive a series of results. We have tried to list the main conclusions accordingly, from the included papers (I-V).

Paper 1

1. Men (25.5%) gambled more often than women (17.7%) and the highest frequency of gambling was in the youngest age group, 18-30 years (18.6%), where also most of the problematic gamblers (1.8%) were found, compared to the total value (0.6%).

2. Slot machines had a low-frequent use (5.1%) but a high problematic gambling frequency (5.7%), while the most frequent play, Lotto (76%), gave a low problematic gambling frequency (0.4%).

3. Pathological gambling and at-risk gambling together for young men (18-30 years) was high (3.15%), and quite high when compared to men over 30 years (0.28%). For females the frequencies are lower, for 18-30 (0.84) and over 30 (0.12%).

Paper II

4. Among types of plays, slot machines were most popular, with football betting followed by lotteries. Among the problematic gamblers, the state lotteries got the highest priority, slot machines was second and football betting number three.

5. The frequency of pathological gambling in the total material was 1.76% (with 2.79% for males and 0.69% for females). The frequency of the total at-risk gambling is 3.46% (5.03% for males and 1.83% for females). Adding the two categories to a total "problematic gambling" category gives a percentage of 5.22% (males 7.82%, females 2.52%).
6. Phone interviews gives higher frequencies than with interviews questionnaire.

7. The prevalence of problematic gambling is higher for phone interviews than for postal questionnaire

8. In the different DSM-IV criteria there is a broad range of affirmative answers (from 0.3% to 7.8%). Criterion 6 ("chasing") was most frequent.

Paper III
9. Only 9.48% of the adolescents did not have access to the internet. Most of the respondents (64.6%) used the internet 1-3 days a week. The response category "use of Internet 2-3 hours per week" was most often selected (37.0%), and the mean weekly use was 4.3 hours.

10. Problematic internet use is defined as internet addiction plus at-risk internet use. The frequency of internet addiction in the total material is 1.98% (2.42% for boys and 1.51% for girls). The frequency of the total at-risk internet use is 8.68%, (9.21% for boys and 8.13% for girls).

11. Adding the two categories, to a total "problematic internet use" gives a percentage of 10.66% (boys 11.63%, girls 9.65%).

12. Forward regression analyses with internet addiction as the dependent variable showed significant t values for different places of internet access (home); internet habits (frequency; numbers of hours per week; and types of activities on the internet) but not for demographic variables (age, gender, geography and social background variables as schooling, work, domicile type).

Paper IV
13. Data/CD games were most frequent in comparison with TV/Video games, while other computer games, where the youth only could play in public places, were less
frequent. Very few participants (1.5%) had not gamed any of the games studied. 77.6% had gamed with TV/Video games, 86.3% with Data/CD games, and 15.6% with other computer games without monetary reward.

14. The frequency of "pathological playing" in the total material was 2.7% (4.2% for boys and 1.1% for girls). The frequency of total "at-risk playing" was 9.8% (14.5% for boys and 5.0% for girls). Adding the two categories to "problematic playing" gives a percentage of 12.5% (boys 18.7%, girls 6.1%).

15. Forward regression analyses with problematic playing as dependent variable showed significant t values for gender (t=18.21, P<0.001), age (t=6.18, P<0.001, and hours gamed (t=3.01, P<0.003), and for type of games: shooting/war games (t=5.151, P<0.001), fighting games (t=2.62, P<0.009), and strategy games (t=2.32, P<0.02).

16. The correlation between frequency of use (within the week) and problematic playing was substantial (TV/Video: r_{xy}=0.345, Data/CD-rom: r_{xy}=0.418, computer games: r_{xy}=0.297, all P<0.01, indicating gaming frequency as related to problematic playing.

Paper V

17. The critical review of 35 different factors where nine factors were "well-established risk factors for PG" 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 are supported by 1-2 studies.

18. A strong group of variables consists of demographics, where age and gender are clear risk factors in a number of studies.

19. Among the strongest variables are the availability of plays, with increased availability, both playing and pathological gambling increases. That could be seen
both in the increase in plays available over time, and differential availability of different plays.

20. Other important variables are sensory characteristics of computer plays (sounds, lights), and the schedules of reinforcement, with random reward being the strongest rewarding factor.

21. From clinical work it is well-known that there is a comorbidity between PG on one hand and obsessive-compulsive disorder (OCD) and drugs on the other. PG is also closely related to delinquency and criminality which is shown in this review.
5. References


Stromgren E: *Psykiatri* (6 ed 1954, after JC Smith, 1939), Copenhagen:

Munksgaard

Stromgren E: *Psykiatri* (11 ed, 1974), Copenhagen: Munksgaard


Paper I
Brief report

Characteristics of gambling and problematic gambling in the Norwegian context
A DSM-IV-based telephone interview study

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Abstract

The gaming business has increased considerably during the past years, and there are also some indications that the prevalence of pathological gambling has also increased. As it is important to know the problem size and character, an epidemiological study was performed in a representative sample of the Norwegian population (N = 2014; response rate 47.8%). The proportion that never gambled was 31.2%, and a majority (47.2%) gambled sometimes, while 21.0% gambled often. Men (25.5%) gambled more often than women (17.7%). Lotto was the most popular game with 76.0%, followed by football tips (10.8%), slot machines (5.1%), and lotteries (4.9%). For some types of plays, there was a discrepancy between rank for playing, and for problematic playing. Slot machines gave higher problematic playing rank. The mean prevalence of problematic gambling (pathological gambling plus at-risk gambling) was 0.60%, with higher prevalence for those younger and for men. Men 18–30 had a very high prevalence (2.83), compared to men over 30 (0.28%) and females 18–30 (0.84) and over 30 (0.12%). The total problematic gambling frequency was 1.97% for 18–30 years, and 0.1% over 30. There are no problematic gamblers over 50 in the material. The DSM-IV with its only 10 questions gives a conservative estimate of pathological gambling. There were significant correlations between degree of gambling and some established risk factors.

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Keywords: Gambling activity; Pathological gambling; Prevalence; Norway; Risk factors

* A preliminary version of this study was presented at the Annual Meeting of the Society of Behavioral Medicine, New Orleans, March 1998.
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E-mail address: gotestam@medisin.ntnu.no (K.G. Götestam).
1. Introduction

Looking at Norwegian reports on gambling, there is a rather steep increase in the availability and prevalence of gambling behavior over the last 20 years (Götestam, 1993). The extent of “pathological gambling,” as defined by, for instance, the DSM diagnostic system (American Psychiatric Association [APA], 1994), has not so far been known for the Norwegian context, although there are reports from other parts of the world (Volberg, Dickerson, Ladouceur, & Abbott, 1996). A few reports have also indicated an increase in pathological gambling during the last years (Ladouceur, Jacques, Ferland, & Giroux, 1999; Shaffer, Hall, & Vander Bilt, 1999). That would then correlate with the increase of gaming and gambling behavior (see Götestam, 1993).

The assessment of problematic and pathological gambling has been made with ordinary diagnostic systems, but during the last years, the South Oaks Gambling Screen (SOGS), introduced by Leisuer and Blume (1987), has taken the DSM-III and DSM-III-R (APA, 1980, 1987), as a starting point, for the development of a new scale, which has been frequently used. SOGS has a high sensitivity for lifetime problematic and pathological gambling in normal populations (Abbott & Volberg, 1996). There also exist other developments from DSM-III and DSM-III-R, but they all depart from DSM in their constructions, with more response categories, specifically weakening the diagnostic power of these instruments. The DSM-III-R has now been improved in the recent DSM-IV (APA, 1994). In this version, especially the dysphoric and escaping signs have been added for pathological gambling. Although DSM-IV has been used in some studies of gambling, so far, it has not been extensively used. There are also other diagnostic instruments used, but none were widely used for gambling.

Although there were some forerunners, the estimation of pathological gambling was started by Volberg and Steadman (1988) in the US, followed by a series of studies there (see Volberg, 1996), and also in Australia and New Zealand (Volberg & Abbott, 1994) and in Canada (Ladouceur, 1996) for a review see Petry & Armentano (1999).

In their New Zealand study, Volberg and Abbott (1994) listed certain risk factors for the development of pathological gambling: male, under 30, non-Caucasian, not married, unemployed. The list was later enlarged with: less than high school, and income under US$25,000 (Volberg, 1996).

The occurrence of problematic gambling has implications for public health. Knowledge of the occurrence and profile of gaming and gambling activities are of great importance when actions for legislation to control the overall rate of gambling problems in the general population and service options for at-risk individuals and groups should be implemented (Volberg, 1994).

From a survey of gambling policy in 38 states in the US, Canada, Australia, and New Zealand (Volberg et al., 1996), it is also clear that the knowledge from prevalence studies is essential for the establishment of adequate types of service for problematic gamblers and their families. States with deficit in such knowledge tended to establish types of service not meeting the needs, not being used, or in other ways failing (Volberg et al., 1996).
The aim of the present study was to get an overview of money gambling behavior in Norway in general, and specifically prevalence rates of pathological gambling. We have chosen DSM-IV as we are mainly interested in identifying individuals with pathological gambling, and individuals potentially at risk for developing such pathological gambling.

2. Methods

2.1. Subjects

A total of 4820 subjects were obtained by random-digit telephone dialing of residential dwellings. This covered the entire country. If the answerer was 18 years old or over, the interview was tried started, and 2014 completed a telephone interview.\(^1\) It included both sexes. The response rate was 47.8%.

The respondents were classified according to gender, age, education, working status, and marital status (see Table 1).

2.2. Questionnaire

We used the DSM-IV (APA, 1994) with the following 10 diagnostic criteria: (1) preoccupied with gambling; (2) needs to gamble with increasing amounts of money; (3) has repeated unsuccessful efforts to control, cut back, or stop; (4) is restless or irritable when attempting to cut down; (5) gambles as a way of escaping from problems; (6) after losing money, often returns ("chasing"); (7) lies to family members, therapist, or others; (8) has committed illegal acts; (9) has jeopardized or lost a significant relationship, etc; (10) relies on others to provide money. The response categories are yes or no. We had extensive experience with the use of the DSM-IV instrument. Affirmative answer of five or more of the criteria constitutes a case with pathological gambling. Cases with three to four criteria fulfilled were defined as "at-risk gambling," and pathological gambling plus at-risk gambling were called problematic gambling.

We also assessed the frequency of gambling for money, if they played never, sometimes (<weekly), or often (≥ weekly), and the yearly amount of money bet on gambling. Which type of plays they used most often had the following options: Lotto (including two additional Lotto type plays, Viking-Lotto and Extra), football tip (including a special three-game type of football tip), slot machines (fruit machines, gambling machines, etc.), lotteries, toto (horse-race wagering: V5 or Rikstoto), bingo, poker/casino, and other types.

\(^1\) A total of 4820 subjects were obtained by random-digit telephone dialing. Among them, 2014 completed a telephone interview, while 2806 did not. The number refusing to answer was 1364, and 1443 were unavailable. If wrong or unused numbers, or outside inclusion criteria, the subjects were not considered to be real noncompleters. A total of 607 fell into that category, thus we considered 2199 (2806 – 607) to be real dropouts, and 4213 (4820 – 607) to be the real sample approached. This gives a response rate of 47.8% (2014/4213).
2.3. Procedure

Respondents were interviewed over the telephone by trained interviewers employed by the Norwegian Gallup Institute, one of the largest social survey research companies in Norway. Up to eight call-backs were made to complete an interview. The data collection was performed in August–September 1997.

To give the present sample a representativity in relation to the general population, a weight index was calculated from Age × Sex × Geography (county). In Table 3, these weights were not used to get true values of the pathological gambling prevalence, while this may have decreased the representativity of the sample in this table. Some sums do not end equal because of the weighing procedure.

Table 1
Demographic data: gender, age, education, working status, and marital status over frequency of gambling with money (numbers and percent; N=2014)

<table>
<thead>
<tr>
<th></th>
<th>Never gambling</th>
<th>Sometimes (&lt;weekly)</th>
<th>Often (≥ weekly)</th>
<th>Problematic gambling</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Total</td>
<td>636</td>
<td>31.2</td>
<td>950</td>
<td>47.2</td>
<td>423</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>251</td>
<td>25.5</td>
<td>472</td>
<td>47.9</td>
<td>251</td>
</tr>
<tr>
<td>Women</td>
<td>385</td>
<td>37.3</td>
<td>461</td>
<td>44.7</td>
<td>183</td>
</tr>
<tr>
<td>Age group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18—30</td>
<td>143</td>
<td>29.2</td>
<td>245</td>
<td>50.1</td>
<td>91</td>
</tr>
<tr>
<td>31—50</td>
<td>196</td>
<td>26.1</td>
<td>384</td>
<td>51.0</td>
<td>169</td>
</tr>
<tr>
<td>Over 50</td>
<td>296</td>
<td>38.1</td>
<td>305</td>
<td>39.3</td>
<td>174</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>College/university</td>
<td>218</td>
<td>36.6</td>
<td>283</td>
<td>47.6</td>
<td>89</td>
</tr>
<tr>
<td>Senior high School</td>
<td>225</td>
<td>26.8</td>
<td>416</td>
<td>49.6</td>
<td>193</td>
</tr>
<tr>
<td>Ten-year school</td>
<td>187</td>
<td>32.4</td>
<td>234</td>
<td>40.6</td>
<td>153</td>
</tr>
<tr>
<td>Work situation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Working or student</td>
<td>380</td>
<td>27.6</td>
<td>678</td>
<td>49.3</td>
<td>307</td>
</tr>
<tr>
<td>Not working</td>
<td>252</td>
<td>39.6</td>
<td>253</td>
<td>39.8</td>
<td>128</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married/living together</td>
<td>388</td>
<td>29.5</td>
<td>638</td>
<td>48.5</td>
<td>283</td>
</tr>
<tr>
<td>Not married/living together</td>
<td>242</td>
<td>34.9</td>
<td>293</td>
<td>42.3</td>
<td>151</td>
</tr>
</tbody>
</table>

Missing values are less than 0.4% and are not indicated in the table. Problematic gambling includes both pathological gambling and at-risk gambling.
Product-moment correlations were computed between degree of gambling and some established risk factors. A chi-square test was also performed for the relation between smoking and gambling.

3. Results

3.1. Demographic data

There were 2014 respondents and the response rate was 47.8%. The sample consisted of 48.9% men and 51.2% women. The age was rather equally distributed when divided into three age groups (18–30, 31–50, over 50). Of the sample, 29.5% had university or college education, 41.7% reached senior high school, and 28.6% attended a 10-year (compulsory) school. Of the sample, 68.3% were in work, studies, or military service and 31.6% were not. Respondents married or living together comprised 65.3% while 34.4% were single (see Table 1).

3.2. Gambling frequency

There was a high frequency of never gambling (31.2%), a majority (47.2%) gambled sometimes, i.e., less than weekly, while some (21.0%) gambled often (see Table 1).

Men (25.5%) gambled more often than women (17.7%). The lowest frequency of gambling often was in the youngest age group (18.6%), while most of the problematic gamblers were found in that group as well.

3.3. Amount bet and type of gambling

In the sample, 0.5% bet over 50,000 NOK (roughly US$5000), 0.2% over 100,000 NOK, and 0.1% over 500,000 NOK. The majority (99.0%) were spending below 50,000 NOK yearly.

The different types of gambling are listed in rank sequence, with percentage of first choices of the total subjects playing (see Table 2). Lotto is the most popular game with 76.0%, followed by football tip (10.8%), slot machines (5.1%), and lotteries (4.9%). Of the cases of problematic gambling, four fall on slot machines (yielding 5.7%), four on Lotto (yielding 0.4%), one on lotteries (yielding 1.5%), and one on football tip (yielding 0.7%). Thus, for some types of plays, there was a discrepancy between rank for playing and for problematic playing. Slot machines gave higher problematic playing rank. So did "Extra" (under Lotto plays; 3.9%) and "Oddsen" (under football tip; 4.2%).

The four most frequently used plays were all connected to problematic gambling. Slot machines, with relatively low-frequent use (5.1%) had a high problematic gambling frequency (5.7%), while the most frequent play Lotto (76%), gave a low problematic gambling frequency (0.4%).
Table 2
Which types of gambling are played most (numbers and percent; N=2014)

<table>
<thead>
<tr>
<th></th>
<th>Sometimes (&lt;weekly)</th>
<th>Often (≥ weekly)</th>
<th>Problematic gambling</th>
<th>Total playing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Lotto/Viking-Lotto/Extra</td>
<td>695</td>
<td>66.4</td>
<td>348</td>
<td>33.2</td>
</tr>
<tr>
<td>Football tip</td>
<td>103</td>
<td>69.1</td>
<td>44</td>
<td>29.5</td>
</tr>
<tr>
<td>Slot machines</td>
<td>55</td>
<td>78.6</td>
<td>11</td>
<td>15.7</td>
</tr>
<tr>
<td>Lotteries</td>
<td>62</td>
<td>92.5</td>
<td>4</td>
<td>6.0</td>
</tr>
<tr>
<td>Toto</td>
<td>12</td>
<td>52.2</td>
<td>11</td>
<td>47.8</td>
</tr>
<tr>
<td>Bingo</td>
<td>7</td>
<td>77.8</td>
<td>2</td>
<td>22.2</td>
</tr>
<tr>
<td>Poker/Casino</td>
<td>5</td>
<td>100.0</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Other</td>
<td>6</td>
<td>75.0</td>
<td>1</td>
<td>12.5</td>
</tr>
<tr>
<td>Total</td>
<td>945</td>
<td>68.6</td>
<td>421</td>
<td>30.6</td>
</tr>
</tbody>
</table>

Missing values are less than 0.6% and are not indicated in the table.
Problematic gambling includes both pathological gambling and at-risk gambling.
* 31.6% (636) never gamble.

3.4. Pathological gambling and at-risk gambling

Table 3 shows the differential results for pathological gambling and at-risk gambling by gender and by age. In total, the percentage for young men (18–30) is very high (3.15%) compared to men over 30 (0.28%) and females 18–30 (0.84%) and over 30 (0.12%). The total problematic gambling frequency was 1.97 for 18–30 years and 0.19% over 30. There were no problematic gamblers over 50 in the material, so we only used two age groups.

Of the DSM-IV criteria, the first one (preoccupied with gambling) seems to be filled out most (3.1% of the total population). Other criteria giving relatively high responses are Numbers 2 (needs to gamble with increasing amounts; 2.4%), 5 (a way of escaping from problems; 2.1), and 6 (returning and “chasing”; 2.1%). No one has affirmed Number 9 (lost significant).

Table 3
Pathological gambling and “at-risk gambling” over gender and age groups (number and percent)

<table>
<thead>
<tr>
<th>Age</th>
<th>Total (N=2014)</th>
<th>Men (n = 948)</th>
<th>Women (n = 1066)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pathological gambling</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>18–30</td>
<td>2</td>
<td>0.44</td>
<td>7</td>
</tr>
<tr>
<td>31–</td>
<td>1</td>
<td>0.06</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>3</td>
<td>0.15</td>
<td>9</td>
</tr>
</tbody>
</table>

This table is based on unweighted data to ensure true data in exchange of the risk of being less representative.
3.5. Risk factors

There were rather low but significant correlations between degree of gambling and most of the established risk factors: sex (male) .142 ($P < .01$), age (low) .094 ($P < .05$), education (low) .081 ($P < .05$). Employment (unemployed), however, showed a negative correlation, $- .095$ ($P < .01$), while civil status was not correlated (compare Volberg, 1996).

Among the subjects who never gambled, 25.8% were daily smokers; among those who gambled weekly, 44.1% were daily smokers; and among those with problematic gambling, 66.7% were daily smokers. Thus, there was a clear relation between smoking and gambling ($\chi^2 = 41.01$, $P < .001$).

4. Discussion

The main findings showed that 68.8% of the normal population was gambling. Lotto was the most popular type of game with 76.0%, followed by football tip (10.8%), slot machines (5.1%), and lotteries (4.9%). A mean of 0.15% were pathological gamblers, according to the DSM-IV, and an additional 0.45% were "at-risk gamblers." The prevalence was higher in lower age, and with males (with 3.15% males 18–30 years being problematic gamblers). Of the problematic gamblers, four have slot machines as first choice (yielding 5.7% problematic gamblers), and four have Lotto (yielding 0.4%). For some types of plays, there was a discrepancy in rank for playing, and for problematic playing. Slot machines gave higher, while Lotto and football tip gave lower problematic playing rank. The fact that slot machines seem to be more problematic is in accordance with earlier reports of preferences for continuous forms of gambling (gambling activities where winnings can be immediately reinforcing), where nearly three-quarters of pathological gamblers reported participating regularly (once a week or more) in continuous forms whereas respondents scoring in the no-problem range on the DSM were more likely to be either regular participants in noncontinuous varieties or do not engage in any form of gambling on a regular basis (Volberg & Abbott, 1994).

The DSM-IV with its 10 questions gives a more conservative estimate of pathological gambling and fewer false-positives, as compared to SOGS, which has more items (see Beaudoin & Cox, 1999; Derevensky & Gupta, 2000) and gives high false-positive rates in studies of the general population (Ladouceur et al., 2000). DSM-IV therefore represents a conservative measure, and we conclude that the level of pathological and at-risk gambling in Norway is at the same level as in most other places (around 1%).

A rapid expansion of legalized gambling (Götestam, 1993) has been shown to result in an increase in pathological gambling (Ladouceur, Boudreau, Jacques, & Vitaro, 1999). This produces tremendous demands for information about the number and characteristics of pathological gamblers and at-risk gamblers in the general population to enable treatment and prevention of the probable increases in pathological gambling in the future. This presents a specific problem for youthful gamblers and males. The nonresponse rates for telephone surveys have also increased in recent years and pose a particular problem for researchers investigating sensitive topics such as gambling (Volberg, 1996).
This is the first Norwegian study of the prevalence of gambling problems and pathological gambling. There are some weaknesses with the present study. The low response rate (47.8%) is threatening the representativity of the study, while there were as much as 2014 respondents. The interview was made by telephone in this cross-sectional study. Further and prospective studies should be implemented to increase the knowledge in this important area of research.

The study of pathological gambling is also of great importance in principle, as it is the only type of addiction where the addictive behavior is not confounded by a drug and its effects. It has sometimes been called "the pure addictive disorder."

Acknowledgements

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References


Paper II
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Paper V
Agneta Johansson & K Gunnar Götestam

RISK FACTORS FOR PROBLEMATIC GAMBLING: A CRITICAL LITERATURE REVIEW
Contents

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0. SUMMARY

After an introduction describing the review approach, referring to earlier critical reviews and some other publications, follows a review of experimental studies (28 studies) with empirical calculations of risks (not including epidemiological prevalence studies, and treatment studies). The main part of the paper is a critical review of risk factors, categorized with 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 critical review unfolded very few “well-established risk factors for PG”, with more than two studies to support the conclusions. 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 areas (22) could be concluded to be probable risk factors (1-2 supporting studies). There are unclear relations between PG and some possible factors of importance for the development of PG (among them physiological/biological factors).

Key words: pathological gambling, risk factors, experimental studies, significance
A. INTRODUCTION

Plays and games for children and adults are as old as culture itself. Playing can take over, become excessive, and develop in a harmful direction, which has been known for a couple of centuries, but problems with pathological gambling has not been much recognized until recently. Public statistics from different countries have shown that available plays and games have more than doubled over ten year periods (cp Götestam, 1993). Jacobs (2000) also states an increase in serious gambling-related problems during the last 15 years.

The level of occurrence of pathological gambling is around 1-2% in the US, Canada, and Europe, around 3% in New Zealand, and around 7% in Australia (Ladouceur & Walker, 1996).

Research into these problems started about 20 years ago, and during the last three years there has been a healthy development of gambling research, into many areas of gambling problems, and with many researchers involved. No specific breakthrough seems to have emerged, but substantial confirmations of earlier knowledge has ben ascertained. Still there is a lack of prospective studies, and more causally directed research. The research has mainly been correlational in character.

In lack of rigid empirical research, many opinions prevail, and preventive actions become delayed. The aim with the present study, is to organize the empirical facts on risk factors for the development and maintenance of pathological gambling, to enable adequate preventive actions, to reduce the extent and seriousness of the problem.

In research on mechanisms and risk factors for development of pathological gambling, researechers have studied both risk-populations and normal populations to investigate the relationships between variables. The search for one main mechanism or risk factor will be futile. What we need is a more collaborative approach, to this manyfolded area, to be able to make more focused preventive actions, and develop treatment programs (see for instance Volberg et al., 1996).

Other researchers have focused on gamblers from the clinical settings. Simultaneous investigations of both gaming frequency and clinical problems and pathological gambling has been relatively rare. Other studies have investigated subgroups of gamblers.

According to Griffiths (1993a), gambling is a behavior that is likely to lie on a continuum from those who do not gamble at all to those who gamble excessively.
It is important that gambling research retains both naturalistic and laboratory work. Naturalistic studies are helpful in identifying specific and new concepts. Laboratory studies can develop appropriate conditions for investigating the specific aspects that will allow new hypotheses to be tested (Sharpe 2002).

1 Review approach

In looking through the literature, although empirically founded critical reviews are almost non-existent, there are some publications with some form of overview. We have found five such studies (Shaffer & Hall, 1996; Spunt et al., 1998; Wardman, el-Guebaly, & Hodgins 2001; Raylu & Oei, 2002; and Walthers, 2002), which will be described below. We have also found a review of treatment studies (Petry & Armentano, 1999), but they are mainly uncontrolled. As the quality of most of them as well as their relevance in this connection is doubtful, we have not included treatment studies in the present review. Oakley-Browne et al (2003) found 17 treatment studies, of which only four fulfilled RCT criteria.

Eber and Shaffer (2000) quantified citations during the period 1964-1999, showing the first reference in 1964, followed by a low level and a dramatic increase from 1985. We thus searched the literature bases Medline and PsycLit for the period 1985 to 2002. In addition we went through all main general psychiatric and psychological journals and specifically addictive behaviour and research journals, where gambling research usually is reported. We found 382 articles (100 in the period 2000-2002) published in international journals.

Criteria for inclusion of publications (partly based on the research synthesis by Shaffer et al., 1999) in the following review was that (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, by of Nathan and Gorman (1998) in classifying the risk factors into three levels: (1) well-established risk factors for PG: several (≥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 so far documented as a risk factor. This also applies to analogue studies with no clinical or real-life applications. 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.

Epidemiological studies (around 1999 there existed over 150; Shaffer et al., 1999) solely aiming at prevalence rates, and not focusing on risk factors were considered of separate interest for further studies.

In the following text “gambling machines” is used for the different terms fruit machines, slot machines, and electromagnetic machines. As the concept “probable pathological gambling” (Lesieur and Blume, 1987) in most texts is equal to pathological gambling (PG), we have used the latter 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 has strived to make the presentation of data very restricted, with only clearly important facts presented in some detail.

2 Earlier critical reviews

Shaffer and Hall (1996) completed a meta-analysis on nine nonduplicative 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 & Blume, 1987), SOGS-RA (Winters et al. (1993a), MAGS (Shaffer et al., 1994), and GA (Gamblers Anonymous twenty questions criteria). The meta-analysis showed reported PG prevalences from 3.5% to 8.5% (with a 95% confidence interval 4.4% to 7.4%).

Shaffer and Hall (1996) also 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 study is not a systematic categorizing review, goes through a couple of a number of studies, and thereby it shows some new angles of the interface between PG and substance misuse.

Wardman, el-Guebaly, and Hodgins (2001) reviewed nine empirical studies, for prevalences of problem gambling and PG, and factors statistically associated with Aboriginal population problem gambling behaviour. There were four adolescence studies, of which three (n=1423)
had only prevalence of problem gambling (from 10.1% to 21%), and the fourth one (n=122700), only had data on general gambling activity (no SOGS or DSM criteria). There were also five adult Aboriginal studies (n=3827), where the prevalence of PG was from 6.6% to 22%. Comparisons of prevalence of PG was made between Aboriginal and non-Aboriginal populations, giving odds ratios from 4.14 to 15.69. In one study both SOGS (Lesieur & Blume, 1987) and DSM-III-R (APA, 1987) criteria were used, and the SOGS criteria gave about threefold prevalence values compared to DSM-III-R criteria.

The authors (Wardman, el-Guebaly, & Hodgins, 2001) also looked at factors for adult PG, and found that pathological gamblers compared to non-problem gamblers had a greater gambling involvement, made greater financial expenditure, and were younger. In comparison between Aboriginal and non-Aboriginal gamblers, Aboriginals more often were female, had incomplete high school, were not married, and had an income of less than $25,000.

Two reviews arrived while we were working with the present manuscript: A comprehensive review of many studies of pathological gambling was performed by Raylu and Oei (2002). They discussed different factors implicated in the development and maintenance of PG (which is the aim of the present review). These were three main rather diverse categories: familial (including learning/genetics), sociological, and individual (including personality, biological/biochemistry [hemispheric dysregulation, neurotransmitters; arousal], cognitions, and psychological states). These rather awkward dimensions makes it difficult to read the message. There was no detailed evaluation or conclusion, only an elaborated discussion of studies. Their aim was to make a review to more clearly view the need for actions against PG and problematic gambling. In their suggestions for future research, they pointed at differences in findings from different forms of gambling (PG is not a single phenomenon), more emphasis on methodological problems, and rather studies on why people stop gambling, than why they start. They also point at the sole Western-based approach usually found in the literature.

Walters (2002) made 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 twin on the gene-gambling relationship. The familial effect was clearest for the relation between sons and problem gambling fathers. They also included moderating variables (sex, nationality, family, severity) in their analysis.

3 Specific non-empirical and theoretical publications
Lesieur and Custer (1981) defined different phases of the PG career: the winning phase, the loosing phase and the desperation phase.

Brown (1986) has elaborated a theoretical model, based on his own and others research on arousal and sensation seeking behaviour, and ends up with an eclectic model, including psychophysiological arousal, cognitive variables, affective factors, behaviourial 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. Brown also indicates important future research venues, as smaller studies to identify specific variables, a large-scale prospective multiple-regression study, detailed smaller studies following the large-scale one, and identification of special groups or “subtypes” of addictive gamblers.

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”.

Griffiths (1995, 1996) overview of PG in adolescents, with many references, is a good source for knowledge about gambling and PG. Griffiths and Wood (2000) represent an updating of earlier accounts (Griffiths, 1995, 1996, 1999) of problematic and pathological gambling, extended to the areas of videogame playing and Internet use. A useful list of risk factors is provided, but is in this connection mainly undocumented. There are also useful suggestions for further research.

Sharpe (2002) formulates a biopsychosocial perspective with a cognitive-behavioural model of problem gambling. Among vulnerability factors she points at ADHD (Attention Deficit Hyperactivity Disorder) and impulsivity as clear vulnerability factors, supporting the diagnostic placement of PG as an impulse control disorder. Furthermore she points at availability, biological markers implicating neurotransmitter systems (D2, NA, MAO), wellknown to be involved in depression and obsessive-compulsive disorder. She also points at the relation between gambling and drinking, also resulting in loss of control in gambling. Furthermore 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
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discuss how to proceed from research to treatment, and casuistically describe the treatment procedure. In a further elaboration Dickson, Derevensky and Gupta (2002), present a conceptual framework with a 16-page table, containing a very good review with risk and protective factors and corresponding prevention findings for adolescent substance use and abuse. With the alcohol area 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.

B. Review of risk factors

Attempts to study risk factors, not meeting our quality criteria might result in 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.

<Table 1 in here>

To give a summary of results, mainly to give an impression about how little real information we have, Table 2 is compiled as a summary, showing how many empirical studies have been performed on each risk factor, and conclusions about which are the well-established and the probable or potential risk factors.

<Table 2 in here>

1. Demographics

Demographic variables have often been mentioned to be of importance as risk factors, but they have much more seldom been empirically tested.

1.1 Age: Age is a typical risk factor, affecting many areas of our daily actions, so also in the area of PG. There are four studies of age as a risk factor.

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

Bondolfi et al. (2000) in a telephone interview prevalence study (n=2526) on gambling in
Italy, used SOGS as diagnostic instrument, to analyse different risk factors in a correlational
design. The results showed that being under the age of 29 was a risk factor ($X^2=17.1$, p=.01).

Potenza et al. (2001) used a gambling helpline (n=562 calls) to identify different
characteristics related to gambling. Logistic analyses were performed on the data collected in a
two-year period. Age (younger) was identified as a significant risk factor ($X^2=25.29$, p<.0001).

Volberg et al (2001), performed a large prevalence study with SOGS-R (and DSM-IV) on
a Swedish normal population (n=8845). Life-time problem and pathological gamblers were
compared to non-problem gamblers in different variables, with the establishment of Odds
Ratios. Age (lower) was shown to be a risk factor (OR=2.51, p=.000).

Taking the four studies of age as a risk factor, we can state that age is a well-established
risk factors for PG.

1.2 Gender: Gender is also a typical risk factor, affecting many areas of our daily actions.
There are five studies of gender as a risk factor.

In a study by Winters et al. (1993b) a 15-18 year old sample (n=702) was interviewed,
using 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). In their study, they compared higher degrees of
problem gambling (pathological gambling and at risk gambling) to different variables. Regards
gender there was no comparison made on gender in relation to problematic gambling, thus the
reference is not included in the table on gender.

In the study by Ladouceur et al. (1999a) on 3426 high school students, a univariate
ANOVA on SOGS and 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), and found 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.
There was a significant relation between problem gambling and gender ($r_{xy}=0.12$, p=.04).

In the study by Bondolfi et al. (2000) also male gender was shown to be a risk factor
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In the Volberg et al (2001) study male gender was 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, there is a clear support for the notion that gender is a well-established risk factor for PG. There are indications (for instance in Wardman, el-Guebaly, & Hodgins, 2001) that females are at higher risk than men in aboriginals, but so far this has not been replicated.

1.3 Education: In the Volberg et al (2001) study education (lower) was shown to be a risk factor for gambling problems ($X^2=12.10$, p=.002).

Only one study has studied the education factor empirically, why we consider education as a probable risk factor for PG.

1.4 Marital status: In the study by Bondolfi et al. (2000) also marital status (married) was shown to be a risk factor ($X^2=7.52$, p=.02).

In the Volberg et al (2001) study marital status (unmarried) was shown to be 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, so we are unable to make any conclusion here.

1.5 Income: In the study by Bondolfi et al. (2000) (higher) income was shown to be a risk factor ($X^2=10.88$, p=.01).

The helpline study by Potenza et al. (2001) reported financial problems was identified as a significant risk factor ($X^2=4.21$, p<.04).

Only two studies have been directed towards income and financial problems empirically, but with contradicting results. We are therefore unable to make any conclusion.

1.6 Employment: In the study by Feigelman et al. (1995), there was a significant relation between unemployment status in 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 DIS (Mental Health Diagnostic Interview Schedule) to reach a DSM-III-R diagnosis of PG, and ASI (Addiction Severity Index) for sociodemographic and other information. Of the 313 patients 25 (8.0%) fulfilled the criteria for PG. There was a clearly significant relation between unemployment status and PG ($t=11.09$, p<.001).

Only two studies have been directed towards employment empirically, thus, we consider employment status as a probable risk factor for PG.
1.7 Social welfare status: In the Volberg et al (2001) study, on social welfare was shown to be a risk factor for gambling problems ($z=2.41$, $p=.05$).

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

1.8 Residence: In the Volberg et al (2001) study residence (big cities) 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.

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

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

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

1.10 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 Volberg et al (2001) study being born outside the country was shown to be a risk factor for gambling problems ($OR=2.08$, $p=.01$).

Only two studies have been directed towards immigrants and ethnic groups empirically, thus, we consider immigration and ethnic groups as probable risk factors for PG.

2. Physiological and biological factors

2.1 Heart rate and arousal: Anderson and Brown (1984) used a laboratory setting, with 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 bet staked and HR increase was significant ($r_{xy}=.741$, $p<.0005$, one-tailed). The student group ($n=12$) did not differ from the gamblers in their reactions to the artificial casino.

A second empirical study was performed by Leary and Dickerson (1985) who followed high- and low ($n=22/22$) frequency players with HR during playing. Playing was significantly
associated with increases in arousal in both groups, but 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. On 12 problem poker machine players and 13 horse race
gamblers, they 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 (study not included in table here).

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

Griffiths (1995) has reviewed the literature on this field (see Griffiths, 1995, Table 1.4, p
18) and it seems as further reports on increased heart rate in relation to plying among gamblers
compared to non-gamblers are weak. An empirical support for such a notion seems to be too
week to qualify more reporting.

2.2 Transmitter activity: Bergh et al (1997) studied monoamines and their metabolites in
CSF (cerebrospinal fluid) 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), an increase in 3,4-dihydroxyphenylacetic acid (DOPAC) and in
homovanillic acid (HVA). The ratio DOPAC/DA and HVA/DA were significantly different as
well. Noradrenaline (NA) and its metabolite MHPG were increased, whereas 5-HT and 5-
HIAA were unchanged. The studies are performed with current methodology of high
standard. The problem is the unclear connection between transmitters and PG, which needs
further clarification.

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. The study is performed with high
standard current methodology. The importance of the findings is still somewhat unclear.

Blanco et al (1996) studied platelet MAO activity on 27 male PG, with matched controls,
with Jackmans procedure. They found that MAO activity was lower in PG than in controls
(p<.01, Wilcoxon matched-pairs signed rank test). The study technique is of high standard,
while the meaning and importance of the results remains unclear.
Three studies have been performed to elucidate transmitter activity in CSF in relation to PG. Several transmitters have been studied, with somewhat inconsistent results. One study showed increased MHPG, while another showed lower levels, among gamblers. The results in detail seems difficult to interpret, and even more so in the third study. Transmitters may still be probable markers for PG risk factors.

2.3 Genetic studies: The dopamine D_{2}A1 allele is usually connected to substance abuse. Comings et al (1996) studied the presence of the dopamine (DA) D_{2} receptor in a PG sample (222) without drug addiction, and a control sample (714). On a group of PG (22) and controls (23), they showed a significantly different occurrence of D_{2}A1 allele (OR=5.03). The studies are performed with usual methodology of genotyping of high standards. The problem is the sofar unclear connection between transmitters and PG, which needs further investigation.

Ibáñez et al. (2001) studied 69 consecutive PG applying for treatment for PG. They used SOGS for diagnosis of PG, and the full DSM-IV clinical interview (both Axis I and II). In addition they assessed depression (BDI), and some other assessments. 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 comorbidity (X^{2}=13.9, p=.003), and the allele DRD_{2} C4 allele was present in 42% with comorbid disorders, compared to 5% without (X^{2}=7.0, p=.008). The association between the DRD_{2} allele distribution and comorbid PG and other psychiatric disorders supports the role of this gene as a liability factor for psychiatric disorders.

Only two genetic studies have been performed, thus, we consider genetic studies as probable instruments for assessment of PG risk factors.

3. Cognitive distortions

Toneatto (1999) makes a review of the gambling-related cognitive psychopathology. Magnification of gambling skill, means that the gambler has an exaggerated self-confidence, in contrast to the severity of losses. Superstitious beliefs are characterized by the belief that gambling outcomes are result of chance, and the simultaneous belief that they have reliable means of manipulating such outcomes in their favour. Subcategories are talismanic superstitions that a certain object increases winning probability, behavioural superstitions that some rituals can increase winning, cognitive superstitions that certain mental states can influence winning. Interpretive biases have several subcategories: attributional bias, a tendency
to overestimate dispositional factors (skills, abilities), and underestimate situational factors (luck, probability); Gambler’s fallacy means that a series of losses is expected to be compensated for. Chasing means the gambler believes that it is possible to recover the loss. Anthropomorphism is the tendency to attribute human characteristics to gambling objects (gambling machines, lottery card, bingo card, horses). Learning from losses, means reframing losses as learning experiences. Temporal telescoping means that the gambler believes that wins are actually nearer temporally, than farther. Selective memory, means recalling wins, especially large ones, and difficulty recalling losses.

Predictive skill means that the gambler recognizes certain cues as predictors to winning, although these predictions do not work. Illusion of control over luck has several subcategories: Luck as uncontrollable or controllable variable, means that some gamblers believe that luck oscillates between periods of good and bad luck, and cannot be manipulated directly. Many gamblers believe that luck can be manipulated in their favor. Many gamblers believe that luck is a trait variable, and that they have special luck with certain games. Some gamblers believe that luck is contagious from other areas of life, so good luck in other areas will predict good luck in games. Illusory correlation means that gamblers often perceive illusory correlations and assign causality to features of the environment.

Few studies has approached the problem of cognitive distortions empirically, but there are some exceptions.

3.1 Erroneous perception or biased evaluations: Gilovich (1983) in a series of experiments investigated factors leading to persistence of gambling, in spite of loosing more than winning. He pointed at the biased evaluations of wins and losses. 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 their memories a week ago, and reported correctly 47% of the losses, but only 27% of wins (t=2.33, p<.05). In a third study (with 49 students and staff members) the experimenter manipulated the information in relation to bets on sports. A 2x2 factorial design was used, with outcome crossed with salience of a fluke play. The results showed a main effect for outcome (F=4.31, p<.05), supporting the conclusions about biased evaluations. The author discusses the old memory studies by Ziegarnik and the effect named after her (third experiment not reported in table).

Savoie and Ladouceur (1995) studied erroneous perceptions in two studies. The question
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 eventually to modify their playing customs.

In their first study 100 Ss regularly participating in a lottery (53M/47F), and 100 (42M/58F), participating occasionally were included. In a short interview their superstitious habits and preferences about lottery, choosing numbers, etc, was investigated. They were also asked about participation in other plays. The probability of winning was estimated higher among 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 players on a lottery were approached with a short questionnaire about playing habits, their concepts about lotteries, participation frequency, number of lots bought, strategies in choosing the numbers, confidence degree, irregular preferences, and gains from the lottery. They were randomly assigned to experimental and control groups (n=22/22). They were offered to compare the exactness of their concepts with the true result probabilities. A month later, they got the same questionnaire again. The results showed that the regular players had more erroneous perceptions. The experimental group had become less confident to win than earlier ($F=7.38$, $p<.025$), while the control group had reduced their playing activity during the time ($t=3.2$, $p<.025$).

Four experimental studies were reported, on biased evaluations on student Ss or Ss playing on lotteries, and not PG Ss. The first two studies are difficult to interpret. The two last studies show a potential to change habits, also in lottery frequency. The tentative conclusion is that erroneous perceptions has an importance in the development of PG.

No experiments on superstitious beliefs have been found in the literature search, except the first experiment by Savoie and Ladouceur (1995), discussed above, as an example of erroneous perception or biased evaluations.

**3.2 Illusion of control:** The illusion of control was studied by Langer (1975). The definition of “illusion of control” was an expectancy of a personal success probability inappropriately higher than the objective probability would warrant. It was predicted that factors from skill situations introduced into chance situations cause individuals to feel inappropriately confident. In a series of six experiments effects on illusion of control by competition, choice, familiarity, active involvement, was studied. In the first, 36 students
participated, to study experimentally manipulated bias (confidence/shyness). The hypothesis that this affected the results was confirmed (t=5.46, p<.005). In a second experiments, 47 males and 6 females participated, where choice was manipulated. This also had an effect on outcome (t=4.33 p<.005). In a third experiment, 13 Ss participated, getting stimulus familiarity manipulated, also with significant effect (t=5.46, p<.005). In a fourth experiment, where involvement was manipulated, a significant effect was shown (F=7.33, p<.01). In the two last experiments, one open and one controlled in a field setting (n=49F/73M Ss, 31M/10F Ss), also got significant results (F=7.58, p<.01; X²=4.19, p<.05). It seems like subjects do not distinguish chance from skill-determined events, but there are other factors, which act as if they make the situation controllable, even in pure chance situations (lottery). The subjects acted as they had an illusion of control over outcome, and they even gave up the opportunity to exert real control. It was not the intrinsic value of a ticket, but rather the sentimental value, that counted and gave it worth.

In a second study Langer and Roth (1975) performed an experiment to find the development of illusion of control (90 stud). 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 overremembered past successes, and expected more future successes.

In two experiments, Gilovich and Douglas (1986) studied the development of illusion of control (n=80 stud, 36 Ss). They showed that evaluations of randomly determined gambling outcomes were biased (F=10.72, p<.001). Those who had lost a first bet, were more apt to greater impact on subsequent bets (compared to those who had won). The losers appeared to use manipulated fluke events to explain away the outcome, whereas winners discounted their significance. In a second experiment (n=160 stud), the outcomes were shown to be biased in randomly determined gambling outcomes (t=2.56, p<.02). By inducing an illusion of control by allowing subjects to make a series of choices (win/loss, fluke/no fluke, illusion of control/no control), the limiting conditions for the phenomenon were tested. Subjects were induced to perceive an “illusion of control”, and the fluke 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 a strong evidence that illusion of control is a phenomenon that could interact with the development of PG. Nine experiments have been performed on normal Ss (students and company employers), but none on PG. A tentative conclusion is that illusion of control is an important factor for the development of PG.

4. Varia

4.1 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 & Lester, 1999; Walker, 1992).

Ladouceur et al. (1999b) tested directly 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 prevalence second time, showed an increase by 75% in the number of pathological gamblers.

When there are few restrictions in the placements of gambling machines, so that the general availability increases, increases in pathological gambling could be expected.

In Australia there has been a particular increase in availability of gambling activities in venues that also provide alcohol. According to Sharpe (2002) Casinos in some US locations (e.g. Reno and Las Vegas), offer consumers free alcohol while they are engaged in gambling. Overlap between drinking and gambling behavior is problematic as drinking alcohol beverages is likely to reduce self control, and thus increase gambling behaviour.

Three studies have been performed on availability as a risk factor for PG, and thus availability is regarded as a well-established risk factor for PG.

4.2 Parent playing: In the study by Winters et al. (1993b), a significant relation was shown between problem gambling and reports of one or both parents gambling ($X^2=21.4$, $p<.001$), indicating more problem gambling among those whos parents gambled.

The study mentioned above (Ladouceur et al., 1999a) also showed a relation between SOGS and parents playing ($X^2=48.12$, $p<.001$).

Only two studies have been directed towards parents playing empirically, thus, we consider parental playing as a probable risk factor for PG.

4.3 Sensory characteristics: Loba et al. (2001) studied the effect of sensory manipulations (fast speed/sound, slow speed/no sound, counter present), to look at subjective selfreported differences in reaction. They used video lottery terminals (VLT), a “continuous”
form of gambling, where time between wager and payout is short. Ss were 60 (22F/38M) regular (playing at least twice a month) VLT players, recruited via advertisements. They used SOGS to assess PG, and used a survey of subjective reactions to VLT manipulations. The experimental conditions were either a video poker game, or a 20 min spinning reels game. PG (29) or non-PG (31) Ss were randomly assigned to one of the conditions. The game versions had varied sensory characteristics (i.e. slow/no sound, fast/sound, control, counter present). The results showed that non-PG were bothered by fast speed and sound, while PG 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), bothered (F=11.29, p<.001). To attain an aim to reduce the risk of abuse of VLTs by PGs, the sensory features manipulations are of importance. A decreased speed and turning off the sound decreased ratings of enjoyment, excitement, and tension reduction for PG relative to non-PG, but only at fast speed with sound facilitated to stop playing PG. The study supports Griffiths (1993b) notion that sensory characteristics is of importance for the development of PG. The authors state that it is important to replicate findings in a more naturalistic setting.

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

4.4 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 just emitted. If negative, it could have a punishing effect. A continuous reinforcement (reward in each trial) is easy to brake. It is just to withdraw the reinforcer, and the behaviour will 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. Games to large extent build on the principles of reinforcement, mainly reinforcement at random, or in a combination with certain skills. The payout interval, and some structural characteristics, are of importance in the
development of PG. The operant psychology, with the reinforcement principles, Skinner built on Thorndike’s Law of effect (Skinner, 1953, p 59 ff). He has also analyzed gambling behaviour with the operant approach (ibid., p 396 ff): “Gambling devices make an effective use of conditioned reinforcers which are set up by pairing certain stimuli with the economic reinforcers which occasionally appear”. “The efficacy of such schedules in generating high rates has long been known to the proprietors of gambling establishments. Slot machines, roulette wheels, dice cages, horse races, and so on pay off on a schedule of variable-ratio reinforcement. Each device has its own auxiliary reinforcers, but the schedule is the important characteristic” (Skinner, 1953, p 104). The operant model or paradigm has become very important in many different areas of research and application. Honig (1966) edited a large volume covering many of these areas, and showing the vast importance of the operant model. This approach is supported by Griffiths (1993), and several other authors in the field.

Early wins induces a skill orientation (Gilovitch, 1983), and reinforces the subject to try again, even if the contingency schedule is rather intermittent, then “next will be a win”. In the same vein big wins on gambling can also be analyzed with the operant model. The concept of near miss, has been forwarded by Griffiths (1991), and is also in accordance with the operant principles.

Skinner himself did not rely on statistical probability testing, he rather designed straight-forward 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 results.

There is a vast body of research of the usefulness and importance of the operant model. The conclusion is that this model can improve the understanding of the mechanisms of development of PG.

4.5 Age of onset: In the study by Bondolfi et al. (2000) also age of onset (before age 21) was shown to be a risk factor ($X^2=10.17$, $p=.01$).

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 have been performed on age of onset, thus, we consider age of onset as a probable PG risk factor.

4.6 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 PGs, distributed as “machine PGs”, or “traditional PGs”. SOGS, and DSM-IV diagnoses were assessed. Comorbid substance use disorder, or depressive disorder, was associated with the latency of PG-onset, but for the main results, social, environmental, and stimulus features were implicated. 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 longer latency 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.

4.7 Playing duration: In the study by Potenza et al. (2001) using a helpline the duration of gambling was identified as a significant risk factor ($X^2=8.00$, p<.005).

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

5. Comorbidity and concurrent symptoms

5.1 Depression: In the 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) there were more depression (Beck Depression Inventory) in the more serious PGs (t=3.4, p=.0001).

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

5.2 Suicidality: The study mentioned above (Ladouceur et al., 1999a) also showed a relation between SOGS and suicide ideation ($X^2=19.82$, p<.001) and suicide attempts ($X^2=60.26$, p<.001).

In the helpline study by Potenza et al. (2001) suicide attempts was identified as a significant risk factor ($X^2=4.19$, p<.05).

In an interesting study by Petry and Kiluk (2002) a group of PG (n=228) were studied in relation to suicidality. They do not, however, use PG as a dependent variable, so no conclusions on suicidality as a risk factor can be drawn (not included in table).

With only two studies on suicidality, we consider suicidality as a probable PG risk factor.

5.3 Anxiety: In the study by Ibáñez et al. (2001) they also included trait anxiety (STAI-T), and showed an increase of trait anxiety among the more severe PG (t=2.0, =.05).
With only one study performed on anxiety, we consider anxiety as a probable PG risk factor.

**5.4 OCD:** Frost et al. (2001) studied the relation between OCD symptoms and PG, diagnosed through SOGS, OCD symptoms by YBOCS (Yale-Brown Obsessive Compulsive Scale), and a hoarding scale. They advertised about the study, and got 89 participants (48F/41M). Thirty-six participants met the 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 OCD, we consider OCD as a well-established risk factor for PG. This connection also supports the close relation between obsessions and obsessive gambling.

**5.5 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 last month ($r_{xy}=0.14, p=.02$) was significantly related to problem gambling.

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

With only two studies on alcoholism, we consider alcoholism as a probable PG risk factor.

**5.6 Other drugs:** In the study by Feigelman et al. (1995), there was a significant relation between problem gambling and major drug problem 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 relation between SOGS and cigarette smoking ($F=20.42, p<.0001$).

In the study by Winters et al. (1993b), more often occuring 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 ($X^2=5.66, p<.02$).

With five studies performed on different drugs, we consider drug abuse as a well-established PG risk factor.

**5.7 Personality disorder:** In a study of Slutske et al. (2001) 7,869 men from 4,497 twin
Risk factors for PG

pairs (Vietnam Era Twin Registry) were diagnosed with DSM-III-R for PG, with DIS for antisocial behavior disorders. Telephone interview were conducted to ascertain PG and antisocial behavior disorders. The results showed elevated prevalence of antisocial disorders among individuals with a history of PG (OR=6.4), and similarly elevated for CD (OR=3.6) and for AAB (OR=6.1).

In the study by Ibáñez et al. (2001) there were more personality disorders in the more severe PG (t=3.0, p=.004), and the relation between PG and comorbid disorders was linearly related (r_{xy}=.40, p<.001).

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

6. Personality symptoms and characteristics

6.1 Coping styles: Getty et al. (2000) studied a group of members of Gamblers Anonymous (n=30), and a matched control group (n=30). The PG diagnosis was made by SOGS. In addition Problem-Focused Styles of Coping Inventory (PF-SOC) was assessed, as well as depression (BDI). The results showed a main effect on the group variable (F=8.35, p<.001). All types of coping styles, suppressive, reactive and reflective were significantly different between the experimental and control group, the experimental group being higher on suppressive (F=13.81, p<.001) and reactive (F=16.22, p<.001), and lower on reflective coping styles (F=7.81, p<.007). The authors conclude that a treatment program including engagement in active, reflective, planful coping when stressors arise would be helpful.

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

6.2 Impulsivity: Vitaro et al. (1997) studied impulsivity among 754 adolescent boys with Eysenck Impulsiveness Scale (EIS), and as teacher rated impulsivity. PG was assessed with SOGS. There was a clear relation between high PG and high impulsivity, both according to the EIS (X^2=30.58, p<.01), and as teacher rated (X^2=27.95, p<.01).

Langewisch & Frisch (1998), studied impulsivity and sensation seeking in PG, with a later correction of the data for sensation seeking. They assessed 144 undergraduate students with SOGS for PG diagnosis, several instruments for impulsivity, and the Sensation Seeking Scale. Their material contained 22.9% PG, and the high PG group was significantly higher on impulsivity than the rest of subjects (t=3.00, p=.003).
With only two studies performed on impulsivity, we consider impulsivity as a probable PG risk factor.

6.3 Hyperactivity (ADHD): Carlton and Manowicz (1994) found that adult pathological gamblers had a higher than average rate 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 attention deficit hyperactivity. The concept of impulsivity and its relationship to gambling problems, therefore seems of importance. The study does not include significance evaluations and is therefore not mentioned in the table.

6.4 Sensation seeking: Anderson and Brown (1984) showed significant differences between artificial and real casino for regular gamblers (n=12) if one looked upon correlations between bet size and different subscales on the Sensation Seeking Scale (Experience seeking: -.25/.54; Disinhibition: -.06/.57; both p <.05). The value of this study is weakened as the subjects studied are regular gamblers.

Blanco et al. (1996) investigated 27 PG and 27 matched controls and showed significant differences between PG and matched controls (WMP=Wilcoxon Matched Pairs) on the Sensation Seeking Scales (subscale Thrill and Adventure seeking, both p <.03; and Disinhibition, p<.01).

After scrutinizing their results, Langewisch and Frisch (1998) adjusted their data (2001), concluding that the results for sensation seeking did not reach significance (t=1.68, p<.05).

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

6.5 Delinquency, criminal and illegal activity: In the study by Winters et al. (1993b), delinquency status and illegal activity, or arrested, was related to problem gambling (X²=47.3, p<.001).

In the study by Feigelman et al. (1995), there was a significant relation between problem gambling and criminality in general (rₓᵧ=0.25, p=.001), and in number of arrests for criminal offenses (rₓᵧ=0.16, p=.01).

The study mentioned above (Ladouceur et al., 1999a) also showed a relation between SOGS and delinquency as measured by SRDS (Self-Reported Delinquency Scale) (F=176.18, p<.0001).

In the helpline study by Potenza et al. (2001) delinquency was identified as a significant

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 for profit ($t=7.83$, $p=.02$).

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

**C. Discussion**

The aim with the present study was to organize the existing empirical facts on 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 shown the importance of such knowledge to enable correct, and effective interventions.

We reviewed scientifically studies, with sound experimental design. Although both epidemiological and treatment studies could contribute to this knowledge, we choose not to include them for two reasons: (1) there are already published several reviews of epidemiological and treatment studies, as opposed to our approach, 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).

For an overview of results, see Table 1. For a condensed version of the empirical review results, see Table 2.

The relations between PG and some probable factors of importance (among them physiological and biological factors) are rather unclear.

If we look into the different groups, the main tendency shows a good deal of documentation for all groups of variables (see Table 2), 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.

The physiological and genetic studies give indications that these types of variables are related to PG in an important way. The meaning of the results in detail seems difficult to
interprete. Transmitters may still be probable markers for PG risk factors.

The cognitive distortions studied among others by Gilovich (1983) and Langer (1975), show clearly that there is a relation between cognitive factors and PG, at least in the analogue setting. Only few studies (i.e., Savoie and Ladouceur, 1995) have been tested in the natural environment of real gambling.

Among the strongest variables are the availability of plays. With increased availability, both playing and pathological gambling increases. That could be seen both in the increase in plays available over time, and differential availability of different plays.

Other important variables are sensory characteristics of the computer plays (sounds, lights), and the schedules of reinforcement, with random reward being the strongest rewarding factor (Skinner, 1953).

From clinical work it is wellknown that there is comorbidity between PG on one hand, and obsessive-compulsive disease (OCD), and drugs on the other. PG is also closely related to delinquency and criminality, which is also shown in this review.

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

Thus there is a great need for empirical research on the development and maintenance of PG. This should go on with epidemiological studies (particularly prospective) 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.
Risk factors for PG  25

References


Cocco N, Sharpe L, Blaszczynski AP (1995). Differences in preferred level of arousal in


Griffiths, M.D. (1993a). Fruit machine gambling: The importance of structural
characteristics. *Journal of Gambling Studies, 9*, 101-120.


Gambling Studies, 12, 215-231.


Table 2. Risk factors for PG – summary of results

<table>
<thead>
<tr>
<th>Domain</th>
<th>Risk factor</th>
<th>No. studies</th>
<th>Level*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Demographics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.1 Age</td>
<td>low age</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Gender</td>
<td>male</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>1.3 Education</td>
<td>low education</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1.4 Marital status</td>
<td>married/unmarried</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>1.5 Income</td>
<td>low income</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>1.6 Employment</td>
<td>unemployed</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>1.7 Social welfare status</td>
<td>on social welfare</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1.8 Residence</td>
<td>large city</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1.9 Acad achievement</td>
<td>low achievement</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>1.10 Immigrant, ethnic grp</td>
<td>immigrant, foreign</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2. Physiological/biological fact</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.1 Heart rate and arousal</td>
<td>increased during play</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2.2 Transmittor activity</td>
<td>increased</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>2.3 Genetic studies</td>
<td>DA rec increased</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>3. Cognitive distortions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.1 Erroneous perceptions</td>
<td>bias</td>
<td>4</td>
<td>2</td>
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<tr>
<td>3.2 Illusion of control</td>
<td>illusion</td>
<td>9</td>
<td>1</td>
</tr>
<tr>
<td>4. Varia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.1 Availability of plays</td>
<td>high</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>4.2 Parent playing</td>
<td>playing</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>4.3 Sensory characteristics</td>
<td>yes</td>
<td>7</td>
<td>1</td>
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<tr>
<td>4.4 Schedules of reinforcement</td>
<td>type</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>4.5 Age of onset</td>
<td>early</td>
<td>2</td>
<td>2</td>
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<tr>
<td>4.6 Rapid onset</td>
<td>short latency</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4.7 Playing duration</td>
<td>long</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>5. Comorbidity &amp; concurrent symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.1 Depression</td>
<td>yes</td>
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<td>2</td>
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<tr>
<td>5.2 Suicidality</td>
<td>yes</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>5.3 Anxiety</td>
<td>yes</td>
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<td>2</td>
</tr>
<tr>
<td>5.4 OCD</td>
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<td>6</td>
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</tr>
<tr>
<td>5.5 Alcohol</td>
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<td>2</td>
</tr>
<tr>
<td>5.6 Other drugs</td>
<td>yes</td>
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<td>1</td>
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<tr>
<td>5.7 Personality disorders</td>
<td>yes</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6. Personality symptoms and characteristics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>--------------------------------------</td>
<td>-------</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>6.1 Coping styles</td>
<td>low</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6.2 Impulsivity</td>
<td>high</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6.3 Hyperactivity (ADHD)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>6.4 Sensation seeking</td>
<td>high</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6.5 Delinquency &amp; illegal acts</td>
<td>yes</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

* (1) well-established risk factor for PG (>2 studies), and (2) probable risk factor for PG (1-2 studies). There are also some weaker studies mentioned in the text but not in Table 1.
### Table 1. Risk factors for pathological gambling (dependent variable: PG) [rev 2004-04-05 final]

<table>
<thead>
<tr>
<th>Domain</th>
<th>Sample</th>
<th>Instrument</th>
<th>Risk factor</th>
<th>Increased risk</th>
<th>Statistics(^1)</th>
<th>p value</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.1 Age</td>
<td>3426 HS stud</td>
<td>SOGS</td>
<td>school level</td>
<td>younger</td>
<td>F=7.73</td>
<td>&lt;.0001</td>
<td>Ladouceur et al (1999a)</td>
</tr>
<tr>
<td></td>
<td>2526 calls</td>
<td>SOGS</td>
<td>age</td>
<td>younger</td>
<td>(\chi^2=17.81)</td>
<td>.01</td>
<td>Bondolfi et al (2000)</td>
</tr>
<tr>
<td></td>
<td>562 calls</td>
<td>questionnaire</td>
<td>age</td>
<td>younger</td>
<td>(\chi^2=25.29)</td>
<td>(\leq .0001)</td>
<td>Potenza et al (2001)</td>
</tr>
<tr>
<td>1.2 Gender</td>
<td>8845 normals</td>
<td>SOGS-R</td>
<td>age</td>
<td>younger</td>
<td>OR=2.51</td>
<td>.000</td>
<td>Volberg et al (2001)</td>
</tr>
<tr>
<td></td>
<td>220 MMTP</td>
<td>SOGS</td>
<td>gender</td>
<td>male</td>
<td>(r_{xy}=0.12)</td>
<td>.04</td>
<td>Feigelman et al (1995)</td>
</tr>
<tr>
<td></td>
<td>3426 stud</td>
<td>SOGS</td>
<td>gender</td>
<td>male</td>
<td>(\chi^2=39.52)</td>
<td>&lt;.001</td>
<td>Ladouceur et al (1999a)</td>
</tr>
<tr>
<td></td>
<td>2526 calls</td>
<td>SOGS</td>
<td>gender</td>
<td>male</td>
<td>(\chi^2=8.94)</td>
<td>.01</td>
<td>Bondolfi et al (2000)</td>
</tr>
<tr>
<td></td>
<td>8845 normals</td>
<td>SOGS-R</td>
<td>gender</td>
<td>male</td>
<td>OR=3.71</td>
<td>.000</td>
<td>Volberg et al (2001)</td>
</tr>
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</table>
### Risk factors for PG – Table – 2

<table>
<thead>
<tr>
<th>Domain</th>
<th>Sample</th>
<th>Instrument</th>
<th>Risk factor</th>
<th>Increased risk</th>
<th>Statistics (^1)</th>
<th>p value</th>
<th>Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.4 Marital status</td>
<td>2526 calls</td>
<td>SOGS</td>
<td>marital status</td>
<td>married</td>
<td>(\chi^2=7.52)</td>
<td>.02</td>
<td>Bondolfi et al (2000)</td>
</tr>
<tr>
<td></td>
<td>8845 normals</td>
<td>SOGS-R</td>
<td>marital status</td>
<td>non-cohabting</td>
<td>(\chi^2=121.67)</td>
<td>.000</td>
<td>Volberg et al (2001)</td>
</tr>
<tr>
<td>1.5 Income/Finance</td>
<td>2526 calls</td>
<td>SOGS</td>
<td>income</td>
<td>higher</td>
<td>(\chi^2=10.88)</td>
<td>.01</td>
<td>Bondolfi et al (2000)</td>
</tr>
<tr>
<td></td>
<td>562 calls</td>
<td>questionnaire</td>
<td>income</td>
<td>higher</td>
<td>(\chi^2=5.79)</td>
<td>&lt;.02</td>
<td>Potenza et al (2001)</td>
</tr>
<tr>
<td></td>
<td>562 calls</td>
<td>questionnaire</td>
<td>problems</td>
<td>more</td>
<td>(\chi^2=4.21)</td>
<td>&lt;.04</td>
<td>Potenza et al (2001)</td>
</tr>
<tr>
<td>1.6 Employment</td>
<td>220 MMTP</td>
<td>SOGS</td>
<td>empl status</td>
<td>non-empl</td>
<td>(r_y=0.15)</td>
<td>&lt;.02</td>
<td>Feigelman et al (1995)</td>
</tr>
<tr>
<td></td>
<td>313</td>
<td>DSM-III-R</td>
<td>unemployed</td>
<td>increase</td>
<td>(t=11.09)</td>
<td>&lt;.001</td>
<td>Hall et al (2000)</td>
</tr>
<tr>
<td>1.7 Social welfare status</td>
<td>8845 normals</td>
<td>SOGS-R</td>
<td>soc welf stat</td>
<td>on welfare</td>
<td>(z=2.41)</td>
<td>.05</td>
<td>Volberg et al (2001)</td>
</tr>
<tr>
<td>1.8 Residence</td>
<td>8845 normals</td>
<td>SOGS-R</td>
<td>residence</td>
<td>big city</td>
<td>(z=4.00)</td>
<td>.01</td>
<td>Volberg et al (2001)</td>
</tr>
<tr>
<td>1.9 Academic achievement</td>
<td>3426 HS stud</td>
<td>SOGS</td>
<td>acad level</td>
<td>lower</td>
<td>(F=19.44)</td>
<td>&lt;.001</td>
<td>Ladouceur et al (1999a)</td>
</tr>
<tr>
<td>Domain</td>
<td>Sample</td>
<td>Instrument</td>
<td>Risk factor</td>
<td>Increased risk</td>
<td>Statistics¹</td>
<td>p value</td>
<td>Study</td>
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</tr>
<tr>
<td></td>
<td>702 (15-18)</td>
<td>SOGS-RA</td>
<td>low grades</td>
<td>more</td>
<td>$\chi^2 = 21.7$</td>
<td>&lt;.001</td>
<td>Winters et al (1993)</td>
</tr>
<tr>
<td>1.10 Immigrants and ethnic groups</td>
<td>562 calls</td>
<td>questionnaire</td>
<td>african/am</td>
<td>increase</td>
<td>$\chi^2 = 3.87$</td>
<td>&lt;.05</td>
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3.2 Illusion of control.

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<td>Langer (1975)</td>
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4. Varia
### Risk factors for PG – Table – 6

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## Risk factors for PG – Table – 9

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<td>220 MMTP SOGS</td>
<td>daily use</td>
<td>more</td>
<td></td>
<td>r_xy=0.14</td>
<td>.02</td>
<td>Feigelman et al (1995)</td>
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<tr>
<td>Drugs</td>
<td>702 (15-18) SOGS-RA</td>
<td>drugs</td>
<td>increase</td>
<td>(\chi^2=46.2)</td>
<td>&lt;.001</td>
<td></td>
<td>Winters et al (1993)</td>
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<tr>
<td>Drugs</td>
<td>3426 stud SOGS</td>
<td>drugs</td>
<td>increase</td>
<td>F=29.09</td>
<td>&lt;.0001</td>
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<td>Ladouceur et al (1999a)</td>
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<tr>
<td>Drugs</td>
<td>562 calls questionnaire</td>
<td>drugs</td>
<td>increase</td>
<td>(\chi^2=5.66)</td>
<td>&lt;.02</td>
<td></td>
<td>Potenza et al (2001)</td>
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<tr>
<td>Cigarettes</td>
<td>3426 stud SOGS</td>
<td>cigarettes</td>
<td>increase</td>
<td>F=20.42</td>
<td>&lt;.0001</td>
<td></td>
<td>Ladouceur et al (1999a)</td>
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<tr>
<td><strong>5.7 Personality disorders</strong></td>
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<tr>
<td></td>
<td>7,869M(twinp) DSM-III-R/DIS</td>
<td>ASPD</td>
<td>increase</td>
<td>OR=6.4</td>
<td></td>
<td></td>
<td>Slutsk et al (2001)</td>
</tr>
<tr>
<td></td>
<td>7,869M(twinp) DSM III-R/DIS</td>
<td>CD</td>
<td>increase</td>
<td>OR=3.6</td>
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<td>Slutsk et al (2001)</td>
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### Risk factors for PG – Table – 10

<table>
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<tr>
<th>Domain</th>
<th>Sample</th>
<th>Instrument</th>
<th>Risk factor</th>
<th>Increased risk</th>
<th>Statistics&lt;sup&gt;1&lt;/sup&gt;</th>
<th>p value</th>
<th>Study</th>
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<td>6. Personality symptoms and characteristics</td>
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<td>6.1 Coping styles</td>
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<tr>
<td></td>
<td>7,869M(twinp)</td>
<td>DSM-III-R/DIS</td>
<td>AAB</td>
<td>increase</td>
<td>OR=6.1</td>
<td></td>
<td>Slutske et al (2001)</td>
</tr>
<tr>
<td></td>
<td>69 pat</td>
<td>DSM-III-R</td>
<td>concurr sy</td>
<td>increase</td>
<td>t=-3.0</td>
<td>.004</td>
<td>Ibáñez et al (2001)</td>
</tr>
<tr>
<td></td>
<td>69 pat</td>
<td>DSM-III-R</td>
<td>comorbid dis</td>
<td>increase</td>
<td>r&lt;sub&gt;y&lt;/sub&gt;=.40</td>
<td>&lt;.001</td>
<td>Ibáñez et al (2001)</td>
</tr>
<tr>
<td>6.2 Impulsivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td></td>
<td>754 adol boys</td>
<td>SOGS/EIS/TRIS</td>
<td>impulsivity</td>
<td>increase</td>
<td>EIS χ&lt;sup&gt;2&lt;/sup&gt;=30.58</td>
<td>&lt;.01</td>
<td>Vitaro et al (1997)</td>
</tr>
<tr>
<td></td>
<td>754 adol boys</td>
<td>SOGS/EIS/TRIS</td>
<td>impulsivity</td>
<td>increase</td>
<td>TRIS χ&lt;sup&gt;2&lt;/sup&gt;=27.95</td>
<td>&lt;.01</td>
<td>Vitaro et al (1997)</td>
</tr>
<tr>
<td></td>
<td>144 students</td>
<td>SOGS/SSS</td>
<td>impulsivity</td>
<td>high</td>
<td>t=3.00</td>
<td>.003</td>
<td>Langewisch &amp; Frisch (2001))</td>
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<tr>
<td>6.4 Sensation seeking</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>12 gambl/ 12 c</td>
<td>SSS</td>
<td>exper seek</td>
<td>increase</td>
<td>r&lt;sub&gt;y&lt;/sub&gt;=-.25/.54</td>
<td>&lt;.05</td>
<td>Andersson &amp; Brown (1984)</td>
</tr>
</tbody>
</table>

<sup>1</sup> Statistics include t-test, F, χ<sup>2</sup>, and r<sub>y</sub>.
### Risk factors for PG – Table – 11

<table>
<thead>
<tr>
<th>Domain</th>
<th>Sample</th>
<th>Instrument</th>
<th>Risk factor</th>
<th>Increased risk</th>
<th>Statistics¹</th>
<th>p value</th>
<th>Study</th>
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<tr>
<td>6.5 Delinquency, criminality and illegal activity</td>
<td>702 (15-18)</td>
<td>SOGS-RA</td>
<td>delinquency</td>
<td>increase</td>
<td>χ²=47.3</td>
<td>&lt;.001</td>
<td>Winters et al (1993)</td>
</tr>
<tr>
<td></td>
<td>220 MMTP</td>
<td>SOGS</td>
<td>delinquency</td>
<td>more</td>
<td>rₓ=0.25 .001</td>
<td></td>
<td>Feigelman et al (1995)</td>
</tr>
<tr>
<td></td>
<td>220 MMTP</td>
<td>SOGS</td>
<td>criminality</td>
<td>more</td>
<td>rₓ=0.16 .01</td>
<td></td>
<td>Feigelman et al (1995)</td>
</tr>
<tr>
<td></td>
<td>3426 stud</td>
<td>SOGS/SRDS</td>
<td>delinquency</td>
<td>increase</td>
<td>F=176.18</td>
<td>&lt;.0001</td>
<td>Ladouceur et al (1999a)</td>
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<tr>
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<td>562 calls</td>
<td>questionnaire</td>
<td>delinquency</td>
<td>increase</td>
<td>χ²=9.53</td>
<td>&lt;.002</td>
<td>Potenza et al (2001)</td>
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<tr>
<td></td>
<td>313</td>
<td>DSM-III-R</td>
<td>incarceration</td>
<td>increase</td>
<td>t=16.53</td>
<td>&lt;.001</td>
<td>Hall et al (2000) se ref</td>
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<tr>
<td></td>
<td>313</td>
<td>DSM-III-R</td>
<td>illegal activity</td>
<td>increase</td>
<td>t=7.83</td>
<td>.02</td>
<td>Hall et al (2000)</td>
</tr>
</tbody>
</table>

**Note**

Some references to weak/inconclusive studies are not included in table, only in text
PG= Pathological Gambling
SOGS = South Oaks Gambling Screen
DSM = Diagnostic and Statistical Manual of Mental Disorders
SSS = Sensation Seeking Scale
rₓ=correlation, t-test, OR=odds ratio, Fₓ=Anova, Fₓ=Regression
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