Pathological Gambling

Treatment and Personality Factors

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“Life is the art of drawing sufficient conclusions from insufficient premises”

Samuel Butler, 1835-1902

Contents

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List of Papers

Paper I

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Paper II

Myrseth, H., Pallesen, S., Molde, H., Johnsen, B. H., & Lorvik, I. M. (2009). Personality factors as predictors of pathological gambling. *Personality and Individual Differences, 47*, 333-337.

Paper III

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Summary

Objectives

Pathological gambling is associated with substantial negative consequences as pathological gamblers typically incur substantial debts and experience family and social relationship problems as well as work-related problems due to their gambling. As prevalence rates of pathological gambling hover about 1-3%, the need for efficacious treatments is evident. Although the effectiveness of different forms of treatment has been established, attrition rates are usually high and relapses are common.

The first paper of this dissertation examined the short- and long-term effectiveness of individual cognitive behavioral therapy (CBT) and pharmacological treatment in combination with individual CBT.

The second paper examined differences in impulsivity, sensation seeking and the dimensions of the five-factor model of personality in treatment-seeking pathological gamblers and in a sample of non-pathological gamblers.

In the third paper the aim was to identify psychopathology and personality characteristics among pathological gamblers, and to investigate whether gamblers could be divided into clinically meaningful subgroups based on psychopathology and personality factors.

Methods

The first study comprised an investigation using a randomized controlled trial design with 30 pathological gamblers who received either eight weeks of individual CBT (n = 15) or 16 weeks of pharmacological treatment (escitalopram 20mg/day) in combination with

eight weeks individual CBT (n = 15). Assessments with outcome measures were conducted at pre-treatment, 8-weeks post-treatment (when one group had received CBT and one group escitalopram only), 16-weeks post-treatment (when both groups had received CBT) and at 3- and 6-months follow-up. Repeated measures ANOVA with intent-to-treat analyses were conducted to measure the effectiveness of the treatment conditions.

The second study used bivariate and multivariate logistic regression analyses to investigate whether demographic variables, impulsivity, sensation seeking and the five domains of the five-factor model were associated with pathological gambling status. The sample consisted of 90 pathological gamblers and a contrast group (n = 66) matched on sex and age.

In the third paper the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) was used to examine psychopathology and personality characteristics among 66 pathological gamblers. Latent class analysis was conducted to identify subgroups of pathological gamblers.

Results

In the first study both treatments were related to significant improvement on most outcome measures both on short- and long-term basis. No differential group effects nor time x group effects were found at any assessments.

In the second study 12 of the 14 predictor variables showed a significant relationship with pathological gambling in the bivariate analyses, but only four of these (Neuroticism, Openness, Impulsivity and Need for Stimulus Intensity) remained significant in the

multivariate analysis. High scores on Neuroticism, low scores on Openness, high scores on pathological Impulsivity and high Need for Stimulus Intensity were significantly related to pathological gambling. These predictor variables accounted for 71% of the variance of clinical status (pathological or non-pathological gamblers).

Study III showed that pathological gamblers in general are characterized by depression and anxiety, and showed elevated mean scores on scales 2-D (Depression) and 7-Pt (Psychastenia) of the MMPI-2. Further, the latent class analysis indicated a three latent cluster solution, where one cluster had all mean scores within the normal range and two latent clusters had elevated profiles and were described by considerably psychopathology. Latent Cluster 2 (comprising 33% of the sample) had elevations on six scales and was characterized by a 2-7 profile type. Latent Cluster 3 (comprising 12% of the sample) had elevations on eight scales and was characterized by an 8-7 profile type.

Conclusions

The present thesis concludes that both CBT and escitalopram seem to be effective in treating pathological gamblers in the short-term, and that CBT (alone or in combination with escitalopram) seems to be effective on a long-term basis. Furthermore, high scores on Neuroticism, low scores on Openness, high scores on pathological impulsiveness, and high scores on Need for Stimulus Intensity are concluded to be significantly related to pathological gambling. It is also concluded that pathological gamblers are a diverse group with different personality configurations and degrees of psychopathology. Latent class analyses indicated three separate latent clusters. Approximately half of the sample showed quite normal personality profiles, while the other half was characterized by considerable pathology.

Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
AISS	Arnett Inventory of Sensation Seeking
BAS	Behavioral Activation System
BDI-II	Beck's Depression Inventory-II
BIS	Behavioral Inhibition System
BIS-11	Barratt Impulsiveness Scale-11
CBT	Cognitive Behavioral Therapy
CG	Contrast Group
CI	Confidence Interval
CSF	Cerebrospinal Fluid
DSM	Diagnostic and Statistical Manual for Mental Disorders
EEG	Electroencephalography
EIS-nI	Eysenck Impulsivity Scale – narrow Impulsiveness subscale
EGM	Electronic gaming machine
ES	Effect Size
FFM	Five-Factor Model
fMRI	functional Magnetic Resonance Imaging
G-SAS	Gambling Symptom Assessment Scale
LCA	Latent Class Analysis
MMPI	Minnesota Multiphasic Personality Inventory
NEO-FFI	NEO-Five Factor Inventory
NODS	National Opinion Research Center DSM Screen for Gambling Problems
NOK	Norwegian kroner
PG	Pathological Gambling
PGBS	Pathological Gambling Behavioral Self-Report Scale

PGs	Pathological Gamblers
PGVAC	Pathological Gambling 100 mm Visual Analog Craving Scale
SCID-I	Structured Clinical interview for DSM-IV Axis I Disorders
SCID-II	Structured Clinical interview for DSM-IV Axis II Disorders
SOGS-R	South Oaks Gambling Screen-Revised
SSRI	Selective Serotonin Re-uptake Inhibitors
SSS-V	Sensation Seeking Scale form V
1-Hs	Scale 1 - Hypochondrias
2-D	Scale 2 - Depression
3-Hy	Scale 3 - Hysteria
4-Pd	Scale 4 - Psychopathic deviate
5-Mf	Scale 5 - Masculine-feminine
6-Pa	Scale 6 - Paranoia
7-Pt	Scale 7 - Psychastenia
8-Sc	Scale 8 - Schizophrenia
9-Ma	Scale 9 - Mania
0-Si	Scale 0 - Social introversion

1. Introduction

For most people gambling is a leisure activity without any negative consequences. However, others develop excessive gambling behavior which has severe negative consequences not only for the gambler, but also for his or her relationships with partners, family members, friends, or colleagues.

Although different forms of gambling have long existed, it has never appeared in as many forms as it does today. Since 1980, the legalization of new forms of gambling has increased in most Western countries. As a result, this greater multitude of availability and diversion has created a situation in which more people have developed serious gambling problems, thus the need for a better understanding of this phenomena and the need for developing effective prevention and treatment strategies are warranted.

1.1 Definition and Diagnosis

A wide array of different definitions of gambling exists. According to one definition, gambling is an activity which involves an attempt to win money by staking money on uncertain events (Ladouceur et al., 2001). Inherent in all forms of gambling is the uncertainty and risk aspect, and gambling itself is a risk-taking activity. Gambling has been associated with factors related to risky decision making (Grant, Brewer, & Potenza, 2006). *Pathological gambling* (PG) is a behavioral disorder that was officially recognized by the American Psychiatric Association in 1980 with the publication of the *Diagnostic and Statistical Manual of Mental Disorders III* (DSM-III), and has since been classified as an *impulse control disorder* (American Psychiatric Association, 1980). The diagnostic criteria for PG were later revised and, according to the latest version of DSM,

the DSM-IV Text Revision (DSM-IV-TR), the essential feature of PG is "persistent and recurrent maladaptive gambling behavior that disrupts personal, family, or vocational pursuits", which is not better accounted for by a manic episode (American Psychiatric Association, 2000, p. 671). In order to receive this diagnosis, one has to fulfill a minimum of 5 out of 10 criteria (see Appendix A for an overview of the diagnostic criteria).

In the literature, one often refers to various levels of disordered gambling, and the term *pathological gambling* (or Level 3 gambling) is usually defined by fulfillment of the DSM-IV diagnostic criteria (a minimum of 5 out of 10) for PG, whereas the term *problem gambling* (or Level 2 gambling) is used when the diagnostic criteria are only partly fulfilled (3-4 out of 10). *Social* or *recreational gambling* (or Level 1 gambling) denotes gambling which does not result in any significant problems (Petry, 2005).

1.2 Historical Overview

Some have proposed that risk-taking is a part of human nature, and Bellringer (1999) suggested that gambling is only a stylized form of risk taking. Accordingly, gambling is a phenomenon known not only to modern society, but is surmised to have been around as long as civilization itself (Bellringer, 1999). Traces of gambling being more then 4000 years old have been found in the Middle East and India. It is also documented that gambling was known in ancient Greece as well (Ladouceur, Sylvain, Boutin, & Doucet, 2002; Petry, 2005). Tragedies (such as familial problems and accumulation of large debts) have always been associated with gambling, and the history of gambling has been characterized by an alternation of liberal and conservative attitudes. After periods of liberal politics, the tragedies become prominent which lead to the

prohibition of legal gambling, though good reasons for the legalization of gambling have also existed despite the terrible consequences. For example, lotteries have been introduced by some states in order to collect money for charity. Many humanitarian and voluntary organizations have become dependent on the money earned from the gambling industry, which has created a huge dilemma in the current debate on the legalization of gambling.

During the 1990s, drastic changes occurred in the gambling market, and the worldwide liberalization of gambling occurred during this decade. The accessibility of gambling increased, the limitations on maximum prizes vanished in Norway, new forms of gambling were introduced and the attitudes towards gambling and social approval changed. During the 1990s, the gambling market in Norway exploded along with the introduction of electronic gaming machines (EGMs), and the maximum stakes increased from 1 to 500 Norwegian Kroner (NOK). Norway became one of the countries with the fastest growing gambling market, and had more gambling machines per capita than most other countries. In 2002, Norway had 23,000 legal EGMs (one machine per 200 people), which was far more than Sweden and Denmark put together. The machines in Norway were considered to be aggressive, and many of these machines were prohibited in Denmark and Sweden. An EGM is considered to be aggressive when the stakes and prizes are high and the games are of short duration, thereby making it possible to lose large amounts of money in a short period of time. Norway was also the first of the Nordic countries to legalize gambling by SMS. Overall, this has made Norway one of the world's most liberal countries in terms of gambling politics (Fekjær, 2002).

From 1998 until 2005, the total turnover in the Norwegian gambling market increased from approximately 7 to 42 billion NOK and the gross turnover of EGMs

constituted approximately 65% of the total Norwegian gambling market (Norwegian Gaming Board, 2004). This significant rise in turnover following the liberalization of the gambling market in Norway attracted public attention, and concerns were expressed in regard to the problems this caused to both individuals and society. For this reason, the Norwegian government decided to put forward an action plan to prevent and reduce these problems. The action plan was published in April 2005 (Norwegian Gaming Board, 2005) and stated that up to 0.5% of the revenue of Norsk Tipping (Norway's leading game company, owned by the state) should annually be earmarked to put the plan into action. These funds have been used to improve the information, prevention, treatment and research into gambling problems.

As of July 2006, it became illegal to use bank notes in Norwegian gaming machines. This has resulted in a turnover in the total gambling market of 38 billion NOK, a 10% decrease compared to the previous year, and starting in July 2007, all slot machines were banned from the market. Consequently the turnover decreased by an additional 29% (Norwegian Gaming Board, 2007), and the total gross turnover in 2007 was reduced to 27 billion NOK. This turnover continued to decrease by 31% in 2008 (the only year without any EGMs in Norway), and was lowered to approximately 19 billion NOK (Norwegian Gaming Board, 2008). However, as the Norwegian gambling market has become more regulated and the national gross turnover has decreased, Norwegians have increasingly spent large amounts gambling on foreign websites/internet games. It was estimated that Norwegians staked a minimum of 6.3 billion NOK on foreign websites in 2007, which is a 10-fold increase from 2001 (Norwegian Gaming Board, 2007). In 2008, this was estimated to be closer to 7 billion NOK (Norwegian Gaming Board, 2008).

1.3 Theories of Addiction

Several different theories of addiction have been proposed, and it is beyond the scope of this thesis to give an account or overview of all. This chapter will only give a short list (but not exclusive) of different theories of addiction, and in a subsequent chapter more specific theories and perspectives related to gambling will be presented. Some theories, such as Skog's Choice Theory (2000) and Slovic's Affect Heuristic theory (2002), emphasizes the *choice* aspect of addictions whereas others emphasize concepts of *impulse* and *self-control*, e.g. Self-regulation theory (Baumeister, Heatherton, & Tice, 1994). Yet others emphasize aspects of *habit* and *instrumental learning*, (e.g., Dopamine Theory of drug reward (Koob & Nestler, 1997)), in explaining addictions.

Jacobs defines addiction as "a dependent state acquired over time by a predisposed person in an attempt to correct a chronic pre-existing stress condition" (Jacobs, 1987, p. 171). He suggested two interacting factors which predispose a person for addiction: An abnormal physiological resting state and childhood experiences. The abnormal physiological resting state may be characterized by either hypo- or hyper-arousal which, when perceived as an aversive arousal state, predispose an individual to respond differentially to potentially addictive substances or experiences. "Reducers" are characterized by a chronic over-mobilization of the physiological resting state, while "enhancers" are characterized by a chronic under-mobilization. Childhood experiences of rejection by parents or significant others convince the person that he/she is inadequate, inferior, unwanted and unneeded. Instead of responding adaptively to these circumstances by building success-producing coping behaviors, these addiction-prone individuals often retreat into wish-fulfilling fantasies in which they may find relief from the painful reality. The use of an addictive substance or behavior offers the possibility of escape from this

painful reality, and is one of the essential reinforcing qualities that maintain the addictive pattern. Thus, the need to ameliorate and escape from the effects of both physiological and psychological stress conditions is assumed to continue addictive patterns of behavior (Jacobs, 1987).

Robert West (2006) presented a synthetic theory of addiction which recognizes that an addiction can involve different aspects of a motivational system, and he viewed addiction as "a chronic condition of the 'motivational system' in which a reward-seeking behaviour has become 'out of control' " (R. West, 2006, p. 174). He suggested three types of abnormality underlying addictions: 1) abnormalities in the motivational system of the individual (such as propensity to anxiety, depression or impulsiveness), 2) abnormalities in the motivational system that stem from the addictive behavior itself (such as acquisition of a strongly entrenched habit), and 3) abnormalities in the individual's social or physical environment (such as presence of strong social pressure to engage in the behavior). According to R. West (2006), addictions affect our choices, needs and desires, emotional attachment to the object, and our sense of identity, but it cannot be understood solely in terms of any single one of these components. Rather, an understanding of addiction requires an understanding of the total motivational system.

Shaffer et al. (2004) propose a syndrome model of addiction which suggests that different addictive disorders might not be independent, but rather a distinctive expression of the same underlying addiction syndrome. They propose that the different expressions of addictions share some commonalities reflected in a shared etiology – an addiction syndrome. Common antecedents of this syndrome include individual vulnerability, object exposure and object interaction. Both neurobiological and psychosocial elements can influence an individual's behavior and either increase or reduce the likelihood of

addiction (e.g., social support and networks). Interaction with the object may lead to neurobiological consequences which are common to all objects of addiction (e.g., activation of reward circuitry), thus increasing the likelihood of further interaction. Repeated interaction with a specific object, followed by desirable neurobiological or social consequences that produce a sought-after shift in a subjective state further increase the likelihood of addiction. The addiction syndrome can manifest itself in many different ways (e.g., gambling or drinking), and increases the risk for continuing the addictive behavior in addition to developing new addictive behaviors. Shared neurobiological antecedents, shared psychosocial antecedents, and shared experiences of different addictions could have implications for treatment. In this regard, Shaffer et al. (2004) emphasize that clinicians often overlook effective chemical addiction treatments for behavioral addictions and useful behavioral treatments for chemical addictions.

1.3.1 Behavioral Addictions

Traditionally, the concept of addiction has been viewed as an addiction to a *substance* (i.e., tobacco, alcohol or drugs), which is why many official definitions include drug ingestion. In 1957, the World Health Organization defined addiction as "a state of periodic or chronic intoxication produced by repeated consumption of a drug, natural or synthetic" (World Health Organization, 1957, p. 9). Even so, there is now a growing tendency to view a number of behaviors as potentially addictive, including behaviors that do not involve the ingestion of any substance. These include behaviors such as gambling (Orford, 2001), overeating (Orford, 2001), compulsive spending (Griffiths, 1990), exercise (Glasser, 1976), sex (Carnes, 1983), kleptomania (Griffiths, 1990) and computer

game playing (Griffiths, 1993). Newer and broader definitions of the concept of addiction have therefore emerged.

Although addictive behaviors have idiosyncratic differences, there are many commonalities across these addictions. Griffiths (2005) postulates that different addictions, both chemical and non-chemical, are characterized by six common core components (modified from Brown, 1993): 1) salience, 2) mood modification, 3) tolerance, 4) withdrawal, 5) conflict and 6) relapse. The *salience* component refers to a particular activity becoming so important to the individual that it dominates their thinking, feelings and behavior. *Mood modification* refers to the subjective experience which arises from engaging in a particular activity (adding positive feelings and/or removing negative feelings). *Tolerance* is established when increasing amounts (e.g., time) of the particular activity are required to produce the former effects. *Withdrawal effects*, either psychological or physical, refer to the unpleasant feeling states and/or physical effects that occur when the activity is suddenly reduced or discontinued. The individual usually experiences *conflicts* related to the particular activity, which could either be conflicts with individuals around them (interpersonal conflict) or within the individual themselves (intrapsychic conflict). The last component is *relapse* which typically accompanies any addiction. As Griffiths (2005) also points out, excessive behavior of all types seem to have many commonalities and these commonalities, may have implications for the treatment of such behaviors.

1.4 Theoretical Perspectives on Pathological Gambling

Research has varied from investigating underlying biological, psychological, or social factors hypothesized to contribute to gambling behavior in an attempt to understand this complex social and psychological problem. Evidence now exists that all these factors are relevant to the development of problematic gambling behavior (Sharpe, 2002). This chapter will give an overview of the most relevant theoretical perspectives of how pathological gambling is developed and maintained.

1.4.1 Psychodynamic Perspective

Psychoanalysts provided the first systematic attempts to understand and treat pathological gambling (National Research Council, 1999). This perspective seeks to explain pathological gambling through motivational forces that derive from unconscious mental processes (Wong, 1989). Pathological gambling is seen as a symptom of or an expression of an underlying psychological condition. Bergler (1970) proposed that the gambler had an unconscious need to lose (referred to as psychic masochism) which was induced by childhood conflicts creating an urge to produce situations in which the gambler felt unjustly treated (Bergler, 1970). Simmel (1920) emphasized narcissistic fantasies, grandiosity and the need to deny feelings of smallness and helplessness in explaining pathological gambling. He suggested that denial of parental love and the good things in life resulted in a need for pleasure, excitement and promise of gain which the gambling seemed to fulfill, and that winning produced feelings of omnipotence (Simmel, 1920). Other psychoanalysts have also suggested deprivation from parents as essential (Greenson, 1947). This is assumed to make the gambler turn to fate or “Lady Luck” to get the love, acceptance and the approval that he or she has been denied (Greenson, 1947;

Niederland, 1967). Boyd and Bolen (1970) explained compulsive gambling as a manic defense against helplessness and depression.

The psychodynamic approach emphasizes the meaning and consequences of one's behavior (Rosenthal & Rugle, 1994) and psychodynamic therapy for PGs attempts to help the gambler understand the underlying source of their distress and to confront it. Rosenthal and Rugle (1994) suggest that the immediate goal in treatment of gamblers should be abstinence. They also propose that understanding the meaning and consequences of the patient's behavior is essential for sustaining abstinence. Therapists should therefore help the patient to identify adverse emotional states that may have been relieved by gambling in order to understand the defensive function of their gambling behavior. Although several researchers have noted the value of psychodynamic treatment for gambling (e.g., Boyd & Bolen, 1970; Rosenthal & Rugle, 1994), there have so far been no controlled or randomized study exploring the effectiveness of this approach (National Research Council, 1999).

1.4.2 Biological Perspective

In opposition to the psychodynamic perspective which emphasizes early experiences and underlying psychopathology in the development of gambling problems, the biological perspective suggests a biological predisposition that gives rise to gambling problems, such as lack of inhibition and difficulty to control impulses (Ladouceur, et al., 2002). Bagby et al. (2007) suggested that the inability to exercise control over one's gambling may be linked to neurobiological substrates that lead to an increased focus on reward, a lowered response to aversive consequences, and impairments in altering

behavior based on risk-reward learning (this will be further elaborated subsequently in this chapter). Research into such mechanisms is still in the early stages. Nevertheless, the possible relationship with other disorders (e.g., substance use), and the nature of the core symptoms of PG allow for making assumptions about the underlying neurobiology (Myrseth & Pallesen, 2010).

Abnormalities in the prefrontal cortex and subcortico-cortical networks projecting to the frontal cortex, areas which are important in executive functions (such as planning, self-regulation, response modulation and inhibition), have been implicated in PG as well as other addictions (Goudriaan, Oosterlaan, deBeurs, & Van den Brink, 2004). PGs show relatively diminished activation of ventral cortico-striatal circuitry (involving the ventro-medial prefrontal cortex and ventral striatum) during response inhibition, decision-making, simulated gambling, and gambling urge paradigm tasks compared to control subjects (Potenza, 2008b). These brain regions have been implicated in aspects of impulsivity.

The lack of self-regulation in PG has further been suggested to result from abnormal neurotransmitter regulation in the “reward pathways” of the brain (e.g., "dopamine system"; Goudriaan, et al., 2004). Dopamine is one of the main neurotransmittors of the mesolimbic brain structures, and abnormal functioning of these structures could result in abnormal sensitivity for rewards and/or losses. These structures further play an important role in regulating arousal, thus abnormalities in the mesolimbic structures could result in an abnormal arousal regulation in PG, resulting in longer play (Goudriaan, et al., 2004).

Blum et al. (1997) suggested the concept of a reward deficiency syndrome, referring to alterations in brain chemistry that interfere with the brain's reward processes. They suggested that genetic commonalities across a spectrum of behavioral disorders (such as substance abuse, smoking, compulsive over-eating, attention-deficit disorder, and pathological gambling) may result in an underlying chemical imbalance (in the dopamine system) that alters the signaling in the brain's reward process. They specifically suggested a common genetic deficiency in the dopamine D2 receptor (the A1 allele gene) as underlying the reward deficiency syndrome. The deficiency syndrome involves a form of sensory deprivation of the brain's pleasure mechanisms, and the biochemical deficiency can supplant an individual's feeling of well being with anxiety, anger or a craving for a substance (Blum, Cull, Braverman, & Comings, 1996).

Few studies have so far investigated differences in the brain activity of PGs compared to individuals without PG. An early electroencephalography (EEG) study on lateralization of EEG activity in PGs (L. Goldstein & Carlton, 1988) showed that PGs had less shifting between hemispheric activity compared to controls, and it took the PGs significantly longer to activate either the left or right hemisphere. These results have been interpreted as an inflexibility in the brain activity of PGs (Goudriaan, et al., 2004). More dysfunctional EEG activity has also been found in PGs compared to healthy controls (Regard, Knoch, Guetling, & Landis, 2003). Further, EEG studies have shown higher arousal levels and increased endogenous dopamine activity in PGs compared to controls (Stojanov et al., 2003). Potenza, Steinberg, Skudlarski et al. (2003) conducted a functional magnetic resonance imaging (fMRI) study investigating urges or craving in PGs, and found that compared to recreational gamblers, PGs showed relatively less blood oxygen level-dependent signal change in the frontal cortical, basal ganglionic and

thalamic brain regions when viewing gambling tapes (Potenza, Steinberg, et al., 2003). Further, decreased activity in the ventral striatum and in the ventromedial prefrontal cortex has been found in PGs (Reuter et al., 2005). It has therefore been suggested that in PGs (who have reduced activation in the ventral striatum) the natural reinforcers of everyday life are not strong enough for obtaining optimal dopamine levels; therefore, they must seek additional and stronger reinforcers such as gambling to compensate for this lack of activation.

Mounting evidence suggests the involvement of multiple neurotransmitter systems in the pathophysiology of PG (Potenza, 2001; van Holst, van den Brink, Veltman, & Goudriaan, 2009). These systems are related to the mechanisms which underlie behavioral disinhibition (serotonergic system), reward mechanisms (dopaminergic and opioidergic system), and arousal (noradrenergic system) associated with impulse control and addictive disorders (Petry, 2005). The following gives an outline of different neurotransmitter systems that are likely to be involved in PG, and empirical evidence for the involvement is presented.

Norepinephrine. Norepinephrine is involved in arousal and excitement, and could be central in the increasing arousal experienced before engaging in gambling behavior (Myrseth & Pallesen, 2010). A study by Roy et al. (1988) found higher levels of norepinephrine and its metabolites in urine, blood, and cerebrospinal fluid (CSF) samples in pathological gamblers (PGs) compared to controls, suggesting that the noradrenergic system may also play a role in the pathophysiology of PG via the effect on arousal. Additionally, an increased heart rate and plasma concentration of norepinephrine during blackjack gambling have been found to a greater degree in PGs in comparison to non-pathological gamblers (Meyer et al., 2004). Furthermore, norepinephrine is also involved

in the physiological functions associated with impulse control (Blanco, Ibáñez, Sáiz-Ruiz, Blanco-Jerez, & Nunes, 2000). Consistent with this, adrenergic drugs have been found effective in treating impulsive features of attention deficit hyperactivity disorder (ADHD), and have been shown to improve response inhibition in both animals and humans (Chamberlain & Sahakain, 2007).

Serotonin. Serotonin, which is a central neurotransmitter in behavior initiation/cessation, has been considered to be of great importance in mediating impulse control (Potenza, 2008a). A dysfunction of the serotonin (5-hydroxytryptamine; 5-HT) system has been shown to play an important role in impulsive behaviors (Blanco, et al., 2000), and studies have specifically indicated that PGs may be characterized by a serotonergic dysfunction (Blanco, et al., 2000). PGs have demonstrated low levels of the serotonin metabolite 5-hydroxy indoleacetic acid (Nordin & Eklund, 1999), and decreased activity of the peripheral marker of the serotonergic function platelet monoamine oxidase B (MAO-B) has been found in men with PG (Blanco, Orensanz-Munoz, Blanco-Jerez, & Saiz-Ruiz, 1996). A hypersensitivity of postsynaptic serotonin receptors in PGs has also been suggested by DeCaria et al. (1996), who found an increased prolactin response to a single dose of *m*-chlorophenylpiperazine (m-CPP) in male PGs compared to controls. Individuals with PG or other disorders characterized by impaired impulse control have also displayed various behavioral and biochemical responses to serotonergic drugs compared to healthy control subjects (Pallanti, Bernardi, Quercioli, DeCaria, & Hollander, 2006). Because of the possible importance associated with the role of serotonergic function in PG, serotonergic medications have been used in the treatment of PG. Even so, treatments with selective serotonin re-uptake inhibitors (SSRIs) show mixed results (Hollander, Kaplan, & Pallanti, 2004).

Dopamine. Dopamine is a neurotransmitter central in the reward and reinforcement systems in the ventral tegmental area and the nucleus accumbens, and it has been hypothesized that this reinforcement system is likely to be involved in the rewarding feeling of pleasure experienced when engaging in gambling behavior (Potenza & Hollander, 2002). Age-related changes in the dopaminergic system have been suggested to be related to the development of PG, e.g. changes in dopamineregic function during adolescence may underlie adolescent vulnerability to gambling problems (Chambers & Potenza, 2003). Bergh, Eklund, Södersten and Nordin (1997) found that CSF levels of dopamine were decreased while levels of dopamine metabolites were increased in PGs, suggesting an increased release of dopamine in these individuals. A low D2 receptor availability has been hypothesized to mediate a vulnerability to addiction (Brewer & Potenza, 2008). One study found that the variant allele of the dopamine D4 receptor gene, which leads to poorer functioning of the receptor, is associated with PG (Perez de Castro, Ibáñez, Torres, Saiz-Ruiz, & Fernandez-Piqueras, 1997). Investigations of dopamine dysregulation in PG show mixed results (Nordin & Eklund, 1999). However, the onset or worsening of PG has been observed in individuals with Parkinson's disease when they are treated with drugs that promote dopamine functions (e.g., levodopa or dopamine agonists) (Brewer & Potenza, 2008; Shah, Potenza, & Eisen, 2004).

Opioids. Opioids have been implicated in pleasurable and rewarding processes, and influence the neurotransmission in the mesolimbic pathway which extends from the ventral tegmental area to the nucleus accumbens or the ventral striatum (Potenza, 2008a). Due to its reinforcing properties, opioids have also been assumed to contribute to the urges experienced before committing a certain behavior (Potenza & Hollander, 2002). Opioid antagonists inhibit the release of dopamine in the nucleus accumbens and have

been found to be effective in treating PGs both in an open-label and double-blind study (Kim & Grant, 2001; Kim, Grant, Adson, & Shin, 2001).

Glutamate. Glutamate is an excitatory neurotransmitter which has been implicated in motivational processes and drug addiction, and findings suggest that antiglutamatergic agents may also be beneficial in treating impulse control disorders (Potenza, 2008a). An open-label treatment study, with a double-blind discontinuation with the glutamatergic modulating agent *N*-acetyl cysteine for PGs, found that 83% of responders randomized to active drugs maintained improvement in the discontinuation phase compared to 29% of those randomized to placebo (Grant, Kim, & Odlaug, 2007).

Over the last few decades, there has been considerable progress in the pharmacotherapy of addiction, and several therapeutic strategies have evolved based on the knowledge related to the mechanisms in the development of addiction and the physiology of the brain reward system (Vetulani, 2001). As previously mentioned, evidence suggest the involvement of serotonergic, noradrenergic, dopaminergic and opioidergic systems in the etiology of PG (Shah, et al., 2004), and pharmacological treatments targeting these neurotransmitter systems have yielded promising results in the early stages of the understanding and treatment of PG (Hollander, et al., 2004). Hollander, Kaplan, and Pallanti (2004) outline several psychopathological domains within PG which could conceivably be targeted for treatment: impulsive symptoms (arousal), compulsive symptoms (anxiety reduction), and addictive symptoms (symptoms of withdrawal). The pharmacological treatment of PG has usually involved the administration of either opioid antagonists, anti-depressants or mood stabilizers. Opioid antagonists block the effects of endogenous endorphins on central opiate receptors and inhibit dopamine release in the nucleus accumbens, which involves reward, pleasure and urge mechanisms (Hollander, et

al., 2004). Most of the antidepressants drugs used in the treatment of PG are SSRIs which appear to have anti-compulsive and anti-impulsive effects (Hollander, et al., 2004). It has been suggested that impulse control disorders and bipolar spectrum disorders may be related, and that the impulsivity in PG seems to resemble that of bipolar disorder. The co-morbidity between bipolar disorder and PG has been estimated to be as high as 30% (Hollander, Buchalter, & DeCaria, 2000). Mood stabilizers are believed to have anti-impulsive effects (Pallanti, Quercioli, Sood, & Hollander, 2002), and are assumed to potentially be effective in the treatment of PG.

A recent meta-analysis of clinical trials using pharmacological interventions to treat PG identified 130 potential studies, though only 16 studies met the criteria for inclusion in the meta-analysis: (a) the target problem was pathological gambling, (b) the treatment was pharmacological, (c) the study was written in English, and (d) the study reported outcomes particularly pertaining to gambling (Pallesen et al., 2007). A total of 597 subjects were included in the outcome analyses of these studies. The analyses showed that at post-treatment the pharmacological interventions were more effective than no treatment/placebo, and yielded an overall ES of 0.78 (95% CI = 0.64, 0.92). A multiple regression analysis demonstrated that the magnitude of the ES at post-treatment was lower in studies using a placebo-controlled condition as compared with studies using a pre-post design (without any control condition). No differences between the three main classes of pharmacological interventions (antidepressants, opiate antagonists and mood stabilizers) were detected.

1.4.3 Behavioral Perspective

Learning mechanisms have been strongly implicated in the development of gambling problems (Blaszczynski & Nower, 2002; Sharpe & Tarrier, 1993), and according to the behavioral perspective excessive gambling is a learned maladaptive behavior. This perspective describes the development and maintenance of pathological gambling using principles of classical and operant conditioning. According to this perspective, gambling behavior is reinforced through a combination of financial rewards and increased autonomic arousal. The gambling environment becomes associated with feelings of excitement through classical conditioning, and this excitement becomes a conditioned reinforcer for continued gambling behavior (Sharpe & Tarrier, 1993). The sequence of outcomes in many forms of gambling (e.g., slot machines) follows a partial reinforcement schedule, where rewards occur with some wagers, but not all (Knapp, 1976; Skinner, 1969). Variable ratio schedules of reinforcement do not produce learning as quickly as fixed ratio schedules of reinforcement, but a greater resistance to extinction of behaviors acquired via variable ratio reinforcement schedules compared to other schedules is often found. This may explain the persistence in gambling despite large losses (Skinner, 1969). Furthermore, the greater the size of the reward, the more resistant the behavior will be to extinction. Thus, gamblers who experience large rewards early in their gambling career may be more susceptible to developing gambling problems (Custer, 1984; Griffiths, 1995; Ladouceur, et al., 2002; National Research Council, 1999).

Research has consistently shown that individuals prefer immediate over delayed rewards, and this may explain why the opportunity to obtain a monetary gain nearly instantly is tempting (S. H. Chung & Herrnstein, 1967). Further, the consequences of loosing money are often delayed, making them less likely to influence behavior

(Weatherly & Dixon, 2007). Gamblers may also habituate to losses over time which further limits the suppressive effect of the losses on the gambling behavior (Weatherly & Dixon, 2007). However, even though learning mechanisms such as variable reinforcement schedules play a role in the maintaining of gambling behavior, these mechanisms do not provide a full account for why some individuals develop pathological gambling behavior whereas others engage in gambling without developing a gambling problem.

According to social learning theory, individuals have a propensity to imitate behaviors they observe that are followed by positive reinforcement (Bandura, 1977), and individuals who observe the gambling behavior of family or friends may be more likely to gamble themselves (Abrams & Kushner, 2004; Brown, 1987). Abraham and Kushner (2004) further state that there is a potential for media-based vicarious reinforcement of gambling behavior, as big lottery winners are much more likely to receive media attention than are the millions of lottery losers.

Negative reinforcement may also explain engagement in gambling behavior. A number of studies have found elevated rates of depression and anxiety in pathological gamblers (Petry, Stinson, & Grant, 2005). It has been hypothesized that anxious or depressed individuals engage in gambling behavior in order to distract themselves from life stressors and escape from unpleasant cognitions (Blaszczynski & Nower, 2002; National Research Council, 1999). This is in line with the finding that gamblers report that they tend to play more in periods when they experience stress and personal problems (Donahue & Grant, 2007).

From a behavioral perspective, pathological gambling may also be explained as insufficient risk-avoidance, and studies have found that problem gamblers show less learned risk-avoidance compared to controls (Goudriaan, Oosterlaan, de Beurs, & Van den Brink, 2005). Failure to develop aversion towards behaviors repeatedly followed by negative consequences (such as gambling followed by losses) have been explained by a lack of ability to form conditioned associations (H. J. Eysenck, 1977). Impaired aversive conditioning may act as a mechanism which explains the development and maintenance of gambling problems. A theory of reward sensitivity was put forward by Gray (1982) who proposed that sensitivity in two systems, the behavioral inhibition system (BIS) and the behavioral activation system (BAS), explain individual differences in the degree to which individuals are sensitive to conditioned punishment (BIS) and reward (BAS) stimuli. BIS/BAS seem particularly relevant to gambling behavior because gambling involves both punishment (losing) and reward (winning) stimuli. High scores on BIS are associated with a passive avoidance of conditioned aversive stimuli, such as losing money in a gambling task. High scores on BAS are on the other hand associated with being sensitive to conditioned rewards, such as winning money in a gambling task (Brunborg et al., 2010). A recent study conducted by Brunborg et al. (2010) investigated the relationship between BIS, BAS, and risk-avoidance on the Iowa Gambling Task. The results showed that aversive conditioning contributed significantly to explaining the variance of risk-avoidance. Participants who did not show aversive conditioning exhibited less risk-avoidance compared to those who did show aversive conditioning. Hence, impaired aversive conditionability may explain why some individuals take greater risk and hence continue gambling and lose more money compared to individuals without this impairment. Brunborg et al. (2010) also suggested that aversive conditioning impairment

may constitute a biological vulnerability factor for developing gambling problems, hence suggesting an interaction between biological and behavioral factors in gambling.

Early behavioral therapies for PG focused on aversion techniques to decrease the positive reinforcement of gambling behavior and increase the punishment associated with gambling. This approach typically involved applying electrical shocks to gamblers during gambling episodes (Hodgins & Petry, 2004). However, aversion therapy for pathological gamblers has by and large been described in case reports and studies with fairly small sample sizes and short follow-up time (e.g., Barker & Miller, 1966, 1968; Koller, 1972; Seager, 1970). Although some reports have indicated positive effects, the number of relapses have often outnumbered the number of successfully treated cases (Barker & Miller, 1968; Seager, 1970).

Throughout the 1970s, other forms of behavioral treatments emerged and techniques such as behavioral monitoring, covert sensitization, relaxation techniques, and contingency contracting became popular (Hodgins & Petry, 2004; National Research Council, 1999). Behavioral interventions range from behavioral monitoring, stimulus control (e.g., limiting access to money), the reinforcement of non-gambling behaviors, contingency contracting (National Research Council, 1999), imaginal relaxation or desensitization techniques (McConaghy, Armstrong., Blaszczynski, & Allcock, 1988), cue exposure with response prevention (Echeburúa, Fernández-Montalvo, & Báez, 2000), and in vitro exposure with relaxation training (Blaszczynski, 1998).

However, much of the early research evaluating behavioral treatments for PG suffered from small sample sizes and lack of control groups (National Research Council, 1999). Larger outcome studies have later been undertaken and provide some support of

the effectiveness of behavioral techniques. One of the first studies applying random assignment when investigating the effectiveness of behavioral treatments for PG compared aversion therapy with imaginal desensitization (McConaghy, Armstrong, Blaszczynski, & Allcock, 1983). They found that the latter treatment obtained better effects at 1-year follow-up than the former treatment. Later, the same group of researchers compared the effectiveness of imaginal relaxation and imaginal desensitization in reducing pathological gambling, and found comparable effects for the two behavioral treatments (McConaghy, et al., 1988). In a later study McConaghy, Blaszczynski and Frankova (1991) randomly assigned 120 participants to one of four treatments: aversion treatment, imaginal desensitization, imaginal relaxation, or in vivo exposure. They found that participant receiving imaginal desensitization reported better outcomes at 1 month and up to 9 years later, although only half of the participants were contacted at follow-up. Since this is the only known study which have been conducted comparing these interventions more research is need to isolate the independent or unique contributions of these behavioral techniques (Hodgins & Petry, 2004).

1.4.4 Cognitive Perspective

Cognitive theories of pathological gambling assume that the acquisition of wealth (winning) is the primary motive for gambling. According to this perspective, pathological gamblers continue to engage in gambling activities, hoping or expecting to win money despite the adverse odds. The persistence of gambling behavior is explained through cognitive distortions contributing to erroneous perceptions of the links between random events. The gambler develops an illusion of control, and typically assumes greater

winning chances that he or she objectively has (Ladouceur, et al., 2002; Ladouceur & Walker, 1996).

The first consistently applied research paradigm investigating cognitive distortions in pathological gamblers was the "talk-aloud" procedure developed by Gabourey and Ladouceur (1989). This approach implied that the gamblers should speak aloud during their thought processes when gambling, while the therapist analyzed the verbalizations into rational or irrational thoughts. Irrational verbalizations have been found to characterize even low frequency gamblers (Gabourey & Ladoceur, 1989). Griffiths (1994) found that the proportion of irrational thoughts varied between 2.5% for irregular gamblers and 14% for regular gamblers. Using the talk-aloud method Delfabbro and Winefield (1999) also found that 14% of the verbalizations constituted irrational thoughts, however, when excluding all verbalizations not related to playing they found that 75% of the play-related verbalizations were irrational in nature. Critique of this method implies that even though a gambler utters an irrational though this does not necessarily imply that he or she actually believe in the content of the verbalization (Coventry & Norman, 1998).

Even though several studies have demonstrated that pathological gamblers exhibit a variety of cognitive distortions, such as skill misperception, illusion of control, skewed temporal orientation, superstitious beliefs, selective memory, and interpretative biases (Ladouceur, et al., 2002; Ladouceur & Walker, 1996), a lack of valid corresponding measures have for long impeded the systematic investigations of these factors (MacKillop, Anderson, Castelda, Mattson, & Donovick, 2006). A recent study using the Gamblers Belief Questionnaire (Steenbergh, Meyers, May, & Wehlan, 2002) to investigate irrational thought among gamblers showed however that levels of cognitive

distortions were related to severity of gambling problems, and that levels of cognitive distortions varied with gaming preference (Myrseth, Brunborg, & Eidem, 2010). A preference for playing skill games was associated with greater illusion of control compared to preference for chance games (Myrseth, Brunborg, et al., 2010). These results indicate that degree of gambling problems is positively related to degrees of cognitive distortions, but one can not draw conclusions of cause and effect as this study employed a cross-sectional design. It is possible that some gamblers develop a gambling problem because they have increased levels of cognitive distortions from the start, but it may also be that gambling excessively causes an increase in cognitive distortions. Moreover, cognitive distortions are also common among non-pathological gamblers, and therefore cognitive biases alone can not explain why some develop problems whereas others do not.

Four major components of cognitive treatments for pathological have been identified: education (about the random nature of gambling), increasing awareness about and identification of cognitive errors, questioning the validity of irrational cognitions, and restructuring cognitive distortions (Hodgins & Petry, 2004; Ledgerwood & Petry, 2005). Information about the independency of events (e.g., each roll with a dice is independent and the probability of getting a certain number does not increase as other numbers are rolled) and information about random number generators in slot machines are examples of factors which are aimed at increasing the patient's awareness of how specific cognitive distortions influence their gambling. Gamblers often show a fundamental cognitive error denoted as "the illusion of control" (the perception that one can influence the outcome of a randomly determined event), which may lead the gambler to persist gambling and believe that "the big win is around the corner" despite accumulative losses. In order to

eliminate such biases the therapist may uncover cognitive errors through Socratic questioning by which the validity of such cognitions are challenged. The therapist helps the patient finding rational statements to replace the irrational thoughts (Hodgins & Petry, 2004). In addition, self-monitoring of gambling or urges to gamble between sessions are a common components of cognitive therapy and standard thought record forms are often used, including description of situational, emotional, and cognitive precipitants to gambling behavior and challenges to any irrational cognitions (Hodgins & Petry, 2004).

Much of the early research investigating the effectiveness of cognitive therapy for PGs did not apply randomized controlled trials (e.g., Ladouceur, Sylvain, Letarte, Giroux, & Jaques, 1998), and few of the later controlled designs have applied purely cognitive therapy interventions (Hodgins & Petry, 2004; Petry, 2005). Elements of behavioral interventions such as stimulus control, self-monitoring, problem solving and social skill training, as well as relapse prevention are often included (Petry, 2005). In one randomized study applying a more purely cognitive approach, Ladouceur, Sylvain, et al. (2001) found significantly greater improvement for the treatment group compared to the waiting-list group at post-treatment. No waiting-list comparison was made at follow-up because the waiting-list group had received treatment in the interim. However, these data were based on analyses for completers only, and a significant proportion (41%) dropped out early in treatment. In a subsequent study Ladouceur et al. (2003) applied the same cognitive therapy in a group format, and found support for the effectiveness of group therapy. This study had a lower drop-out rate (26% compared to 41%), and 65% of those assigned to immediate treatment no longer met the criteria for PG at post-treatment compared to only 20% in the waiting-list control group. At 24-month follow-up, 68% of those who

completed the follow-up evaluation (33% of the original sample) did not meet the criteria for PG.

Another study of the efficacy of cognitive therapy which partly combined behavioral techniques was authored by Echeburúa, Báez and Fernández-Montalvo (1996). Here, a sample of 64 pathological gamblers was randomized to one of four conditions: Individual stimulus control and in vivo exposure with response prevention, cognitive restructuring delivered in a group format, a combination of the first two, and a wait-list control group. At 6-months follow-up the best improvement was associated with the first two treatment conditions which significantly outperformed the control group. Success was defined as abstinence or having had up to two gambling episodes in which the total amount spent did not exceed the amount gambled in the week prior to treatment. According to this criterion, the treatment success rates in the two first treatment groups were 75% and 63%, respectively. The effects of the combined individual behavioral treatment and cognitive group treatment did surprisingly not differ significantly from the results obtained from the wait-list control condition. This unexpected result may be attributed to the relatively small sample (16 per condition).

Although many studies investigating the efficacy of cognitive therapy for PG conclude that it seems to be effective, the inclusion of other interventions (such as relapse prevention, problem solving training etc) represents a confounding factor. Furthermore, few studies have demonstrated whether cognitive therapy which focuses on cognitive distortions actually modifies these illusions. Breen, Kruedelbach, and Walker (2001) assessed cognitive aspects of gambling in response to treatment using the Gambling Attitude and Beliefs Survey (Breen & Zuckerman, 1999), and found that the scores decreased after 28-days inpatient treatment and that reductions were mediated by level of

depression. However, they evaluated erroneous cognitions after a relatively long interval, and the treatment implied a multimodal approach. More research examining the specific mechanisms of change is needed (Petry, 2005).

Evidence also suggests that cognitive restructuring training may have preventive effects on PGs. A randomized controlled trial of a prevention program involving cognitive restructuring and problem-solving training found that gamblers who received cognitive restructuring training showed less cognitive distortions after completing the program and had lower scores on measures of PG after the program compared to a control group (Dorion & Nicki, 2007).

A recent study comparing a cognitive approach that specifically addresses cognitive distortions with other interventions (behavioral, motivational, and minimal intervention) found that the cognitive approach did not yield superior outcomes compared to the other interventions and the changes in gambling related cognitions were not greater for the cognitive therapy group (Toneatto & Gunaratne, 2009). The authors suggested that there are likely several pathways to therapeutic change that may not necessarily require modification of cognitive distortions. However, as this study included only 22-28 gamblers in each treatment condition the sample size may hence have been too small to detect differences between active treatment approaches.

1.4.5 Cognitive Behavioral Perspective

Sharpe and Tarrier (1993) integrated the behavioral and cognitive perspective in their cognitive-behavior model of problem gambling. They criticized the behavioral explanations of PG for not taking into account the complexity of gambling behavior and

for being purely descriptive. Sharpe and Tarrier (1993) acknowledge, in line with the behavioral perspective, that gambling initially is reinforced through a combination of financial rewards and increased autonomic arousal which is experienced as rewarding. The combinations of these reinforces provide a basis for quick acquisition of behavior and increased resistance to extinction. Gamblers further learn to expect wins to be intermittent which increases the likelihood of continuing gambling even after initial losses. The gambling environment becomes associated with the autonomic arousal which again becomes associated with gambling-related cognitions, such as accepting losses on the basis of expected future winnings, belief in a particular gambling system, or in a personal ability or skill in gambling. Through classical conditioning thoughts, situations and autonomic arousal act as triggers which increase the likelihood that the person will gamble. Triggers can hence be either external (situations, times, places etc) or internal (autonomic patterns of arousal or gambling related cognitions). Sharpe and Tarrier (1993) further argue that the probability of engaging in gambling once an urge develops is mediated by effective coping skills, such as ability to control autonomic arousal, ability to challenge irrational beliefs, ability to delay decision making and apply problem-solving skills, and ability to delay reinforcement. Hence, individual differences in coping skills and self-control are seen as mediating factors which may explain why some individuals lose control over their gambling behavior while others do not. Winning episodes tend to reinforce the beliefs about likelihood of winning (e.g. “I have a lucky day”). As losses are expected to occur sometimes, gamblers may continue to believe that “a big win is just around the corner” after a series of repeated losses (the gamblers fallacy). Studies have found that 60% of gamblers gamble more heavily after a loss than after a win (Leopard, 1978). Losses may also trigger further gambling because the gambler is motivated to “chase” their losses. Both wins and losses can hence produce cognitions that increase the

likelihood of further gambling. Sharpe and Tarrier (1993) suggest that poor coping skills operates as a predisposing factor for developing gambling problems, and this vulnerability may either develop through an environmental deficit or be a result of a biological predisposition. Hence, this perspective acknowledges the involvement of both cognitive, behavioral, and biological factors in the development of PG.

Within the cognitive behavioral perspective, treatment approaches vary in their relative focus on cognitive versus behavioral techniques (Hodgins & Petry, 2004). Functional analysis is a basic technique within cognitive behavioral treatments and consists of identifying triggers or precipitants to gambling. Certain events, times, days, people, and emotions may have been paired with gambling in the past and can act as triggers for gambling episodes or urges to gamble. Gambling episodes are often analyzed and in addition to breaking episodes into triggers, the positive and negative consequences of the episodes are evaluated. When analyzing the gambling episode the therapist helps the client to identify irrational thoughts, challenge the validity of the thought, and to restructure the erroneous beliefs about gambling. Yet another basic technique within this treatment approach is reinforcement of non-gambling activities, and clients are encouraged to plan rewarding non-gambling activities for high-risk times. Behavioral techniques, such as assertiveness training or relaxation training, may also be taught (Hodgins & Petry, 2004).

Sharpe and Tarrier (1992) presented a cognitive behavioral treatment approach for problem gambling, and suggested that the treatment consist of different stages: Stabilization, self-management, cue exposure, construction of alternative behavioral repertoires, improvement of self-esteem, and maintenance. In their strategic treatment approach they emphasized the importance of the client's cognitions and beliefs in

initiating and maintaining gambling behavior. They also recommend incorporating motivational interviewing techniques in the treatment approach (Sharpe & Tarrier, 1992).

Several randomized controlled trials of CBT for PG have been conducted during the last decade, and a recent meta-analysis of CBT trials suggests that CBT has robust short-term effects which also seem to endure up till 24 months post treatment (Gooding & Tarrier, 2009). However, most of the controlled research have compared CBT with waiting-list controls (Hodgins & Holub, 2007) but few have examined the relative efficacy of CBT and other intervention programs (Toneatto & Dragonetti, 2008). Some studies have shown that CBT failed to show superior results to brief interventions (Hodgins, Currie, el-Guebaly, & Peden, 2004; Petry, Weinstock, Ledgerwood, & Morasco, 2008; Toneatto & Dragonetti, 2008).

1.5 Integrated Models of Pathological Gambling

1.5.1 A Pathways Model of Problem and Pathological Gambling

Blaszczynski and Nower (2002) proposed a pathways model of problem and pathological gambling which integrates the contribution of multiple variables such as biological, personality, developmental, cognitive, learning theory and ecological determinants of problem and pathological gambling. They suggested the existence of various subgroups of gamblers, each with a distinct pathway characterized by specific vulnerability factors, demographic features, and etiological processes (see Figure 1).

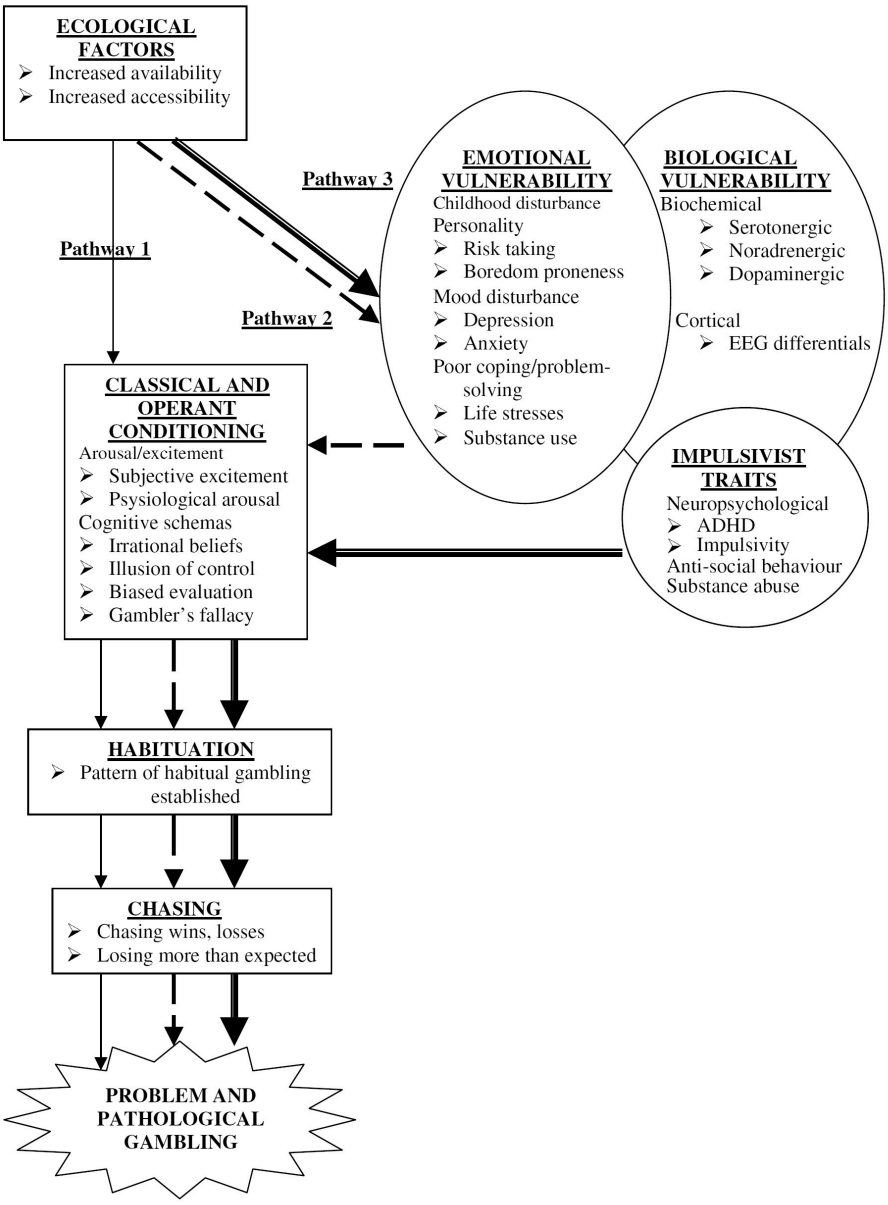

Figure 1. Integrated model of problem gambling

(reprinted from Addiction, vol. 97, Blaszczynski, A. & Nower, L., A pathways model of problem and pathological gambling, pp. 487-499, (2002) with permission from Blackwell Publishing)

The first group is labeled the *behaviorally conditioned problem gamblers* (Pathway 1), and Blaszczynski and Nower describe such gamblers as being "essentially 'normal' in character; that is, they do not show signs of premorbid psychological disturbance" (Blaszczynski & Nower, 2002, p. 496). These individuals lose control over gambling due to classical and operant conditioning and distorted cognitions related to the probabilities of winning. It is further proposed that this subgroup would benefit from minimal intervention programs. The second subgroup proposed is characterized by pre-morbid anxiety and/or depression, disturbed family and personal histories, poor coping and problem-solving skills, and affective instability. This group is labeled the *emotionally vulnerable problem gamblers* (Pathway 2). For these individuals, gambling serves the function of emotional escape through dissociation while gambling. The psychological dysfunction in these gamblers makes them more resistant to change, and Blaszczynski and Nower suggested that the treatment of these gamblers should also address their underlying vulnerabilities as well as their gambling behavior. The third group is called the *antisocial impulsivist problem gamblers* (Pathway 3), and in addition to an emotional vulnerability, this group is characterized by a biological vulnerability toward impulsivity, early onset, attentional deficits, antisocial traits and poor response to treatment. These gamblers are less motivated to seek treatment, have higher attrition rates and respond poorly to any form of intervention. According to this model, PG is a heterogeneous and multidimensional disorder with a complex interaction of genetic, biological, psychological and environmental factors (Blaszczynski & Nower, 2002). This model is however theoretical, and some of its aspects still remains to be empirically tested.

1.5.2 A Comprehensive Biopsychosocial Model of Pathological Gambling

Sharpe (2002) proposed a biopsychosocial cognitive-behavioral model of pathological gambling, in which she acknowledged both a possible genetic vulnerability (e.g., responsible for neurotransmitter dysfunction) as well as the impact of environmental circumstances in the development of gambling problems. Psychological traits, such as impulsivity, may also make the individual more prone to develop gambling problems given appropriate environmental circumstances. Early experiences (e.g., family attitudes towards gambling) may also confer a psychological vulnerability for developing gambling problems. She also acknowledges that levels of exposure to gambling opportunities may play an important role. Further, she suggests that life circumstances and stress can trigger the loss of control, and that gambling can function as an escape from a dysphoric mood and avoidance from stressful circumstances.

According to Sharpe (2002), early gambling experiences are likely to contribute to the development of cognitive biases, such as “the gamblers fallacy”, which may promote persistence in gambling both within and between gambling sessions. Cognitive biases may also lead the gambler to attend more strongly to positive consequences, such as wins, and may lead people to overestimate their chances of winning. Wins may also be associated with gambling-related arousal (excitement) and hence contribute to gambling through mechanisms of positive reinforcement. She further postulates that when the frequency of gambling behavior increases, it is likely that these patterns become automatic and less effortful. The individual has then a greater risk of losing control over the gambling behavior. The negative consequences of gambling further affect the mood and create greater autonomic arousal which leads to escape through gambling. When

these patterns have been sufficiently entrenched, they become difficult to break (see Figure 2).

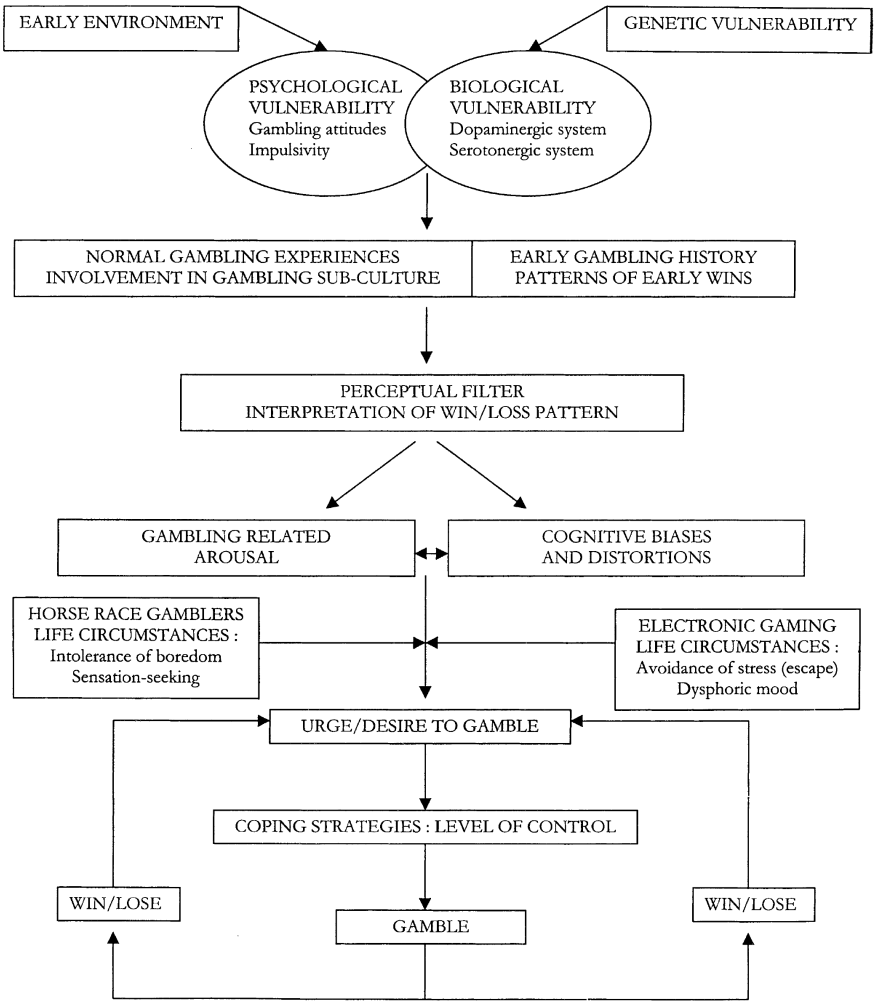

Figure 2. *A biopsychosocial model of pathological gambling*

(Reprinted from Clinical Psychology Review, vol. 22, Sharpe, L., A reformulated cognitive-behavioral model of problem gambling. A biopsychosocial perspective, pp. 1-25, (2002) with permission from Elsevier)

According to Sharpe (2002) the primary target in the short-term management of the gambling problem should be to stop the gambling behavior and break the associations between gambling cues and the gambling behavior. Behavioral strategies such as restricting access to money or gambling venues may be necessary. Furthermore, the association between arousal and gambling cues may be reduced through techniques such as imaginal desensitization. As memory biases toward winning experiences are common in gamblers, rehearsal of losing scenarios and the associated negative consequences may also be beneficial. She further underlines that challenging irrational beliefs about gambling is important in the treatment process. Additional treatment components such as psycho-education about probabilities of winning, stress management, problem-solving training, handling of relationship problems, and coping with co-morbidity may be important in order to stop gambling and restore the client's pre-gambling lifestyle (Sharpe, 2002).

1.5.3 Similarities and Differences between the Models

Both models acknowledge that ecological factors, such as increased availability and accessibility to gamble are important contextual variables that are associated with increases in probabilities to develop gambling problems. Classical and operant conditioning are recognized as important processes where the reinforcement schedules inherent in all forms of gambling lead to the acquisition of behavior which is resistant to extinction. The excitement and arousal in response to gambling become conditioned responses and may also act as reinforcers of gambling activities. Both models emphasize the importance of cognitive biases and irrational beliefs that develop as a result of

gambling experience. The cognitive biases become automatic and promote engagement in gambling. Furthermore, both models recognize an interaction between both genetic vulnerability and psychological (emotional) vulnerability, however Blaszczynski and Nower (2002) argue that the biological vulnerability and impulsivity is only a specific vulnerability factor for a sub-group of gamblers; the "anti-social impulsivist". Sharpe (2002) argue that the more impulsive an individual, the more likely the individual generally is to develop gambling problems. Sharpe (2002) describes the psychological vulnerability as consisting of positive attitudes towards gambling and impulsivity, and those who have more positive attitudes are more likely to engage in gambling behavior. On the other hand, Blaszczynski and Nower propose that emotional vulnerability, in which mood disturbance alters the risk for problem gambling, is only characteristic of a sub-group of gamblers; "the emotional vulnerable gamblers". The major difference between the two models seems to be that Blaszczynski and Nower (2002) argue that there exist distinct subgroups of gamblers, whereas Sharpe (2002) argue that there are individual differences in the degree to which each factor is involved in the development of PG (Sharpe, 2008).

1.6 Prevalence of Pathological Gambling

Shaffer, Hall, and Vanderbilt (1999) conducted a meta-analysis of all prevalence studies conducted in North America before 1997, and identified 120 studies to be included in their analysis. The lifetime prevalence of problem gambling among adults was estimated to be 3.85% (with a confidence interval [CI] of 2.94% - 4.76%), and the past year prevalence rate was 2.80%. For pathological gambling, the lifetime prevalence rate was estimated at 1.60% (CI = 1.35% - 1.85%), and the past year prevalence rate was

1.14%. These prevalence rates were later updated by Shaffer and Hall (2001), and the lifetime prevalence rate for adults was estimated at 1.92% for pathological gambling, with a past year prevalence rate of 1.46%. For problem gambling in the adult population, the new estimates were 4.15% and 2.54% for lifetime and past year prevalence, respectively.

Prevalence rates among adolescents are usually higher than prevalence rates for the adult population. Shaffer and Hall (2001) estimated lifetime and past year prevalence rates of pathological gambling among adolescents to be 3.38% and 4.80% respectively, while prevalence rates of problem gambling were estimated to be 8.40% and 14.60% for lifetime and past year, respectively.

Several national prevalence studies have been conducted in Norway over the past decade, and the prevalence rates have traditionally been lower than those observed in North America. Lund and Nordlund (2003) estimated the lifetime prevalence rates of the adult population in Norway to be 0.6% and 0.8% for pathological and problem gambling, respectively, and the past year prevalence rates were estimated to be 0.3% and 0.4% for pathological and problem gambling, respectively. Götestam and Johansson (2003) found the past year prevalence rate of the adult population in Norway to be slightly lower for pathological gambling, 0.15%, and slightly higher for problem gambling, 0.45%. In 2005, Kavli and Berntsen reported higher prevalence rates in a representative community sample in Norway; 1.9% for pathological gambling and 3.6% for problem gambling (Kavli & Berntsen, 2005). However, the difference in prevalence rates compared to the two earlier studies may be related to the different instruments used. The two former studies used the DSM-IV criteria (Götestam & Johansson, 2003) and the National Opinion Research Centre DSM-Screen for Gambling Problems (NODS; Lund & Nordlund, 2003) which is closely related to the DSM-IV criteria, while the latter study

used the Canadian Problem Gambling Index (CPGI; Kavli & Berntsen, 2005) which has been shown to create slightly higher prevalence rates than the DSM-IV criteria (Stinchfield, Govoni, & Frisch, 2007). The latter study also included a more narrow age span (18-30 years) which may also have affected the higher prevalence rates, as gambling problems consistently have been shown more prevalent among the younger population (Petry, 2005). In the most recent prevalence study in Norway among adults, Bakken and Weggeberg (2008) found past year prevalence rates of 0.6% for pathological gambling and 0.2% for problem gambling. However, this study had a fairly small sample size (N = 3500, in which the sample of problem gamblers were only n = 28) and the response rate was low (35%). Further studies are therefore needed.

Among the adolescent population in Norway, there has been an estimated prevalence rate of 1.8-2.5% for pathological gamblers and 1.9-3.5% for problem gamblers (Johansson & Götestam, 2003; Molde, Pallesen, Bartone, Hystad, & Johnsen, 2008). A study using samples from two previous published studies (Götestam & Johansson, 2003; Johansson & Götestam, 2003) found higher prevalence rates using the Lie/Bet Scale (LBS), where 0.54% of adults and 5.6% of youths were classified as probable pathological gamblers (Götestam, Johansson, Wenzel, & Simonsen, 2004). This underscores the fact that the choice of instrument has significant impact on the estimation of prevalence rates, and one should consequently be careful with comparing prevalence rates of studies using different instruments to measure gambling problems.

The prevalence of gambling worldwide is assumed to be on the rise due to expanding gambling opportunities and the general social approval of the gambling industry (Dowling, Smith, & Thomas, 2007; Ledgerwood & Petry, 2005). Although the prevalence rates reported by Shaffer and Hall (2001) were slightly higher than in the

former meta-analysis (Shaffer, et al., 1999), the authors conclude that further research is needed to determine whether the prevalence rates of gambling problems increase as the gambling opportunities become more readily available and more socially approved (Shaffer & Hall, 2001).

1.7 Risk Factors

Blaszczynski and Silove (1995) suggest that the development of PG is based on complex interactions among ecological, psycho-physiological, developmental, cognitive and behavioral components. Several risk factors for PG have been identified (Johansson, Grant, Kim, Odlaug, & Götestam, 2009), and demographic and genetic risk factors will be discussed later in this chapter, whereas the role of neurobiology has been discussed in a former chapter and the role of personality will be elaborated in a subsequent chapter.

1.7.1 Demographic Risk Correlates

Gambling pathology and gambling involvement are not uniform across demographic groups, and several demographic risk factors have been identified (Petry, 2005).

Age. Prevalence rates of PG are usually twice as high for adolescents as for adults (Petry, 2005; Shaffer, et al., 1999). A national study conducted in the U.S. found that the rates of pathological or problem gambling markedly declined with age: 3.1% among respondents between 18-29 years were classified as problem or pathological gamblers compared to 1.5% of those aged 30-39 years, 2.4% of those aged 40-49 years, 2.8% of

those aged 50-64 years, and 0.3% for those 65 years or older (National Research Council, 1999). Lund (2006) found a disproportionally high prevalence of gambling problems among young men aged 15-24 years compared to both other age groups (males) and women of similar age.

Gender. Studies have shown a three to five times higher proportion of male gamblers than female gamblers (Jacobs, 2000). In the overview of prevalence studies in the United States (U.S.) and Canada conducted by Shaffer et al. (1999), 18 of the general population surveys provided breakdowns of prevalence rates by gender. In 17 of the 18 studies, a greater proportion of men were classified as problem and pathological gamblers. In a national survey performed in the U.S., Welte, Barnes, Wieczorek, Tidwell and Parker (2001) found a significant gender effect where men had an approximate 40% increased risk of developing disordered gambling relative to women. Prevalence studies from other countries also confirm the association between gender and disordered gambling (Petry, 2005). In Norway, Götestam and Johansson (2003) found that among men 0.74% and 0.21% were classified as problem and pathological gamblers, respectively, while the corresponding rates for women were 0.19% and 0.09%. Lund (2006) also found that men were more likely to have gambling problems compared to females (5:1 ratio). However, as most studies addressing the validity of screening instruments are mainly based on male respondents, there is a concern that the instruments may be male biased and that the validity therefore may differ for boys and girls (Rossow & Molde, 2006).

Ethnicity. Ethnicity is a highly problematic, contested, and historically variable concept, with overlapping categories based on color, nationality, religion, culture and language (Roberts & Campbell, 2006). Still, belonging to certain ethnic groups or to an

ethnic minority has often been proposed as a risk factor for developing PG (e.g., Petry, 2005; Shaffer, LaBrie, LaPlante, Nelson, & Stanton, 2004). However, the concept of ethnic minority is problematic because what is considered an ethnic minority may differ from country to country. Frable (1997) offers a definition distinguishing between race and ethnicity. Accordingly, *ethnicity* is used to refer to "distinctions based on national origin, language, religion, food, and other cultural markers", whereas the term *race* is used to refer to "distinctions drawn from physical appearance (skin color, eye shape, physiognomy)" (Frable, 1997, p. 145). Several studies have reported that non-white ethnicity is associated with increased risk of development of PG (e.g., Potenza et al., 2001; Volberg, Abbot, Rönneberg, & Munck, 2001; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2004), but few studies have reported how they have defined ethnicity/race and whether or not they have differentiated between the terms race and ethnicity. Potenza et al. (2001) mixed the terms race and ethnicity, using categories such as "Caucasian race", "African race" and "Hispanic ethnicity". Volberg et al. (2001) defined ethnic minorities as being born outside Sweden, and did not further discriminate between counties of birth. Welte et al. (2004) refrained from using either terms (race/ethnicity) and merely stated that minority status (being African American, Hispanic, or Asian) is related to increased risk for gambling pathology.

Preferences for terms referring to racial or ethnic groups often change (American Psychological Association, 2010), and in the following I will use the terms reported in the specific studies when referring to different racial and/or ethnic groups. In a U.S. national survey, 71.5% of the respondents were Caucasian, while the rest of the sample comprised 11.1% African Americans, 10.2% Hispanics, and 7.3% individuals of other ethnicities (Gerstein, Hoffmann, Larison, & et al., 1999). In yet another study, higher rates of

pathological and problem gambling were found among Black Americans (4.2%) compared to Caucasian (1.8%) or Hispanics (1.7%) (National Research Council, 1999). Welte et al. (2001) found similarly that African-Americans were overrepresented with increased risk of developing gambling problems, and that all other groups except from Asian-Americans had significantly higher rates of gambling problems than Caucasians. In Shaffer et al.'s (1999) overview of the general population studies, 18 studies examined the prevalence of problem and pathological gambling in Caucasians compared to at least one other racial group, and in every study the proportions of minorities (non-Caucasians) identified as problem or pathological gamblers were higher than the proportion of Caucasians. The Norwegian prevalence study conducted by Lund and Nordlund (2003) also found significantly higher prevalence rates for ethnic minorities than for the ethnic majority; 5.5% of respondents born in a non-western country compared to 1.2% of respondents born in Norway were classified as problem or pathological gamblers.

Socioeconomic status. A lower socioeconomic status has quite consistently been associated with an increased rate of PG (Petry, 2005); however, this variable is often confounded with other variables that may be independently or interactively related to psychiatric disorders in general, for example, ethnic minority. Welte et al. (2001) reported that Americans with a lower socioeconomic status had higher than average rates in terms of current PG.

Education level. A low education level has frequently been associated with a greater risk for developing pathological gambling (Bakken, Götestam, Gråwe, & Wenzel, 2009), although this variable is often incorporated in operationalizations of socioeconomic status.

Mental health. Gambling often occurs in conjunction with other mental health problems, and this will be further elaborated under the section on co-morbidity.

1.7.2 Genetics

Genetic studies of impulse control disorders (such as PG) suggest similarities to other addictions (Brewer & Potenza, 2008). Genetic contributions have been estimated to account for as much as 60% of the variance in the risk for substance addictions, and similarly robust findings of genetic contributions have been found for PG (Brewer & Potenza, 2008). Approximately 20% of PGs have first-degree relatives with PG (Ibáñez, Blanco, & Saiz-Ruiz, 2002). Genetic factors have been estimated to account for between 35% and 54% of the liability for DSM-III-R symptomatology in PG (Eisen et al., 1998), in which genetics explained more of the variance when the PG was more severe. Yet, another study found that a significant proportion of the risk for PG (12-20% of genetic variation and 3-8% of environmental variation) was accounted for by the risk of alcohol dependence (Slutske et al., 2000). Furthermore, a meta-analysis of 17 family studies revealed a stronger familial effect for the father-son than the mother-daughter relationship, and a stronger influence for higher severity of PG (Walters, 2001).

The dopamine D_2A1 allele has been linked to substance abuse (Johansson, et al., 2009), and the presence of the D_2A1 allele has been shown to occur more often in PGs compared to controls (Comings et al., 1996). The D_1 receptor gene has also been associated with PG in a family study (da Silva Lobo et al., 2007). Several specific genes have been implicated as risk factors for PG (Comings et al., 2001). Comings et al. (2001) investigated 31 different genes involving dopamine, serotonin, norephinephrine, GABA

and other neurotransmitter systems, and found the following seven genes to be the most significantly related to PG: the dopamine receptor genes (DRD_2 and DRD_4), the dopamine transporter (DAT1), the tryptophan hydroxylase (TPH), the adrenergic $\alpha 2_C$ receptor (ADRA2C), the NMDA receptor (NMDA1) and the presenilin 1 (PS1) genes. The dopamine, serotonin and norepinephrine genes contributed equally to the risk for PG (Comings, et al., 2001).

1.9 Co-morbidity of Pathological Gambling

Knowledge regarding co-morbidity is important for generating hypothesis regarding the etiology of pathological gambling, as well as for designing prevention and interventions strategies. Few large representative population studies of co-morbidity in the gambling field have been conducted, and a sophisticated understanding of these relationships is lacking (Petry, et al., 2005). Few nationally based studies have been conducted, and most studies rely on telephone surveys suffering from high refusal rates. Some studies have evaluated co-morbidity of other psychiatric disorders in treatment-seeking pathological gamblers. However, in the following I will only describe general population surveys as they represent the most accurate account of co-morbidity.

1.9.1 Substance Use Disorders and Pathological Gambling

General population surveys have shown substance use disorders to be prevalent among problem and pathological gamblers. Bland, Newman, Orn and Stebelsky (1993) found in a Canadian survey in Edmonton, Alberta, that over 60% of pathological gamblers also had a lifetime substance use diagnosis, compared to less than 20% of the

non-gamblers. However, this study was based on a fairly small sample of gamblers (n = 30). Feigelman, Wallisch, and Lesieur (1998) found lower rates of co-morbidity in a telephone survey in Texas. They found that among respondents classified as lifetime problem or pathological gamblers (n = 265) 35% also had a substance use problem, while only 6.5% of the non-gamblers had a lifetime substance use diagnosis. In a more recent nationally based U.S. survey Petry et al. (2005) found that among the respondents classified as pathological gamblers (n = 195) 73% also had had an alcohol abuse/dependence, 38% had an drug abuse/dependence, and 60% had a nicotine dependence. The comparable figures for the non-gamblers (n = 42 898) in this survey showed that 25% had an alcohol abuse/dependence, 8.8% had a drug abuse/dependence, whereas the results for nicotine dependence were not stated.

1.9.2 Mood Disorders and Pathological Gambling

Studies have found that being a gambler increases the chances of having an affective disorder, and both Bland et al. (1993) and Petry et al. (2005) found that the proportion of pathological gamblers who also had an affective disorder was more than twice the rate of non-gamblers (33% and 50% for pathological gamblers vs. 14% and 19% for non-gamblers respectively).

Cunningham-Williams, Cottler, Compton, and Spitznagel (1998) investigated co-morbidity in a sample of 2 954 individuals in which 161 were classified as problem or pathological gambler, and found higher rates of major depression in problem and pathological gamblers compared to non-gamblers (9% and 5%, respectively), but also that gamblers without any gambling related problems were more likely to have both major

depression (OR = 1.7, 95% CI = 1.1.-2.6) and dysthymia (OR = 1.8, 95% CI = 1.0-3.0) compared to non-gamblers. Hence, simply gambling was related to depressive disorders in this sample. In a representative U.S. sample, Petry et al. (2005) found elevated rates of both major depression, dysthymia, and bipolar/manic episodes for pathological gamblers (37%, 13%, and 23%, respectively) compared to non-gamblers (12%, 4%, and 3%, respectively).

1.9.3 Anxiety Disorders and Pathological Gambling

Cunningham-Williams et al. (1998) found that among anxiety disorders only phobias were more likely to occur in problem and pathological gamblers (15%) compared to non-gamblers (10%) in their sample. Bland et al. (1993) also found a significant higher rate of phobias among pathological gamblers (17%) compared to non-gamblers (7%). They also found that a greater proportion of gamblers had obsessive-compulsive disorders (17%) compared to non-gamblers (2%) and slightly higher rates of panic disorders among gamblers (3%) compared to non-gamblers (less than 2%). Petry et al. (2005) found however a much higher rate of panic disorders among pathological gamblers (18%), while only 4% of the non-gamblers in their sample had a panic disorder. Similarly, they found much higher rates of phobias in pathological gamblers compared to non-gamblers; 5% of gamblers had agoraphobia compared to 1% of non-gamblers, 11% of gamblers had social phobia compared to 5% of non-gamblers, and 24% of gamblers had a simple phobia compared to 8% of the non-gamblers.

1.9.4 Other Axis I Psychiatric Disorders and Pathological Gambling

The DSM-IV-TR is based on five different dimensions (axis) in which axis I describes clinical symptoms and disorders grouped into different categories, including adjustment disorders, anxiety disorders, and pervasive developmental disorders (American Psychiatric Association, 2000). The co-morbidity of PG with other axis I disorder has not been extensively studied (Petry, 2005). The abovementioned study of Bland et al. (1993) found actually lower rates of schizophrenia and anorexia among pathological gamblers (0.0% for both disorders) compared to non-gamblers (0.7% for schizophrenia and 0.1% for anorexia). However, this study included only 30 pathological gamblers. Cunningham-Williams et al. (1998) had a larger sample of gamblers (n = 161) and found that 4% of problem and pathological gamblers also had schizophrenia, compared to 1% of the non-gamblers in their sample. They further found that 9% of the problem or pathological gamblers had a somatoform disorder, whereas only 4% of the non-gamblers had a somatoform disorder.

1.9.5 Personality Disorders and Pathological Gambling

Personality disorders (axis II disorders) reflect long-standing, dysfunctional personality traits, which is often characterized by impulsivity and emotional dysregulation. These traits are also associated with PG and several investigators have therefore focused on personality disorders as a possible vulnerability for PG (e.g., Bagby, Vachon, Bulmash, & Quilty, 2008; Blaszczynski & Steel, 1998; Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996; Steel & Blaszczynski, 1998).

Personality disorders considered to be part of Cluster B (borderline, histrionic, narcissistic and antisocial personality disorder), particularly antisocial personality disorder, have been more strongly associated with PG than personality disorders from Clusters A and C (Petry, 2005). In two community samples Bland et al. (1993) and Cunningham-Williams et al. (1998) found higher rates of antisocial personality disorder among gamblers (40% and 35%, respectively) than among non-gamblers (3% and 5%, respectively). In a national representative sample in the U.S., Petry et al. (2005) found that 23% of the pathological gamblers had an antisocial personality disorder, while only 3% of the non-gamblers had such diagnosis. They further reported higher rates for gamblers compared to non-gamblers for avoidant personality disorder (14% vs. 2%, respectively), dependent personality disorder (3% vs. 0.5%, respectively), obsessive-compulsive personality disorder (29% vs. 6%, respectively), paranoid personality disorder (24% vs. 3%, respectively), schizoid personality disorder (15% and 3%, respectively), and histrionic personality disorder (13% and 2%, respectively).

Bagby et al. (2008) examined the association between PG and personality disorders in a non-treatment seeking sample of pathological gamblers and a comparison group of non-pathological gamblers using both self-report and interview measures of personality disorders. They found much higher prevalence rates for the self-reported personality disorders, both for pathological and non-pathological gamblers. When the prevalence rates of personality disorders were assessed using structured clinical interviews (SCID-II), there were significant differences in prevalence rates between the two groups; 23% of the pathological gamblers and 5% of the non-pathological gamblers fulfilled the criteria for at least one personality disorder (Bagby, et al., 2008).

Studies reporting co-morbidity of personality disorders among treatment-seeking PGs range from 25% (Specker, et al., 1996) to 93% (Blaszczynski & Steel, 1998). Blaszczynski and Steel (1998) reported that in their sample of 82 PGs the average number of personality disorders per subject was 4.6. Bagby et al. (2008) examined the association between PG and personality disorders and found in their review 15 peer-reviewed studies examining gambling and at least one personality disorder. They concluded that there are great inconsistencies between the studies and that especially differences in design, sample and measurement contribute to this inconsistency. In the studies using self-report to assess personality disorders and PG the prevalence rates of personality disorders ranged from 87-93%, whereas in studies using interview-based assessment the prevalence raged from 25-61%. The studies also differed in terms of treatment-seeking vs. non-treatment-seeking samples. Treatment-seeking samples are more likely to be characterized by greater levels of distress and psychiatric co-morbidity compared to non-treatment-seeking samples which is likely to inflate the rates of personality disorders (Bagby, et al., 2008). Furthermore, most of the studies either did not use a comparison group or used a non-gambling comparison group. Bagby et al. (2008) argue that using a comparison group that gamble but are not pathological gamblers decreases the probability of conflating gambling behavior with personality pathology, and addresses more directly the potential differences between these two groups. Another weakness of all of the 15 studies was that none of them controlled for overlap with other axis I disorders, or for the overlap between different personality disorders. As axis I and axis II disorders are likely to share some co-morbidity a failure to control for axis I disorders may produce inflated rates of co-morbidity between PG and personality disorders.

1.10 Personality Factors

To date, results from studies on personality factors among gamblers have been largely inconsistent. Some have claimed the existence of an "addictive" personality type. This concept may have an intuitive appeal in explaining the high co-morbidity among different addictive behaviors (Petry, 2005). Even though similarities exist between different addictions, there is no empirical evidence for one underlying personality type predisposing for the development of addictions in general. More likely, there are multiple types of addictive personality factors which may be driven by different biological and learning processes (Zuckerman, 1999). PG has typically been linked to both externalizing psychopathology, such as antisocial personality disorder and drug and alcohol disorders (Petry, et al., 2005), as well as internalizing psychopathology such as anxiety and depression (Petry, et al., 2005). Studies addressing the general personality profile of gamblers are scarce and inconclusive (Álvarez-Moya et al., 2007). Most studies have only focused on specific personality factors, such as impulsivity, risk taking, poor coping, negative affects, novelty seeking or sensation seeking (Argo & Black, 2004) to mention a few. However, few have utilized a wider array of measures of personality.

In sum, there is a lack of research investigating the relationship between personality and pathological gambling, although it has been hypothesized that personality is one of many factors which contribute to the development and maintenance of PG (Bagby, et al., 2007; Blaszczynski, Steel, & McConaghy, 1997). More knowledge about personality characteristics could be helpful in understanding the antecedents related to PG, as well as for designing more effective prevention and intervention strategies. Studies applying broad measures and standardized personality tests are needed. In the following I will first give an outline of research conducted on pathological gamblers using the

Minnesota Multiphasic Personality Inventory (MMPI). Secondly, I will review some recent studies using the five factor model of personality to describe PG, and lastly the literature regarding the specific factors of impulsivity and sensation seeking in PGs will be reviewed.

1.10.1 MMPI Profiles of Pathological Gamblers

The MMPI is a comprehensive measure of psychopathology and personality which was developed by McKinley and Hathaway in 1943. The most typical use of the MMPI is to evaluate the profile configuration defined by the 10 standard scales, particularly the combination of the two or three scales with the highest scores. Previous studies have found that the MMPI profiles of gamblers closely resembled the profiles of alcoholics (Ciarrocchi, Kirschner, & Fallik, 1991). The PGs had a higher education level and socioeconomic status than the alcoholics, but there were no other differences between the two groups on any of the standard scales of the MMPI (Ciarrocchi, et al., 1991). This lends some support to the general theory of addictions, implying that personality characteristics may represent a general predisposition to develop an addiction, but that situation-specific factors influence the development of the specific addiction (Jacobs, 1986). Both the gamblers and the alcoholics were characterized by highest mean scores on scales 4-Pd (Psychopathic Deviate) and 2-D (Depression), with scales 7-Pt (Psychastenia) and 8-Sc (Schizophrenia) being the two next highest (Ciarrocchi, et al., 1991).

The most consistent findings of MMPI profiles among PGs are elevated scores on both scale 4-Pd and scale 2-D (Ciarrocchi, et al., 1991; Graham & Lowenfeld, 1986;

Moravec & Munley, 1983). This profile type is associated with significant psychopathology including depression, anxiety and substance abuse, and these individuals tend to be manipulative and show long-term maladaptive personality characteristics. In a study of 100 pathological gamblers, Graham and Lowenfeld (1986) found that a cluster analysis grouped 89% of the sample within four different clusters. The most common profile type which characterized 35% of the sample was a 4-9 profile. This type is associated with anti-social behavior; being immature, hostile, rebellious, restless and grandiose. The second cluster comprised 28% of the sample and showed elevations on scales 8-Sc, 7-Pt, 2-D, and 4-Pd. Persons with this combination tend to be suspicious, jealous, rigid, and withdrawn, and are often diagnosed with paranoid personality disorder or paranoid schizophrenia (Graham & Lowenfeld, 1986). The third cluster showed elevated scores on scale 2-D, and moderate elevations on scales 3-Hy (Hysteria), 4-Pd, and 7-Pt. Common for this profile is anxiety, alcoholism, depression, and passive-aggressive personality. The last cluster type showed a marked elevation on scale 4, and moderate elevations on scales 2-D, 7-Pt, and 9-Ma (Mania). These persons tend to be immature, irresponsible, demanding, and impulsive, and are often diagnosed with passive-aggressive personality or emotionally unstable personality (Graham & Lowenfeld, 1986). However, the authors do not specify what type of cluster analysis or distance measures were used, hence replications of these findings are difficult as different clustering techniques tend to generate somewhat different results.

In Graham and Lowenfeld's study (1986) a 4-2-7 profile type was most characteristics for the sample on average. This profile type is associated with impulsivity, inability to delay gratification of impulses, little respect for social standards and values, acting out as well as excessive drinking (Butcher, 2005). Impulsivity and inability to

delay gratifications has often been associated with gambling, and the immediate reward (both monetary and excitement/arousal) may explain why gambling is continued despite the negative consequences which are often delayed.

McCown and Chamberlain (2000) conducted a study of MMPI-2 profiles of pathological gamblers (N = 387) recruited from several different treatment settings. They identified two distinct clusters that accounted for 56% of the gamblers in treatment. A third cluster was identified in some gambling populations but not all. The first cluster, comprising 30% of the gamblers, was characterized by elevations on scales 1-Hs (Hypochondrias), 2-D, and 3-Hy. This group was characterized by depression, anxiety and overarousal, and gambled primarily to distract themselves from their miserable internal states. Women and video gamblers were overrepresented in this group. The second cluster (comprising 26% of the gamblers) was characterized by elevations on scales 9-Ma and scale 4-Pd and a low scale 0-Si (Social Introversion). Men were overrepresented in this group and they tended to gamble table-games, including poker and games of chance where there are dealers, and often gambled for higher stakes. A 4-9 profile is often common among gamblers with antisocial personality disorders. The third cluster that appeared in some but not all samples was characterized by elevations on scales 6-Pa (Paranoia), 7-Pt, and 8-Sc. McCown and Chamberlain (2000) related this pathology in the gamblers to misconceptions about the world and the failure to understand the basic laws of probability. They further suggested that the problems in this group were largely culturally based, and are more likely to occur among the poor or other socially deprived groups.

As early as 1987 Adkins, Kreudelbach, Toohig and Rugle acknowledged that gamblers do not represent a homogenous personality type and argued for the need for

individualized treatment planning based on the different personality types of gamblers (Adkins, Kreudelbach, Toohig, & Rugle, 1987). They investigated differences between gamblers with preference for luck vs. skill games on four of the MMPI scales: 2-D, 4-Pd, 9-Ma, and 0-Si. The results showed that the luck group scored significantly higher on scale 2-D, lower on scale 9-Ma, and higher on scale 0-Si. No statistical differences were found for the 4-Pd scale. The luck group was characterized by lower impulsivity, lower ego inflation, increased social alienation, and increased self depreciation. The clinical impression of the skill group was associated with being stimulus seeking, impulsive, grandiose and gregarious, which correlated with the higher score on the hypomania scale. The skill group typically played games at the race track or in the casino where the environments typically provided excitement and social contact. Even though no differences between the two groups were found on scale 4-Pd, both groups scored very high on this scale (more than 2 standard deviations above the mean).

1.10.2 The Five-Factor Model of Personality

The Five-Factor Model (FFM) of personality is one of the best documented personality theories (McCrae & Costa, 2003), and proposes that there are five main dimensions underlying individual differences in personality. These five dimensions can be described as follows: Neuroticism (easily upset, maladjusted, not calm), Extraversion (assertive, energetic, talkative), Openness (imaginative, independent-minded, intellectual), Agreeableness (cooperative, good-natured, trusting), and Conscientiousness (dependable, orderly, responsible) (Costa & McCrae, 1992). The FFM has demonstrated a

high heritability (Jang, McCrae, Angleitner, Riemann, & Livesley, 1998) and stability across different cultures (Jang, et al., 1998; McCrae & Costa, 1997).

Phillips, Butt, and Blaszczynski (2006) investigated the relationship between NEO Five Factor Inventory (NEO-FFI) profiles and gambling on mobile phones and suggested that the five factors have the potential to account for differences in gambling behavior. They hypothesized that individuals with high scores on Neuroticism would be prone to addictions and problem behavior (Phillips, et al., 2006). Further, Extraversion has been shown to predict sensation seeking which again consistently has been linked to problem behaviors, particularly risk taking (Trimpop, 1994). Consequently it is likely that gamblers, who often are characterized by sensation seeking and risk taking, would score high on this trait. High scores on Openness are often associated with being less conform, having more unusual and wide-spread interests (Anastasi & Urbina, 1997) and Phillips et al. (2006) speculated that high Openness may predict the likeliness of trying new games and technologies (i.e. gambling on mobile phones). Furthermore, because high scores on Agreeableness are linked to complying with appropriate usage and guidelines, Phillips et al. (2006) speculated that low Agreeableness would be linked to gambling. Low scores on Conscientiousness are associated with difficulties in working towards goals, and this lack of determination have been suggested to increase the likeliness of wasting time playing games (Phillips, et al., 2006). The main finding from their study was that people scoring low on Agreeableness reported spending more time playing games on their mobile phone than people with higher scores. Conscientiousness was negatively related to gambling on the mobile phone only because of its correlation with Agreeableness. Low scores on Agreeableness typically reflect low trust, selfishness, uncooperativeness, and being self-centered, and Costa and McCrae (1990) have indicated an association between low

Agreeableness and Narcissistic and Antisocial personality disorders which have also been linked to pathological gambling (Petry, et al., 2005).

Bagby et al. (2007) studied personality differences between non-treatment seeking PGs and non-pathological gamblers using the NEO Personality Inventory Revised (NEO PI-R) (Costa & McCrae, 1992). They found that Neuroticism and Conscientiousness were the only two domains with overall significant differences between the groups. The PGs scored higher on Neuroticism and lower on Conscientiousness. PGs scored significantly higher on four of the facet traits related to Neuroticism (Depression, Self-consciousness, Impulsiveness, and Vulnerability). At the same time, the PGs scored significantly lower on four of the facet traits related to Conscientiousness (Competence, Dutifulness, Self-discipline, and Deliberation). In addition, the PGs scored lower on one facet trait (Trust) of the Agreeableness domain. There were no differences between the two groups on any of the facet traits of Extraversion or Openness. Three of the four impulsivity-related facet traits (impulsiveness, self-discipline, and deliberation) distinguished PGs from non-pathological gamblers. Further, both groups demonstrated elevated scores on the excitement seeking facet trait. However, as no difference between the groups was obtained for the excitement-seeking facet the authors concluded that excitement-seeking, a personality construct closely resembling sensation seeking, was not specific for PG, but rather a characteristic of all those who gambled. They further concluded that PGs often show a personality profile which combines high impulsivity with emotional vulnerability (Bagby, et al., 2007).

A recent study of the relationship between pathological gambling and personality traits measured by the FFM supported the findings of Bagby et al. (2007) showing that PGs scored significantly higher on the Neuroticism domain, and significantly lower on

the Conscientious domain compared to a non-gambling control group on the Estonian Personality Item Pool-NEO which is analogous to the NEO-PI-R (Kaare, Mõttus, & Konstabel, 2009). No other overall difference was found between the two groups for the other three domains (Extraversion, Openness, and Agreeableness). However, significant differences between the groups were also found on two of the facet traits in the Extraversion domain (Activity level and cheerfulness) and on one of the Openness facet (Adventurousness). Also in this study no significant differences emerged for the excitement-seeking facet, even though the control group consisted of non-gamblers. This finding does not support the conclusion of Bagby et al. (2007) that excitement-seeking is characteristics of all those who gamble. However, the Estonian study (Kaare, et al., 2009) did not report T-scores (only raw scores), hence comparison to the normative sample or to the scores reported by Bagby et al. (2007) cannot be made.

High scores on Neuroticism and low scores on Conscientiousness seems to be the most consistent findings among PGs, and this combination is often associated with psychopathology in general. A meta-analysis conducted by Malouff et al. (2005), examining the relationship between the FFM and symptoms of clinical disorders, found that the typical pattern associated with clinical disorders was high scores on Neuroticism and low scores on Conscientiousness, Agreeableness and Extraversion.

1.10.3 Impulsivity

Pathological gambling is characterized as an impulse control disorder in the DSM-IV. The essential feature of impulse control disorders is the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person him/herself or to

others (American Psychiatric Association, 2000), indicating that impulsivity is an important aspect of the disorder. Several studies have proposed that PGs are characterized by higher rates of impulsivity than non-(pathological) gamblers (Alessi & Petry, 2003; Blaszczynski, et al., 1997; Carlton & Manowitz, 1994; Nower, Derevensky, & Gupta, 2004; Steel & Blaszczynski, 1998), and a few studies have demonstrated that impulsivity works as a mediating factor for the severity of the gambling problem (Alessi & Petry, 2003; Blaszczynski, et al., 1997; Steel & Blaszczynski, 1998). However, Langewisch and Frisch (1998) found that impulsiveness did not correlate with severity of gambling pathology among those characterized as PGs. Another study found that levels of impulsivity were not higher in gamblers than in other substance abuse control groups, which suggests that impulsivity may not be specific for gambling addiction, but rather associated with addictions in general (Allcock & Grace, 1988). Impulsivity has also been shown to be a mediator between depression and PG (Clarke, 2005). Other studies have also revealed that impulsivity is related to drop-out from treatment (Leblond, Ladouceur, & Blaszczynski, 2003), hence it has been suggested that individuals exhibiting high levels of impulsivity may need additional motivational enhancing treatment interventions (Leblond, et al., 2003).

Both PG and substance dependence are disorders characterized by a lack of self-regulation (R. Z. Goldstein & Volkow, 2002; R. Z. Goldstein, Volkow, Wang, Fowler, & Rajaram, 2001). The limited self-regulation is displayed when the addicted person is not able to inhibit the urge for the desired behavior (Goudriaan, Oosterlaan, De Beurs, & Van den Brink, 2008). The tendency to act upon acute impulses is referred to as *disinhibition* in the field of neuropsychology, whereas in personality theories it is often referred to as

impulsivity (Goudriaan, et al., 2008). During the last decade there has been an increased interest in the field of neuropsychology and pathological gambling.

Impulsivity is negatively related to self-regulation, and diminished neurocognitive self-regulatory functions have been found in PGs (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2006). Abnormalities in the brain reward circuitry have been found in neuroimaging studies of PGs (Potenza, Leung, et al., 2003; Reuter, et al., 2005), and studies have indicated specifically that diminished dopamine receptor availability (which may be related to substance dependence or a pre-existing vulnerability) causes a chronic reward deficiency in the brain which results in a vulnerability to engage excessively in rewarding behaviors (R. Z. Goldstein & Volkow, 2002). PGs have further showed a preference for immediate smaller rewards at the expense of larger delayed rewards in neurocognitive studies of decision-making (Goudriaan, et al., 2005).

It has further been argued that the reward deficiency will lead treated pathological gamblers to seek behaviors that normalize the deficient state (e.g., through gambling). Thus, the underlying reward-deficiency syndrome creates a vulnerability to relapse (Volkow, Fowler, Wang, & Goldstein, 2002). Goudriaan et al. (2008) investigated the role of self-reported impulsivity and reward sensitivity vs. neurocognitive measures of disinhibition and decision-making in the prediction of relapse in pathological gamblers. Self-reported impulsivity was assessed with the Barratt Impulsiveness Scale-11 and self-reported reward sensitivity was assessed with the BAS reward sensitivity subscale. They found that self-reported impulsivity and reward sensitivity did not significantly predict relapse, but that duration of the disorder and neurocognitive indicators of disinhibition and decision-making were significant predictors of relapse in PGs, explaining 53% of the

variance in relapse one year after treatment. This indicates that self-report may not be as accurate as neurocognitive measures of impulsivity and reward sensitivity.

1.10.4 Sensation Seeking

Sensation seeking has also been associated with impulsivity (Nower & Blaszczynski, 2006; Zuckerman, 1994), and Zuckerman (1994) has suggested combining sensation seeking with impulsivity to create a super trait called "impulsive sensation seeking". Sensation seekers can be defined as "those who seek novel, varied or complex sensations or experiences and who are willing to take risks for the sake of such experiences" (Breen & Zuckerman, 1999, p. 1099). Zuckerman (1999) proposed that PGs are the prototype of high sensation seekers, and suggested that engaging in gambling activities could be a way of maintaining optimum levels of stimulation (Zuckerman, 1994).

Although many studies have shown that PGs are high in sensation seeking (Anderson & Brown, 1984; Kuley & Jacobs, 1988), research in this field has been contradictory and several studies have failed to support this notion (Blanco, et al., 1996; Blaszczynski, Wilson, & McConaghy, 1986; Coventry & Brown, 1993; Hammelstein, 2004; Langewisch & Frisch, 1998; Sharpe, 2002). Langewisch and Frisch (1998) found that levels of sensation seeking did not predict gambling severity in pathological gamblers (as measured by scores on the SOGS). However, for the group of non-pathological gamblers the levels of sensation seeking were positively correlated with degree of gambling severity (SOGS scores). Breen and Zuckerman (1999) suggested that the failure of some studies to support the sensation seeking hypothesis in PGs may be due to

methodological weaknesses such as insufficient statistical power or the failure to control for age or sex, since adults are usually found to score lower on sensation seeking than adolescents, and as males are often higher in sensation seeking than females (Arnett, 1994). There has also been a controversy regarding the assessment of sensation seeking (Hammelstein, 2004). The most widely used measure of sensation seeking has been Zuckerman's Sensation Seeking Scale, Form V (SSS-V; Arnett, 1994; Zuckerman, Eysenck, & Eysenck, 1978). However, this scale has been criticized for using a "forced choice" format and its validity has been questioned (Arnett, 1994; Hammelstein, 2004). In the SSS-V it is referred to highly specific behaviors when operationalizing sensation seeking, and the measure explicitly includes risk taking and illegal behaviors. In a review of all studies investigating sensation seeking in pathological gamblers between 1970 and July 2003, Hammelstein (2004) found that the only study that could find significantly higher sensation seeking among pathological gamblers was one of the two studies using a different measure of sensation seeking than the SSS-V. The failure to show that pathological gamblers are characterized by higher sensation seeking than controls has led some authors (e.g., Sharpe, 2002) to conclude that PG is completely independent of sensation seeking. However, physiological data suggest that sensation seeking is related to PG. Low platelet MAO activity is regarded as a physiological correlate of high sensation seeking (Brocke, Beauducel, & Tasche, 1999; Schalling, Asberg, Edman, & Oreland, 1987; Schooler, Zahn, Murphy, & Buchsbaum, 1978; Zuckerman, Buchsbaum, & Murphy, 1980). Two studies investigating sensation seeking and platelet MAO activity in PGs (Blanco, et al., 1996; Carrasco, Saiz-Ruiz, Hollander, & Cesar, 1994) found that self-reported sensation seeking, measured by the SSS, did not support higher levels of sensation seeking in PGs compared to controls, but the physiological data suggested significantly *lower* platelet MAO activity in PGs (indicating that gamblers are sensation

seekers). Hammelstein (2004) hence suggested that the failure to support the notion that gamblers are high in sensation seeking in the studies using the SSS-V to assess sensation seeking may be due to improper operational definition of the concept inherent in the SSS-V.

Arnett (1994) suggested a new concept of sensation seeking which emphasizes *novelty* and *intensity* as the two components of sensation seeking, and he has developed a scale measuring the need for novelty and need for intensity of stimulation known as the Arnett Inventory of Sensation Seeking (AISS). In opposition to Zuckerman's concept of sensation seeking, Arnett does not presuppose that the sensation seeking trait must be expressed in norm-breaking or antisocial ways (Arnett, 1994). For this reason, the new scale does not include items covering specific types of behaviors, but uses instead more generally formulated items which aim to identify the underlying needs for novelty and stimulation (Hammelstein, 2004). Former studies have found that need for stimulus intensity, but not need for novelty, distinguish pathological gamblers from non-pathological gamblers (Nower, et al., 2004; Powell, Hardoon, Derevensky, & Gupta, 1999).

1.11 Sub-typing Pathological Gamblers

Vachon and Bagby (2009) acknowledge that converging lines of evidence suggest that PGs form a heterogeneous group with qualitatively unique subtypes, and they emphasize that an empirically derived taxonomy of PG may enhance therapeutic outcomes by providing more specific guidelines on how to tailor treatment for different subtypes of PGs. Different taxonomies of PG have been offered, where some have sub-

typed gamblers based on personality characteristics, while other have suggested subgroups based on assumed aetiology or motivations for engaging in gambling behavior. Some of the taxonomies bear resemblance to each other. Still, they may be supplementary rather than exhaustive.

Already in 1970, Moran acknowledged that gambling is likely to be a heterogeneous group of conditions whose common feature is excessive gambling resulting in disturbance for those involved. He suggested that the gambling problems seemed to arise from two main sources; social factors and individual characteristics. Based on a clinical study of 50 male gamblers he suggested the following five varieties of pathological gambling. The first category was named *subcultural variety,* and here the gambling was understandable in terms of the individual's social setting, such as social pressures. In the second variety, *neurotic variety*, the gambling was related to some stressful circumstances or emotional problems and the gambling provided some form of relief from the underlying tension, e.g. a disturbed marital relationship resulting in one partner gambling excessively in an attempt to escape from the problem or to punish the other partner. In the *impulsive variety*, the gambling was associated with loss of control and ambivalence towards the activity where symptoms of craving were apparent. In the *psychopathic variety*, the pathological gambling was seen as part of a global disturbance, namely psychopathy. In the last subgroup, *symptomatic variety*, the pathological gambling was associated with mental illness which appeared to be the primary disorder (Moran, 1970a, 1970b).

Blaszczynski and Nower (2002) also proposed different subtypes of problem gamblers in their Pathways model described earlier in this thesis. The three subtypes somewhat resemble those proposed by Moran (1970b). In both the behaviorally

conditioned gamblers in Blaszczynski and Nower's model and the sub-cultural variety type of Moran's taxonomy external environmental factors, such as availability and social setting, rather than internal underlying psychopathology are seen as central for developing gambling problems. The emotionally vulnerable problem gamblers may resemble Moran's neurotic variety. In both subtypes gambling is assumed to be related to stress and emotional problems and the gambling may consequently function as an escape. The third subtype in Blaszczynski and Nower's model, the antisocial impulsivist, resembles both the impulsive variety and the psychopathic variety in Moran's taxonomy in which impulsivity is a central factor. However, these qualitative taxonomies of gambling sub-types remain to be confirmed empirically and are both based on treatment-seeking PGs which embodies only 7-12% of all PGs (Slutske, 2006).

Yet another sub-typing of gamblers was suggested by Ledgerwood and Petry (2006) based on a measure of gambling experience. They also acknowledged that there may be different underlying psychopathology which explains why people gamble. For some, a neurological deficit may contribute to impulsiveness resulting in gambling, while others gamble to cope with painful experiences, yet others may develop a gambling disorder through direct behavioral conditioning. Based on a new instrument, the Gambling Experience Measure, a principal component analysis was performed which identified three factors or subtypes of gamblers: (1) *Escape gamblers*, which were significantly related to female gender, (2) *Dissociation gamblers*, which tended to have greater severity of gambling problems, and (3) *Egotism gamblers*, which were characterized as narcissistic and attention seeking. These subtypes bear resemblance to two of the former suggested subtypes by Blaszczynski and Nower (2002), namely the emotional vulnerable gamblers and the antisocial impulsivist. The behavioral conditioned

gamblers in Blaszczynski and Nower's model did not seem to be represented in Ledgerwood and Petry's classification. As the escape and dissociation gamblers both were more likely to use gambling as a means of escaping from painful experiences and these two factors were moderately correlated one may speculate whether it is clinically meaningful to separate these two.

Most of the abovementioned sub-typings of PGs are based on non-representative samples of treatment-seeking PGs and generalization to the total population of gamblers may be difficult. Stewart, Zack, Collins, Klein, and Fragopoulos (2008) cluster analyzed a community sample of 158 gamblers on the basis of scores from the Inventory of Gambling Situations (Turner & Littman-Sharp, 2006) and the Gambling Motives Questionnaire (Stewart & Zack, 2008) and found three distinct clusters of PGs based on their affective motivations to gamble: *the enhancement gamblers* (who gamble purely for positive reinforcement), *the coping gamblers* (who gamble for both positive and negative reinforcement, but primarily for negative reinforcement), and *the low emotion regulation gamblers* (who gamble for reasons other than direct regulation of affect). These three subtypes bear strong similarities to the previously suggested subtypes of gamblers offered by Blaszczynski and Nower (2002).

Vachon and Bagby (2009) also investigated subtypes of gamblers in a non-treatment seeking community sample of gamblers, and identified three subtypes based on the FFM; *the simple PG cluster* characterized by low rates of co-morbid psychopathology and trait scores near the normative mean, *the hedonic PG cluster* characterized by moderate rates of co-morbid psychopathology and a tendency toward excitement seeking and positive affect, and the third cluster labeled *the demoralized PG cluster* which was characterized by high rates of co-morbidity and a propensity toward negative affect, low

positive emotionality and disinhibition. The simple PGs were indistinguishable from non-gambling controls both in terms of scores on NEO-FFI and in terms of co-morbid axis I and II disorders. DeYoung (2006) proposed two higher order latent traits for the FFM; plasticity and stability. Vachon and Bagby (2009) found that the hedonic PG cluster was characterized by high plasticity, indicated by shared variance of Extraversion and Openness reflecting a tendency for curiosity, exploration, and sensation seeking. The demoralized PG cluster was characterized by low stability, indicated by shared variance of Neuroticism (reversed), Agreeableness, and Conscientiousness, reflecting the tendency to avoid disruption in social, emotional, and motivational domains. Plasticity is suggested to represent variance in the dopaminergic system which directs cognitive flexibility and exploratory behavior, while stability is suggested to represent variance in the serotonergic system, regulating emotions and the constancy of behavior (DeYoung, Peterson, & Higgins, 2002). This indicates that there might be a neurological substrate predisposing individuals to develop PG, which may be different for the various subtypes of PG.

1.12 Assessment of Pathological Gambling

The growing overall concern about problem gambling over the last three decades has fostered a need to identify problem gamblers among patients in mental health care services in order to better provide appropriate treatment services. Measures of PG are warranted in order to determine the extent of the problem in the general population as well. The amount and diversity of existing assessment instruments for PG is enormous (Albrecht, Kirschner, & Grüsser, 2007), and it is beyond the scope of this chapter to give a full description of all instruments. However, an overview of the most commonly used instruments is given in Appendix B. The most widely used and thoroughly evaluated

instrument to assess PG is the South Oaks Gambling Screen (SOGS) developed by Lesieur and Blume in 1987. This instrument was developed for use in clinical samples and is derived from the diagnostic criteria for PG in DSM-III-R (American Psychiatric Association, 1987). It has also been widely used in epidemiological surveys (Lesieur & Blume, 1993; Shaffer, et al., 1999). The items cover themes such as spending more money on gambling than intended, arguing with family members about gambling, borrowing money from different sources in order to gamble or pay gambling debts, etc. A SOGS score > 5 indicates probable pathological gambling, whereas a score of 3-4 is indicative of problem gambling. The SOGS was originally a measure of lifetime gambling problems, but the revised version, SOGS-R, also includes a measure of gambling problems over the past three months. The SOGS has demonstrated good consistency and convergent validity with other instruments used in the assessment of PG (Albrecht, et al., 2007; Lesieur & Blume, 1993). Yet, recent changes in the diagnostic criteria (e.g., in DSM-IV) have not been incorporated into the SOGS. The instrument has also been criticized for creating too many false positives (Culleton, 1989).

The National Opinion Research Center DSM-IV Screen for Gambling Problems (Gerstein, et al., 1999) is based on the DSM-IV criteria for PG, and contains both an assessment of PG during one's lifetime and over the last 12 months. The NODS is a diagnostic interview comprised of 34 items with total scores ranging from 0-10, and a score of 5-10 indicateing PG. The NODS has demonstrated good test-retest reliability, as well as reasonable sensitivity and specificity in recognizing PGs (Albrecht, et al., 2007).

1.12.1 Issues in Measuring Gambling Behavior

The variety of existing instruments for measuring gambling behavior, along with the relative scarcity of information about the reliability, validity and classification accuracy of the instruments in the field represents a challenge for both researchers and clinicians regarding the selection of instruments (Stinchfield, et al., 2007). The failure of using uniform outcome measures in treatment evaluation in the gambling field make it difficult to compare the relative efficacy of various approaches (Walker et al., 2006). Walker et al. (2006) proposed some guidelines for reporting efficacy of treatment outcome studies in the gambling field: measures of gambling behavior, measures of the problems caused by gambling and measures of the processes of change.

Most assessments of PG rely on self-report, and the validity and accuracy of such measures has been questioned. Factors such as impression management and the gamblers perception of social desirability can affect the validity of such measures. It has also been suggested that many PGs are notorious liars (which is also reflected in one of the diagnostic criteria), thereby raising the question of whether or not self-report data can be trusted (Stinchfield, et al., 2007). Wood and Williams (2007) investigated the comparative validity of different wordings of the questions used to assess gambling expenditure and found that slight variations in wording resulted in a significant variation in reported expenditures. They suggested both the ambiguity of the question and incorrect memory as possible causes for invalid self-reports.

Another measurement issue concerns the assessment period. The time frames of various instruments differ from lifetime, past year, past month(s) or past week. Because of this, different measures will have a different sensitivity when it comes to the detection of recent changes in gambling behavior, and the time period should be determined by the

purpose of the assessment. For instance, gambling often varies according to the amount of available resources, and gamblers tend to play more immediately after receiving their salaries; thus measures of gambling expenditure should correspond with the frequency of pay (e.g., weekly vs. a monthly basis).

Yet another issue is how to define recovery. As Nower and Blaszczynski (2008) state, recovery in PG is an imprecise concept which has been variously and inconsistently defined as the abstinence of clinical symptoms, abstinence of the diagnostic criteria, or the achievement of personal development, independence and function. Some clinicians and researchers promote a zero-tolerance of gambling, and consequently define recovery as total abstinence, while others will argue that a substantial reduction in the frequency and amount spent on gambling should qualify as recovery. Nower and Blaszczynski (2008) suggest that the concept of recovery should be clearly conceptualized and that the nature and extent of improvement should be evaluated along a spectrum which includes the measurement of both (a) frequency and the time spent gambling, (b) abstinence or controlled gambling that meets financial obligations, (c) abstinence of symptoms of impaired control, and (d) abstinence of negative consequences and an improved quality of life over time. As a result, assessments of PG should include measures of both gambling behavior (frequency, time spent gambling and net loss) in addition to perceived control and the negative consequences of gambling.

1.13 Relevance of the Present Research

Problem gambling poses a cost not only to the gambler and his or her family, but also to the society. The need for cost-effective treatment is apparent due to the high

prevalence rates of PG and the devastating consequences of the disorder. CBT is the most well documented treatment approach to PG (Gooding & Tarrier, 2009; Petry et al., 2006; Toneatto & Ladouceur, 2003), and is often considered the treatment of choice. However, despite the evidence of the effectiveness of CBT for PG, providing sufficient treatment for this group of patients is confronted with challenges. One is that, even though many countries have therapists trained in CBT, few of these have sufficient training in how to provide psychological treatment to the large number of individuals with gambling problems. Waiting lists for treatment are often long, and long distances between treatment services offering treatment for gambling problems in Norway may lower the accessibility of treatment. Another challenge is that only a small proportion of gamblers seek treatment (Slutske, 2006), and drop-out rates from treatment are usually high (Ladouceur, Gosselin, Laberge, & Blaszczynski, 2001). There is hence a need for designing effective prevention and treatment interventions that improve access to treatment and lower thresholds for treatment seeking, as well as keep gamblers in treatment.

One potential solution in order provide effective treatment interventions which improve access and lower the threshold for treatment seeking, is to examine whether the effects of pharmacological treatments can be comparable to the well documented effects of CBT. Effective pharmacological treatment of PG could significantly reduce clinicians' time and thereby reduce waiting lists and improve the cost-effectiveness of treatment for PG. As pharmacological treatment is less time consuming for the patient, it may also have the potential of lowering thresholds for treatment seeking. Further, because general practitioners can prescribe the medication it will be more available compared to traditional CBT in remote areas of the country.

So far, the effectiveness of psychological treatments compared to waiting lists is well established (Gooding & Tarrier, 2009; Pallesen, Mitsem, Kvale, Johnsen, & Molde, 2005) and pharmacological treatments for PG are also gaining support (Pallesen, et al., 2007). A meta-analysis of 22 studies of psychological treatment showed that the overall ES (Cohen's *d*) was 2.01 at post-treatment and 1.59 at follow-up (Pallesen, et al., 2005). In comparison, a meta-analysis of pharmacological treatment for PG, including 16 studies, yielded an overall ES (Cohen's *d*) of 0.78 at post-treatment (Pallesen, et al., 2007). Despite the need for effective prevention and treatment interventions, few, if any studies have so far compared the effectiveness of psychological and pharmacological treatments for PG. In the present research project I will therefore investigate the effects of the well established CBT approach compared to the less documented pharmacological treatment for PG.

The challenge regarding high drop-out rates and relapses (during and after treatment) raises questions of how to keep gamblers in treatment and make sustainable changes during treatment. Levels of psychopathology and personality characteristics have been suggested as factors affecting treatment attrition and treatment outcome in PGs (Dowling, 2009). Personality factors, such as impulsivity and emotional dysregulation, has further been suggested as a possible vulnerability for developing PG (Bagby, et al., 2008). Sensation seeking has also been suggested as a motivation for gambling, and some studies have found that pathological gamblers are typically characterized as sensation seekers. However, research in this field has been contradictory. Methodological issues such as small sample sizes, lack of control groups, failure to control for age and gender and different conceptualizations and measurements of impulsivity and sensation seeking may have contributed to the inconsistencies of findings.

The identification of psychopathology and personality factors in treatment seeking gamblers may be important in order to improve the effectiveness of clinical interventions and in designing more targeted interventions. The inconsistency of results regarding personality and psychopathology characterizing PGs highlights the need for more knowledge in this field. In the present research project I will therefore investigate levels of psychopathology and personality characteristics associated with treatment seeking pathological gamblers.

1.14 The Present Research Aims

The overarching aim of this thesis is to address the need for effective treatment for PG by investigating pharmacological treatment, which may lower the threshold for treatment seeking and improve access to treatment (avoid waiting lists), as well as address personality factors among PGs which may have implications for designing more effective and targeted treatment interventions. This aim will be addressed by (1) investigating the effects of a well established treatment (i.e., CBT) vs. a potentially effective and alternative treatment strategy (i.e., pharmacological treatment) and by (2) investigating psychopathology and personality characteristic among treatment-seeking pathological gamblers.

1.14.1 Aim I

The first aim is concerned with investigating the effectiveness of CBT compared to pharmacological treatment with escitalopram and whether the combination of the two

would yield a better effect than single treatment. The following research questions are addressed in the first study:

1) Does psychological treatment (CBT) yield a better effect than pharmacological treatment (escitalopram) for pathological gamblers?
2) Will the combination of psychological treatment (CBT) and pharmacological treatment (escitalopram) be more effective than CBT alone?

1.14.2 Aim II

The second aim is to investigate differences in impulsivity, sensation seeking and the five-factor model of personality among treatment seeking pathological gamblers, compared to a sample of non-pathological gamblers matched on sex and age. The following research questions are raised in the second study:

1) Which personality and demographic variables are significantly associated with pathological gambling?
2) Which of these predictor variables will still remain significant while controlling for the joint contribution of the other variables?

1.14.3 Aim III

The third aim is to identify psychopathology and personality characteristics among treatment-seeking pathological gamblers, and to investigate whether pathological

gamblers can be divided into subgroups based on psychopathology and personality characteristics as measured by the MMPI-2. The following questions are addressed:

1) How do pathological gamblers score on the standard scales of MMPI-2, and which profile type is characteristic for pathological gamblers?
2) Can the gamblers be divided into clinically meaningful subgroups or latent clusters based on the MMPI-2 profiles? If so, what characterizes these latent clusters, and how do they differ from one another?

2. Methods

2.1 Samples and Procedures

The three studies included in this thesis have been conducted in parallel, and the data in Study II and III were collected as part of the screening procedure for Study I. Study II and III also includes additional subsamples which will be described in more detail in the following.

2.1.1 Sample and Procedure: Study I

Potential participants were recruited through newspaper advertisements, through referrals from the national helpline and through referrals from health professionals in the region. To be included in the study, participants had to meet the following criteria: (1) be over 18 years of age, (2) fulfill diagnostic criteria for PG according to the DSM-IV (American Psychiatric Association, 2000), (3) not having used SSRIs during the previous six months, (4) not suffer from epilepsy or reduced liver or kidney functions, (5) exhibit no evidence of psychosis or organic mental disorder, (6) no concurrent alcohol or drug dependency, and (7) a willingness to undergo randomization. A total of 49 subjects contacted us for inclusion in the study, with four subjects failing to appear at the screening interview.

The initial screening interview included administering the NODS and the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) and the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II) in addition to registering information about background, gambling behavior and whether the participants fulfilled the inclusion criteria. Forty-five participants appeared for the screening interview, though

six subjects were excluded due to the following reasons: 1) already using SSRI (n = 3), 2) not fulfilling diagnostic criteria for PG (n = 2), and 3) hallucinating (n = 1). As a result, 39 subjects completed the assessment interview and met the criteria for inclusion in the study, but four withdrew from the study prior to randomization. Altogether, 35 participants were randomized to one of two groups: one group (n = 19) receiving individual CBT for eight weeks and one group (n = 16) receiveing only escitalopram for eight weeks, followed by a combination of escitalopram and individual CBT for the subsequent eight weeks. As a result, the study included two groups, but three treatment conditions (CBT vs. escitalopram vs. escitamlopram + CBT). The two groups will be referred to as the CBT group and the escitalopram + CBT group, but at the 8-weeks post-treatment assessment the second group had only received escitalopram.

Outcome assessments were conducted at pre-treatment, 8-weeks post-treatment (when one group had received CBT and the other escitalopram), 16-weeks post-treatment (when both groups had received CBT) and at 3- and 6-months follow-ups. Participants in the combined treatment condition were offered to continue receiving escitalopram until the completion of the 6-months follow-up. Only one participant quit the medication after the initial 16 weeks of treatment. Five participants dropped out of the study before starting the initial treatment. Hence, 15 subjects started treatment with CBT and 15 subjects started treatment with escitalopram. Additional six subjects dropped out before completion of the treatment while another two were excluded due to failure to comply with medication. One participant discontinued medication due to an adverse side effect (manic reaction).

2.1.2 Sample and Procedure: Study II

The net sample consisted of 90 pathological gamblers (PGs) (66 men and 24 women) and a contrast group (CG) of non-pathological gamblers (n = 66) matched on sex and age. The PGs comprised three different treatment seeking samples. The first sample (n = 29) comprised of the sample described in Study I (except from one individual who was recruited to the treatment study after Paper II were submitted for publication). The second treatment sample comprised patients who were recruited to participate in an outpatient CBT-based group treatment for PGs (n = 40) (Molde, Johnsen, Myrseth, & Pallesen, 2010), and the third treatment sample (n = 21) comprised patients recruited to an inpatient treatment study for PG (Molde, Foss, & Lorvik, 2007). Patients were recruited through advertisements in regional newspapers, referrals from the national helpline or by referrals from general practitioners. All of the participants in the PG group satisfied the DSM-IV diagnostic criteria for PG (American Psychiatric Association, 2000), fulfilling five or more symptoms (M = 7.8, SD = 1.6) of PG over the last three months. Their mean age was 37.9 years (SD = 12.7), and all participants signed an informed consent prior to inclusion in the study. Participants in the PG group completed the instruments as part of the screening procedure before entering treatment. The CG was also recruited through advertisements in a regional newspaper, and the participants were offered a gift certificate with a value of 12.5 EUR/18 USD/9.5 GBP (at the time of testing) for completing a research protocol consisting of measures of gambling behavior, impulsivity, sensation seeking and personality characteristics. In order to be included in the CG, the participants had to receive a score of three or less on the South Oaks Gambling Screen Revised (Lesieur & Blume, 1987). Two of the respondents received a SOGS-R score > 3 and were therefore excluded from the analyses. The mean age for the CG was 40.2 years (SD = 12.3).

2.1.3 Sample and Procedure: Study III

The study sample comprised 66 PGs (52 men and 14 women) who were recruited to two outpatient treatment programs at the University of Bergen, Norway. In total, 59% of the sample (n = 39) was recruited to an outpatient cognitive behavioral group treatment program for PGs, and 41% (n = 27) was recruited to out-patient individual treatment (recruited to participate in Study I). They were either self-referred, recruited by advertisements in a regional newspaper or via the national gambling helpline (n = 50), or they were referred by their general practitioner (n = 16). Out of a total of 113 referrals, 32 dropped out before initiating treatment, and 12 were excluded (three did not fulfill the diagnostic criteria for PG, one was excluded due to drug dependency, four were excluded due to delusions or hallucinations, and four were excluded because they were already using SSRIs). According to the DSM-IV (American Psychiatric Association, 1994), at least five out of a total of 10 criteria must have been present in the last three months in order to fulfill the diagnosis. All the participants in the current study fulfilled the DSM-IV diagnostic criteria for PG and were assessed using the Structured Clinical Interview for DSM-IV axis I Disorders (First, Spitzer, Gibbon, & Williams, 1995) in order to screen for co-morbid disorders. The SCID-I was administered to the patients during a screening interview conducted by four trained psychologists. A total of 69 subjects completed MMPI-2 after the screening interview, but three had too many items missing (> 10%) and had to be excluded from the analyses. Norwegian norms were used when calculating the MMPI-2 scores. The mean age of the sample included in the analyses was 38.1 years (SD = 12.0).

2.2 Power Analysis Study I

The power analysis for Study I was based on effect sizes from two former meta-analyses of psychological treatment (Pallesen, et al., 2005) and pharmacological treatment (Pallesen, et al., 2007) for PG in which a mean ES (Cohen's *d*) of 2.01 was obtained for psychological treatments, whereas a mean ES (Cohen's *d*) of 0.78 was obtained for pharmacological treatments, hence the difference in effect was 1.21. The power analysis was conducted with the G*Power 3.03 software (Faul, Erdfelder, Lang, & Buchner, 2007). With an estimated difference in ES of 1.20, power set to .80, alpha level set to .05 (two-tailed), allocation ratio = 1.0, 12 participants would be needed in each group.

2.3 Treatment Conditions in Study I

2.3.1 Cognitive Behavioral Therapy

The individual CBT consisted of eight weekly sessions (each session lasting approximately 50 minutes), and was based on both a therapist and patient manual (Myrseth, 2006a, 2006b). The treatment manuals were based on a manual originally developed for group therapy (Prescott & Skjerve, 2002; Skjerve & Prescott, 2002), in addition to a modification of this manual conducted at the University of Bergen (Molde, 2005a, 2005b). The manual outlines the structure of each session and provides information regarding issues to be covered. A motivational interviewing (MI) therapist style was utilized (Wulfert, Blanchard, & Martell, 2003), with a focus on the client's strengths in order to enhance self-efficacy with regard to change, stimulate commitment to change and helping clients to develop a plan for change. Functional analysis, the identification of triggers or precipitants to gambling and evaluating both positive and

negative consequences of gambling were all central components of the treatment. The treatment combined cognitive restructuring techniques, which involved training to identify cognitive distortions about gambling with in vivo and in vitro exposure with response prevention. The identification and restructuring of patient's erroneous beliefs about gambling were essential to all of the therapy sessions, and the gambler's thoughts prior to, during and following gambling episodes were therefore examined. Reinforcement of non-gambling activities and the development of alterative strategies to handle high-risk situations were also key treatment components. Information about principles of operant conditioning, erroneous beliefs and cognitive traps were provided. For more specific and detailed overview of the content of each treatment session, see Appendix C.

2.3.2 Escitalopram

Escitalopram is an SSRI and has a similar pharmacology to citalopram. Escitalopram was first approved in 2002 for major depression and in 2003 for generalized anxiety disorder, and is probably the most selective of the SSRI antidepressants and well tolerated (Black, Shaw, Forbush, & Allen, 2007). Because of its wide spectrum of therapeutic properties and the suggested effect of other SSRIs in treating PG, escitalopram was considered as a good alternative for the drug of choice. Moreover, at the time of initiating the present study, nalmefene (an opiate antagonist which has been associated with effectiveness in treating PG) was at the time still not approved by the Norwegian Medicine Agency, and the older equivalent, naltrexone, was associated with dose-dependent liver toxity. Escitalopram, which has been approved for the treatment of

major depression, generalized anxiety disorder, social anxiety disorder and panic disorder and has been associated with less negative side-effects than older SSRIs (Huska, Catalano, & Catalano, 2007). It was further assumed that general practitioners will be more comfortable with prescribing escitalopram for PG compared to the less familiar nalmefeme, and by choosing escitalopram as the study medication in present study the practical applicability of the results was considered to be greater.

Patients randomized to treatment with escitalopram (Cipralex) had to continue using escitalopram for a minimum of 16 weeks (eight weeks with escitalopram only, and eight weeks with a combination of escitalopram and CBT). The starting dosage was 5 mg/day for one week, continuing with 10 mg/day the following week and 20 mg/day the remaining treatment weeks. Patients in this group met with the study coordinator once a week for a short meeting (10 min) to receive medication for the following week and to report any adverse effects. The interaction was limited to a discussion of the clinical effects of the drug or adverse effects of the medication, and no advice concerning reduction in gambling behavior was given. After eight weeks of treatment with escitalopram, the patients were assessed with the primary and secondary outcome measures (see subsequent chapter) before initiating CBT, and after the eight weeks of combined escitalopram treatment and CBT, patients were offered to continue medication throughout the follow-up period (an additional six months). Only one subject did not wish to continue during this follow-up period.

2.4 Ethics

The studies were approved by the Regional Committee for Medical Research Ethics in Western Norway and the Norwegian Social Science Data Service, and the treatment study was also approved by the Norwegian Medicines Agency and registered in the European Clinical Trial database (EudraCT number 2006-000948-35). Written informed consent was obtained from all participants prior to inclusion in the studies, and participants were informed that they could withdraw from the study at any time without stating a reason. The research was carried out in accordance with the tenets of the Declaration of Helsinki.

2.5 Instruments

2.5.1 Instruments in Study I

The National Opinion Research Center DSM Screen for Gambling Problems (Gerstein, et al., 1999) is a structured interview which is comprised of 34 items in relation to gambling and the consequences of gambling, and consists of both a lifetime and past year gambling frame. The NODS is a diagnostic instrument based on the DSM-IV criteria for PG.

The Structured Clinical Interview for DSM-IV Axis I Disorders (First, et al., 1995) is a structured interview for assessing Axis I disorders based on the DSM-IV (American Psychiatric Association, 1994). The interview is divided into six modules: mood episodes and mood disorders, psychotic symptoms and disorders, substance use disorders, anxiety disorders, adjustment disturbances and other disorders.

The Gambling Symptom Assessment Scale (G-SAS; Kim, et al., 2001) measures past week gambling urges, thoughts and behavior, and comprises 12 items with response options of severity ranging from 0 to 4; thus the total score range is from 0 to 48. Scores of 31 or above indicate severe symptoms, scores of 21-30 indicate moderate symptoms, and scores of 20 or less signify mild symptoms. Cronbach's alpha for the G-SAS ranged from 0.89 to 0.96 over the five assessments in the present study.

The Pathological Gambling 100 mm Visual Analog Craving Scale (PGVAC; Hollander, Pallanti, Allen, Sood, & Rossi, 2005) is a self-rated five-item 100 mm visual analog scale measuring urges and control related to gambling. For each item, the scale ranges from 0 to 100, and the range of total scores is 0-500. Cronbach's alpha for the PGVAC ranged from 0.77 to 0.96 over the five assessments in the present study.

The Pathological Gambling Behavioral Self-Report Scale (PGBS; Hollander, et al., 2005) is a behavioral measure of pathological gambling and comprises three open-ended questions measuring net losses due to gambling during the last week, the number of occasions gambled over the last week (frequency), and the total time spent on gambling (in hours and minutes).

The Beck's Depression Inventory-II (BDI-II; Beck, Brown, & Steer, 1996) is a self-report measure of the severity of depressive symptoms, which comprises items rated from 0-3 in severity. The total scores range from 0 to 63, and the BDI-II has demonstrated good psychometric properties (Beck, et al., 1996). Cronbach's alpha for the BDI-II ranged from 0.71 to 0.90 over the five assessments.

2.5.2 Instruments in Study II

The South Oaks Gambling Screen – Revised (Lesieur & Blume, 1993) is a self-reporting screening instrument comprising 16 items that measure gambling problems which have occurred over the past three months. The scores range from 0 to 20. According to Lesieur and Blume (1987) a score of 5 points or higher serves to identify probable PGs. The SOGS is based on the DSM-III-R criteria for PG (Lesieur & Blume, 1987).

The NEO-Five Factor Inventory (NEO-FFI) is a short version of the NEO PI-R which consists of 60 items, and provides a brief, comprehensive measure of the five domains of the Five-Factor Model: Neuroticism, Extraversion, Openness, Agreeableness and Conscientiousness. Each subscale comprise 12 items, each scored on a 5-point Likert scale (0-4) (Costa & McCrae, 1992). The Cronbach alphas for the scales in the present study were 0.90 (Neuroticism), 0.83 (Extraversion), 0.78 (Openness), 0.66 (Agreeableness), and 0.82 (Conscientiousness).

The Eysenck Impulsivity Scale, Narrow Impulsiveness subscale (EIS-nI; S. G. B. Eysenck & Eysenck, 1977) is a measure of narrow (pathological) impulsivity consisting of 13 items regarding the ability to plan, postpone or think before acting. The instrument has good convergent validity with other measures of impulsivity, and has been found to consistently identify a specific form of impulsivity which correlates with the personality trait of psychoticism (Nower, et al., 2004). The instrument uses dichotomous answer categories of “yes” and “no”, and the Kuder-Richardson-20 value for the EIS-nI in this study was 0.81.

The Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995) is a 30 item measure of impulsivity using a 4-point Likert scale (1 = never/seldom, 2 = sometimes, 3 = often and 4 = always/almost always) to indicate the severity of each item. The BIS-11 consists of three subscales: Motor Impulsiveness, Attentional Impulsiveness and Non-planning Impulsiveness. The Cronbach alpha for the BIS-11 was 0.87 in the present study.

The Arnett Inventory of Sensation Seeking (Arnett, 1994) is a Likert-based instrument comprising 20 items, in which the response categories indicate how well each statement fits (1 = very well, 2 = somehow, 3 = not good, 4 = not at all). The AISS consists of two subscales: Need for Novelty and Need for Stimulus Intensity. The Cronbach's alpha for the AISS was 0.74 in this study, and 0.63 and 0.66 for the subscales Need for Novelty and Need for Stimulus Intensity, respectively.

2.5.3 Instruments in Study III

The Minnesota Multiphasic Personal Inventory-2 (Butcher et al., 1989) was used to measure symptoms and personality characteristics in the sample. In the revised version, MMPI-2, several items were replaced, others were modified, and several new scales were developed. MMPI-2 comprises 567 items, which make up 10 standard scales, three validity scales and a number of subscales. The 10 standard scales in MMPI-2 are: Scale 1-Hs (Hypochondrias), Scale 2-D (Depression), Scale 3-Hy (Hysteria), Scale 4-Pd (Psychopathic deviate), Scale 5-Mf (Masculinity-Femininity), Scale 6-Pa (Paranoia), Scale 7-Pt (Psychastenia), Scale 8-Sc (Schizophrenia), Scale 9-Ma (Mania), and Scale 0-Si (Social introversion). The validity scales are: L (Lie scale), F (Infrequency scale), and

K (Correction scale). T-scores are the most commonly reported output, in which the mean score derived from a norm group is set to a T-score of 50 for each scale, and the standard deviation is 10 T-scores. For the MMPI-2, a T-score > 65 is considered elevated, thereby indicating clinically significant problems (more than 1 ½ SD above the mean). On the MMPI-2, *uniform T scores* are used on all the ten standard scales, which ensure that the percentile ranks are equivalent across all the scales, i.e., a T-core of 65 would fall roughly at the 92nd percentile for all the ten standard scales (Butcher, 2005). Norwegian adult norms (for men and women) were used when calculating the MMPI-2 T-scores (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 2004).

The most typical use of the MMPI is to evaluate the profile configuration defined by the 10 standard scales, particularly the combination of the two or three clinical scales with the highest scores. Code types are summary indexes which include the most elevated scale scores of the eight clinical scales (excluding Scales 5-Mf and 0-Si). Code type interpretation often produces a more accurate and clinical useful interpretation than merely the interpretation of individual scales, but is considered more appropriate for populations with T-score elevations > 65 (Groth-Marnat, 2003). Code types should be well-defined (i.e., a T-score difference > 5 points between the lowest scale in the code type and the next highest clinical scale) in order to avoid the influence of measurement error (Graham, 2006).

2.6 Statistics

2.6.1 Statistics in Study I

The data were coded and processed using the SPSS version 15.0. Analysis of variance and chi square tests were used in order to examine demographic and clinical variables at pre-treatment. Modified intent-to-treat analyses (excluding the four individuals randomized to CBT and one individual randomized to SSRI + CBT who withdrew before the treatment was initiated) based on end point data were used throughout the study. Pre-treatment data were brought forward and used at all subsequent time points for participants who dropped out or were excluded during the first eight weeks of the study (n = 8), whereas 8-weeks post-treatment data were brought forward for participants who dropped out between 8- and 16-weeks post-treatment (n = 2). Repeated measures ANOVA were conducted to compare the effectiveness of the two different interventions in terms of treatment effects. Within-group effect sizes expressed as Cohen's *d* (with pooled SD) were calculated to express the magnitude of change from pre-treatment to 8- and 16-weeks post treatment, as well as to the 3- and 6-months follow-up. Paired sample *t*-tests were also conducted to compare pre-treatment scores with post-treatment and follow-up levels, and between-group effect sizes (Cohen's *d*) were also calculated for each variable at each assessment point. According to Cohen's benchmarks for interpreting effect sizes (Cohen, 1977), a value of 0.2 represents a small effect, 0.5 a medium effect and 0.8 a large effect. Clinical significance was evaluated as end state functioning (Kendall & Grove, 1988) and was defined by the proportion of subjects who reached a G-SAS score < 21 (indicating minimum to mild symptoms of PG).

2.6.2 Statistics in Study II

The data were coded and processed using the SPSS version 15.0, and T-scores for the NEO-FFI subscales were calculated using adult norms for men and women, respectively. Before conducting the logistic regression analyses, the inter-correlation between the independent variables were investigated in order to check whether multi-collinearity between the predictor variables could obscure the findings. All bivariate correlations between the predictor variables were less than .7, which is a prerequisite for doing a regression analysis as suggested by Tabachnick and Fidell (2001). Further, multi-collinearity diagnostic statistics for the logistic model were examined to exclude the possibility of interdependency between the predictor variables. High tolerance values may signal problematic multi-collinearity, which poses a threat to the validity of the logistic regression model. Tolerance values between .38 and .87, and VIF values between 1.14 and 2.61 indicated that the validity of the regression model was not threatened by multi-collinearity.

Crude (unadjusted) logistic regression analyses were conducted to examine whether different demographic variables (gender, age, education level) and personality variables (Neuroticism, Extraversion, Openness, Agreeableness, Conscientiousness, EIS-nI, BIS-11 and AISS) were separately related to PG. The predictor variables were subsequently entered into an adjusted (multivariate) analysis, thus controlling for every other predictor. The results are presented as odds ratios (OR) with 95% CI, and an OR is significant when the 95% CI does not include 1.00.

2.6.3 Statistics in Study III

The data were coded and processed using the SPSS, version 15.0. T-scores for the standard MMPI-2 validity and standard scales were calculated, and means and standard deviations were computed for each variable. In order to be able to explore whether subgroups existed among the PGs with respect to psychopathology and personality, different latent class clusters were explored in Latent GOLD 3 (Vermunt & Magidson, 2003). Latent class analysis (LCA) is a statistical method which classifies respondents into mutually exclusive groups with respect to a latent (not directly observed) trait (Notelaers, De Witte, Vermunt, & Einarsen, 2006). The LCA starts with the assumption that there is only one class and subsequently estimates up to *n* different classes until a latent class model is found that statistically fits the data (Goodman, 1974a, 1974b; McCutcheon, 1987). Magidson and Vermunt (2001) refer to such models as latent class cluster models since the T-nominal categories of the latent variable serve the same function as the T clusters desired in cluster analysis. An important difference from traditional cluster methods (such as *K*-means clustering) is that LCA is based on a statistical model that can be empirically tested (Magidson & Vermunt, 2002). As a consequence, determining the number of latent classes is less arbitrary than when using traditional cluster methods.

Modeling latent classes is an iterative procedure that determines the need for a certain number of clusters starting from the one cluster model. To determine how many clusters are needed in order to explain the associations, the Bayesian Criterion Information (BIC) is used (Magidson & Vermunt, 2004; McCutcheon, 1987). In addition to BIC it is also important that the latent variable, as in a traditional measurement model, explains the associations between the indicators. In Latent GOLD, this can be inspected

using bivariate residuals (BVRs) output. These should be lower than or equal to 3.84 which corresponds to a non-significant χ^2 with one degree of freedom, meaning that all bivariate associations are explained by the latent variable. In practice with many indicators, the reduction in BVR should be at least 85% (Notelaers, De Witte, Vermunt, & Einarsen, 2006), which can prevent Latent GOLD from adding an additional cluster for each pair or few pairs of BVRs that is higher than 3.84.

3. Results

3.1 Results Study I

There were no differences between the two groups at pre-treatment in terms of demographic variables, gambling severity (expressed as scores on the NODS) or on any of the primary and secondary outcome measures. The results showed a significant time effect on all measures for both treatment groups; although no significant group or time by group effects were found. Between-group effect sizes for all outcome measures at all assessments were small. At 8-weeks post-treatment, when one group had received CBT and one group escitalopram, paired sample *t*-tests revealed that both groups had improved significantly on the G-SAS and on the net loss measure compared to pre-treatment scores. The CBT group also improved significantly on the PGVAC from pre-treatment to 8-weeks post-treatment. All within-group effect sizes for the CBT group were large, while the escitalopram + CBT group obtained somewhat lower effect sizes on all measures. At 16-weeks post-treatment, the CBT group also improved significantly on the G-SAS and the PGVAC, while the escitalopram + CBT group improved significantly on the net loss during the last week. Within-group effect sizes for both groups were large. At the 3- and 6-months follow-up, both groups still showed significant improvement on most measures compared to pre-treatment scores and the corresponding within-group effect sizes were moderate to large.

3.2 Results Study II

In Study II, the univariate logistic regression analysis, which comprised 14 predictor variables and PG status (0 = not PG, 1 = PG) as the criterion variable, revealed

that 12 of the 14 predictor variables showed a significant relationship ($p < .05$) with PG: Education level (OR = 0.27), Neuroticism (OR = 1.11), Extraversion (OR = 0.96), Openness (OR = 0.88), Agreeableness (OR = 0.96), Conscientiousness (OR = 0.89), EIS-nI (OR = 1.32), Motor Impulsiveness (OR = 1.28), Attentional Impulsiveness (OR = 1.34), Non-planning Impulsiveness (OR = 1.30), Need for Novelty (OR = 0.90) and Need for Stimulus Intensity (OR = 1.01). There were no statistical significant differences between the PGs and the CG in terms of age (t = -1.12, *n.s.*) or gender (χ^2= 1.2, *n.s.*). In the multivariate logistic regression analyses, only four of these remained significant: Neuroticism (OR = 1.18), Openness (OR = 0.76), EIS-nI (OR = 1.48) and Need for Stimulus Intensity (OR = 1.19) were significantly related to PG. The Nagelkerke R squared was 0.71 in the adjusted analysis, suggesting that a large amount of the variance in PG status was explained by the predictor variables.

3.3 Results Study III

The mean profile for the total sample was a 2-7 two-point code type, and 14% of the sample had a 2-7/7-2 profile. On Scale 2-D 53% of the sample had a T-score > 65 (M = 67, SD = 14.7), whereas nearly 49% of the sample had a T-score > 65 on Scale 7-Pt (M = 65.4, SD = 16.3). No other scales had elevated mean T-scores.

Scores on the eight clinical MMPI-2 scales were entered into the LC analyses. Hence, the indicators were treated as interval measures in Latent Gold. Following the BIC criterion, a latent class model distinguishing three clusters fitted best with the data (BIC = 4200). However, the three-cluster model was associated with three large bivariate residuals (BVRs), indicating that scale 1-Hs and 3-Hy, scales 2-D and 7-Pt and scales 7-Pt and 8-Sc had shared variance. Because these pairs of scales share common items (20

items, 13 items, and 17 items, respectively) it is not surprising that the BVRs are large. There are several ways to deal with these problematic bivariate residuals (Magdison & Vermunt, 2004). The first way consists of adding more clusters. However, subsequent four- and five-cluster models provided higher BIC values (BIC = 4218 and 4248, respectively). The second way is to increase the number of latent variables in the model, by using latent class factor models. Models 6 and 7 showed that adding a second and third latent variable provided a better fit as indicated by lower BIC values (4143 and 4121, respectively). However, these models portrayed again large BVRs. The third alternative is to relax the local independence assumption. This approach is extremely useful when an external factor which is not really related to the latent class variable is responsible for creating an "irrelevant" association between indicators. Allowing such a direct-effect parameter (Hagenaars, 1998) may be advisable if for examples symptoms are strongly related (Uebersax, 2009). Since the high BVRs in the 3 cluster model were related to three pairs of scales which share common items and elevations on these pair of scales often occur together (e.g. Graham, Lowenfeld, 1986; McCown & Chamberlain, 2000) we relaxed the assumption of local dependency. In a subsequent model, we therefore allowed scales 1-Hs and 3-Hy, scales 2-D and 7-Pt, and scales 7-Pt and 8-Sc to covary. The results portrayed that model 8 was associated with the lowest BIC (4120) of all the tested models. This solution was therefore considered to represent the best fit. In this three cluster solution with local dependencies, the classification errors were estimated to be 1.6%, which equals the number of erroneously classified subjects in adjacent latent classes.

Results from the latent class analysis hence suggested that there were three distinct latent clusters of gamblers in the sample: One latent cluster which had a profile within the

normal range scoring rather low on psychopathology, and two latent clusters with elevated profiles. Latent cluster 1 (n = 36) comprised 54.5% of the sample and had all mean scores of the eight clinical scales in the MMPI-2 profile within the normal range (T< 65). Latent cluster 2 (n = 22), which comprised 33.3% of the sample, had elevated mean scores (T > 65) on six of the eight clinical scales: 1-Hs (M = 69.9, SD = 13.4), 2-D (M = 78.3, SD = 8.6), 3-Hy 8 (M = 71.6, SD = 10.6), 4-Pd (M = 71.3, SD = 11.4), 7-Pt (M = 73.5, SD = 6.8) and 8-Sc (M = 67.0, SD = 10.6). This cluster was characterized by a 2-7 profile type. Latent cluster 3 (n = 8), comprising 12.1% of the sample, had a profile with elevations on seven of the eight clinical scales: 1-Hs (M = 85.4, SD = 15.8), 2-D (M = 86.8, SD = 5.7), 3-Hy (M = 79.9, SD = 6.8), 4-Pd (M = 73.0, SD = 19.0), 6-Pa (M = 90.6, SD = 20.3), 7-Pt (M = 94.9, SD = 7.2), and 8-Sc (M = 101.6, SD = 15.4). This latent cluster was characterized by an 8-7 two point code type.

Demographic characteristics also differed between the latent clusters. Latent cluster 2 and 3 had higher mean age (41 and 46 years respectively) compared to latent cluster 1 (35 years). Latent cluster 2 and 3 had also larger percentage of gamblers with low education (23% and 38% with elementary school only) compared to latent cluster 1 (8%), and a larger percentage of gamblers in latent cluster 2 and 3 were unemployed or on social security payment (40% and 62%, respectively) compared to latent cluster 1 (17%).

4. Discussion

The specific findings and research aims of the three studies are discussed in detail in the papers. The following section consists of a general discussion of the main findings, considerations regarding methodological issues, clinical implications as well as future directions.

4.1 Summary of Findings and General Discussion

4.1.1 Treatment of Pathological Gambling

The first aim of this thesis was to investigate the effectiveness of CBT and escitalopram in the treatment of PG. The main finding of the treatment study was that irrespectibly of treatment type, there was an effect of time. This is in line with previous findings that CBT is effective in treating PG (e.g., Gooding & Tarrier, 2009). Escitalopram has also previously shown promising effectiveness in two open-label trials with treatment of PGs (Black, et al., 2007; Grant & Potenza, 2006). The specific CBT approach used in the present study has previously been found to be effective in a group format in two former controlled studies compared to waiting lists (Molde, Johnsen, et al., 2010; Myrseth, Litlerè, Støylen, & Pallesen, 2009). Still, it is possible that the improvement on the different outcome measures can be attributed to other factors than the specific treatments delivered. Threats to statistical conclusion validity or internal validity may lead to invalid inferences about the co-variation between two variables (e.g., treatment delivered and outcome measures) (Shaddish, Cook, & Campbell, 2002). Factors such as history or maturation may have affected the results. History refers to all events that have occurred between pre-treatment and post-treatment that could have produced

the observed outcome in the absence of treatment (Shaddish, et al., 2002). For example, a partner may have presented the gambler with an ultimatum; “either you stop gambling, or I will leave you”. This may have motivated the gambler to stop gambling irrespectively of the treatment delivered. Maturation refers to natural changes that would occur even in absence of treatment (Shaddish, et al., 2002). An example of this could be changes in gambling behavior due to changes that occurred in the gambling market in Norway (removal of all EGMs from the market in July 2007) during the treatment implementation phase. This may well have affected the gamblers, and changes in their gambling behavior during this period may be due to changes in the market (loss of opportunity to gamble) rather than a result of the treatment delivered. However, as most of the gamblers started treatment after the EGMs were removed from the market and nearly 70% of the total sample had other forms of gambling as their main problem, this change in the market could possibly only have affected a small proportion of the participants.

Furthermore, there is reason to believe that gamblers seek treatment when the problems have accumulated and they experience distress and negative consequences of their gambling. Hence, the improvement of both groups may be a result of a “floor-effect” and regression towards the mean and thereby reflecting natural recovery. The improvement may also reflect non-specific treatment factors, (e.g., motivation, social support, expectancies), intra-therapeutic factors (e.g, empathy, compassion, warmth; Korn & Shaffer, 2004) rather than the unique impact of the treatments delivered. However, as CBT is well documented and has shown effectiveness compared to waiting lists in numerous studies (Gooding & Tarrier, 2009; Molde, Johnsen, et al., 2010; Myrseth, Litlerè, et al., 2009), it is plausible to attribute the improvement in the CBT group to the treatment delivered and not merely the passing of time. The possibility that the

improvement in the escitalopram + CBT condition reflects a placebo effect will be further discussed in the chapter “Methodological Considerations”. The design of the present study do not allow for making assumptions of the specific mechanisms of change. Thus, even though both treatment conditions seem to be effective, the relative effectiveness is unclear.

The second main finding from this thesis was that there were no significant group effects. This finding may indicate that the treatments were equally effective as opposed to the previous meta-analyses which have found greater ES for psychological treatment compared to pharmacological treatment (Pallesen, et al., 2005; Pallesen, et al., 2007). The discrepancy of the present finding and the previous meta-analyses may have several explanations. Firstly, Pallesen et al. (2005) found considerably larger ES compared to a previous meta-analyses of psychological treatment for PG (Oakley-Brown, Adams, & Mobberly, 2003) and suggested that the inclusion of pre-post designs in the latter meta-analyses inflated the overall ES obtained by Oakley-Browne et al. (2003) which only included randomized controlled trials. Hence, the difference in effect (between psychological and pharmacological treatment) may not be as large as indicated. Alternatively, differences in the effects of psychological and pharmacological treatments may exist but were not detected in the present study due to insufficient statistical power to detect a possible relationship (the issue of power will be further elaborated under the chapter Methodological Considerations). Heterogeneity of the gamblers within each treatment condition may also make the detection of an effect more difficult, especially when the size of the present sample was fairly small. The dependent variable Net Loss (during the last week) had large standard deviations, indicating large within-group variance, making it more difficult to obtain statistical significant results. However,

because the between-group effect sizes, which are independent of sample size, were small for all outcome measures at all assessments, it seems reasonable to infer that the results indicated that treatment with escitalopram was as effective as CBT, and the combination of CBT and escitalopram seemed not to have any additional effect. The CBT group showed significant improvement on most measures both on a short- and long-term basis. Yet for the escitalopram condition there was only one assessment (after eight weeks) with escitalopram as single treatment. At the following assessments, this group had received both escitalopram and CBT and the improvement at these assessment points can thus be attributed to both treatment modalities. Because pharmacological treatments often have high placebo effect in the beginning (e.g., Blanco, Petkova, Ibáñez, & Sáiz-Ruiz, 2002), the improvement on the outcome measures for this condition may be attributed to a placebo effect. Further studies investigating the long-term effect of escitalopram are needed.

The sample size in the present study was fairly small. Hence one cannot exclude the possibility that the two groups indeed were different even though no statistically significant differences were observed in respect to the outcome measures. Such differences may have confounded the results, and lead us to conclude that the different treatment modalities seemed to be equally effective even though the CBT may have been more effective than escitalopram (or vice versa). Further larger scaled studies are needed to follow up the results form this pilot study and to further evaluate the relative effectiveness of CBT vs. escitalopram and the combination of these treatments.

The presence or absence of co-morbidity may have influenced the effects of the treatments. For instance, it is likely that PGs with co-morbid affective and/or anxiety disorders would benefit more from treatment with escitalopram as the drug also targets

the co-morbid disorder (Myrseth et al., 2010). Neurobiological and genetic models of PG suggest that specific deficits or dysregulations of structural and/or functional abnormalities in the brain predispose some individuals to respond preferentially to certain activities which implicate these neurotransmitter systems (Nower & Blaszczynski, 2008). In accordance with this, it is likely that gamblers with certain neurobiological dysfunctions may profit more from pharmacological treatments that target these specific neurotransmitter systems. Some gamblers may have a dysfunction in the serotonin system, while others primarily have a dysfunction in the dompaminergic system, and one can assume that they will respond differently to the pharmacological treatment targeting the different systems. This should be further investigated in future research of pharmacological treatments of pathological gamblers.

4.1.2 Predictors of Pathological Gambling

The second aim of this dissertation was to investigate which demographic and personality variables that can predict PG. Four personality variables were associated with PG. Specifically; high scores on Neuroticism, Pathological Impulsivity, Need for Stimulus Intensity, combined with low scores on Openness was associated with PG, even after controlling for other personality and demographic variables. In the bivariate analyses 12 of the 14 predictor variables showed a significant relationship with PG. The only two non-significant predictor variables were gender and age. From prevalence studies, we know that being male and of younger age are related to a greater risk for PG (Petry, 2005). Because PG and some personality variables (e.g., sensation seeking) differ in prevalence between gender and different age groups, it was considered important to

control for the effects of gender and age, and the contrast group was therefore matched on sex and age. As the groups were already matched on these two predictor variables, it was not surprising (rather expected) that the bivariate analysis showed that these variables did not significantly explain the difference in PG status.

The fact that only one of the subscales of the AISS, namely the Need for Stimulus Intensity (and not Need for Novelty), remained significant in the adjusted analyses shows that it is important to differentiate between these two subscales and that the need for intensity of stimulation is more strongly associated with PG than the need for novelty. This finding is in line with former studies showing that PGs are characterized by need for stimulus intensity but not need for novelty (Nower, et al., 2004; Powell, et al., 1999). Hence, talking about gamblers as sensation seekers as such may be inaccurate, and there is a need to further investigate the nature of sensation seeking related to gambling.

The results from Study II further showed that pathological impulsivity was related to PG. All four measures of impulsivity were associated with PG in the bivariate analyses, but only one of the four measures (pathological impulsivity) remained significant in the adjusted analysis. This may indicate that some of the variance in the three predictors (the three subscales of BIS-11) which failed to reach significance was accounted for by other variables, (e.g., pathological impulsivity). As mentioned in the Introduction, one previous study of impulsivity in PGs found that self-reported impulsivity (as measured by the BIS-11) did not predict relapse in PGs, but neurocognitive measures of impulsivity (disinhibition) did. This may suggest that impulsivity as measured by the BIS-11 does not appropriately account for the impulsivity in PGs, but that impulsivity in gamblers is better accounted for by other measures, such as pathological impulsivity (as measured by the EIS-nI) or measures of disinhibition.

In sum, this study showed that these four personality variables are associated with PG, although no conclusions about cause and effect can be drawn as this study had a cross-sectional design. For that reason, we do not know whether high scores on Neuroticism made the individuals vulnerable to developing PG in the first place, or whether high scores on this variable reflect negative consequences related to gambling (financial difficulties, depression, hopelessness, etc.). It has been proposed that anxiety and depression can either be a risk factor for PG or a consequence of gambling (Blaszczynski & Nower, 2002; Zangeneh, Grunfeld, & Koenig, 2008).

4.1.3 Psychopathology and Personality Characteristics

The aim of the third study was to identify psychopathology and personality characteristics among PGs and potential subgroups of gamblers. The results showed that approximately half of the pathological gamblers were characterized by significant symptoms of depression, anxiety and tension. This finding is in line with studies reporting high rates of co-morbidity with affective disorders and anxiety disorders (50% and 41%, respectively; Petry, et al., 2005). Study III also showed that PGs are a heterogeneous group. While approximately half of the sample had personality profiles and levels of psychopathology within the normal range, the other half was characterized by considerable psychopathology. This may have implications for designing effective treatments, which will be discussed further in a subsequent chapter of Clinical Implications of the findings.

The LCA identified three distinct subgroups based on the scores on the eight clinical MMPI-2 scales. The first latent cluster had profiles all within the normal range,

and seemed to resemble the "behaviorally conditioned gamblers" in Blaszczynski and Nower's model who do not have any pre-morbid psychological disturbance. The equivalent in Stewart et al.'s model (2008) is the "low emotion regulation gamblers" who presumably gamble for reasons other than that of emotion regulation. Vachon and Bagby (2009) also identified a cluster with low rates of co-morbid psychopathology and scores near the normative mean, called "the simple PG cluster". One might assume that Latent Cluster 1 resemble these previously suggested subtypes and gamble for reasons other than coping with psychopathology, and seem not to be predisposed by certain personality traits.

Latent Cluster 2 showed the most elevated scores on the scales related to anxiety and depression. One interpretation of this finding can be that gambling behavior is motivated by escaping from negative feelings. In comparisons to previous studies, this group resembles the "coping gamblers" (Stewart, et al., 2008), the "emotionally vulnerable gamblers" (Blaszczynski & Nower, 2002), and the "demoralized PG cluster" (Vachon & Bagby, 2009). A common feature of these subgroups is that their participation in gambling activities is motivated by a desire to modulate affective states or to meet psychological needs. Latent Cluster 2 in the present study was characterized by a 2-7 profile type associated with a rigid thinking and problem-solving style, which may contribute to the wide array of cognitive distortions that typically characterize PGs. These individuals tend to be tense and feel pessimistic and hopeless about the world in general, and often overreact to minor stress. In comparison to previous studies of MMPI profiles among gamblers, this latent cluster somewhat resemble the third cluster of Graham and Lowenfeld (1986) and the first cluster of McCown and Chamberlain (2000) all scoring high on depression, anxiety and negative affects.

The other pathological cluster in Study III, Latent Cluster 3, was characterized by an 8-7 profile type commonly associated with depression, worries, tension, restlessness, confusion and fear of loss of control/panic. Individuals who score in this manner can be described as passive-dependent individuals who withdraw from social interactions. This latent cluster showed more extensive elevations on all scales indicating more severe psychopathology and maladjustment. Although the most pathological cluster in the present study (Latent Cluster 3) and the previously suggested subtypes, the "antisocial impulsivist" (Blaszczynski & Nower, 2002) and the "demoralized PG cluster" (Vachon & Bagby, 2009), portray similar symptoms in terms of greater maladjustment and more severe psychopathology, the two previously suggested subtypes were in addition characterized by high levels of impulsivity. Moreover, in relation to previous findings in the MMPI literature among gamblers the Latent Cluster 3 bears resemblance to the second cluster of Graham and Lowenfeld (1986) and the third cluster of McCown and Chamberlain (2000), who all score high on scales 8-Sc and 7-Pt.

The results from this thesis did not confirm previous findings of a 4-9 profile among gamblers, and the findings that scale 4-Pd is the most elevated scale among pathological gamblers was not confirmed in the present study. This may be due to possible cultural differences between American samples and the Norwegian sample. However, differences in results may also be attributed to other differences among the samples. Significant cohort effects have been observed for some of the MMPI scales. For example, there seems to be an age-effect where older people score lower on scale 4-Pd. However, the mean age of Graham and Lowenfeld's sample was two years older than the mean age of the present sample; hence age has probably not contributed to these differences. McCown and Chamberlain (2000) do not describe their sample in terms of

age, gender or other demographic characteristics, hence comparison with this sample is difficult. Furthermore, elevated scores on this scale are often observed among minority groups, reflecting alienation, but as Graham and Lowenfeld's sample included only Caucasians, ethnicity has probably neither contributed to the higher scores on scale 4-Pd. Graham and Lowenfeld included only male gamblers in their sample, whereas the present sample also comprised women (21%), hence differences in distribution of gender may have had an impact as male gender is associated with higher scores on scale 4-Pd (Havik, 2003).

All the former studies of MMPI-profiles among gamblers have also utilized treatment seeking samples of gamblers (Ciarrocchi, et al., 1991; Graham & Lowenfeld, 1986; McCown & Chamberlain, 2000; Moravec & Munley, 1983), but there are differences in terms of inpatient vs. outpatient samples. The samples of Graham and Lowenfeld (1986) and Ciarrocchi et al. (1991) were all inpatients, whereas in Moravec and Munley's study (1983) a majority of the sample (86%) were outpatients. McCown and Chamberlain (2000) do not state whether the gamblers in their sample were inpatients or outpatients. Outpatients may be of higher functioning, and therefore explain the differences in results.

Furthermore, differences between the samples in terms of co-morbid substance abuse may well have contributed to this discrepancy of findings, because a 4-9 profile is typically found among substance abusers. In the present study concurrent alcohol or drug dependency was an exclusion criterion, however only one of the potential eligible participants was excluded on this basis. No data on alcohol or drug abuse were reported by Graham and Lowenfeld (1986), however the high score on the Mac-Andrew Alcoholism (MAC) scale (M = 27.7) in this sample may indicate substance abuse

problems. It is also possible that the sample in the present study was not sufficient to detect this subtype. Further, it is possible that the different results can be attributed to differences in heterogeneity of PGs over time, as the gambling market today is quite different from that of the 1980s and 1990s when the former studies were conducted. Another possible explanation for the discrepancy between the present and the former studies in this field, is that the present study utilized the revised version, MMPI-2, whereas the former studies (with the exception of McCown & Chamberlain, 2000) have used the original MMPI. As many of the scales have been updated and new norms collected, this may well have contributed to the difference in findings.

Overall, the present findings support previous findings of different sub-types of PG. Previous findings of a "normal" group, with little psychopathology and scores within the normal range, and a group characterized mainly by depression and anxiety, which has been suggested to engage in gambling in order to cope with underlying psychopathology, were replicated in the present study. The results of this study further underscore the importance of differentiating among different types of gamblers. Most of the research in this field has not investigated differences between different types of gamblers. The sub-typing of gamblers could have important implications for research and clinical practice.

4.2 Methodological Considerations

Strengths and limitations of the three studies are already presented in the discussion in the three papers, but the following section will elaborate on some of the issues raised.

4.2.1 Sample Size

The relatively small sample sizes in these three studies represent a potential threat to the statistical conclusion validity. Due to difficulties in the recruitment process and large drop-out rates, the final sample in Study I consisted of only 30 PGs. According to the power analysis conducted, 12 participants in each group should be efficient in order to detect a difference at T1 (8-weeks post-treatment). However, only 11 participants in the CBT group and 13 participants in the escitalopram + CBT group completed the 8-weeks post-treatment. Still, the relatively small between group effect sizes (which are independent of sample size) at 8-weeks post-treatment support the conclusion that both treatments were equally effective in the short term. The issue of drop-outs at different assessment points lowers the potential power, though implementing intent-to-treat analyses throughout the analyses still provide an idea of the effects of the treatments (Shaddish, et al., 2002).

Even though a power analysis was conducted before initiating the study, one could argue that interim power analysis should have been conducted in order to assess whether the effects obtained in the present study were comparable to the effects obtained in the previous meta-analyses on which the power analysis was based on. It has been found that studies using passive control conditions, such as waiting-list, may be associated with greater improvement compared to studies using active control conditions, such as taking a placebo pill (Nordhus & Pallesen, 2003). The discrepancy in ES between the meta-analyses of psychological (Pallesen, et al., 2005) and pharmacological (Pallesen, et al., 2007) interventions have been proposed to be accounted for by the fact that the former used passive control conditions, whereas active control conditions were primarily used the latter (Pallesen, et al., 2007). Hence, the ES that the present power analysis was based

on may be obscured, and one may need a larger sample size in order to obtain significant results if the difference in effect is smaller than initially assumed.

Furthermore, the power analysis was primarily valid at 8-weeks post-treatment as this was the only assessment point where the single treatments were compared. In order to detect whether adding a component (escitalopram) to an already established treatment (CBT) will be more effective than a single treatment (CBT), one probably would need a larger sample size. Power analyses were not conducted for the following assessments when the combined treatment (escitalopram + CBT) was compared to single CBT. Consequently, we cannot draw any firm conclusion about the relative effectiveness of the different treatment conditions (CBT vs. escitalopram vs. escitalopram + CBT) based on this small-scaled pilot study. However, the small between-group ES throughout the assessment points support the preliminary conclusion that the treatments did not differ in terms of effectiveness. Hence it seems reasonable to conclude that the present pilot study indicates that both the single treatments and the combination of the two treatments were effective in treating PGs. The relatively small sample size of this study did not allow for a further examination of whether treatment effects were moderated by gender, the presence/absence of co-morbidity or gaming preference.

The sample in Study II comprised 90 PGs and a contrast group of 66 non-pathological gamblers. Regarding the sample size in logistic regression analyses the number of cases have to be appropriately related to the number of predictors. In the presence of categorical predictors with limited cases in each category the ratio cases/predictors should be higher than when predictors based on continuous variables are employed. In the present study, all predictor variables, except gender and education level comprised continuous variables. Green (1991) has suggested some rules of thumb when

deciding how many cases are necessary. The rule of thumb is: $N \geq 50 + 8m$ (where *m* is the number of independent variables). According to this rule, 162 cases were needed when the number of predictors in the present study was 14 (50 + (8x14) = 162 cases). The present study only had 156 cases, which is just below the suggested number of cases. Ideally the size of the sample should have been larger; however the sample size of the present study was close to achieving the suggested sample size. The size of this PG sample did not allow for differentiating among various types of gamblers (i.e., gamblers who prefer different types of games) and investigating whether the differences in gambling preference are related to differences in personality characteristics. As previous studies and the third study in this thesis indicated that gamblers represent a heterogeneous population, it may be wise to replicate this study with a larger sample and investigate whether different groups of gamblers and the contrast group can be identified empirically using LCA to determine different latent classes in the sample.

In Study III, the total sample consisted of 66 PGs, and the LCA performed was based on 8 parameters. LCA is usually performed with larger sample sizes, and ideally the ratio between the number of indicators (MMPI-2 scales) and the number of subjects should be greater than 10. However, when indicators can be treated as interval measures and not categorical indicators, the LCA is less data consuming. Thus, as indicators in the present study were treated as interval measures the small size of the sample did not hamper the estimation of the number of latent class clusters. Still, it would be wise to replicate also this study with a larger sample.

4.2.2 Representativeness

There are several relevant issues to evaluate when discussing representativeness. Firstly, one need to consider who the sample should be representative of, and to what population one wants to generalize the results. When investigating the effectiveness of treatments for PG do we want these results to generalize to the total population of gamblers, or only to treatment-seeking gamblers? The participants in all three studies were treatment seeking gamblers who are probably not representative of the total population of gamblers, and generalization to the total population of gamblers should therefore be made with caution. However, generalization of results of treatment effectiveness is probably not relevant for those who do not seek treatment anyway. Only a small portion (7-12%) of gamblers seek treatment (Slutske, 2006), and there is reason to believe that treatment seeking gamblers differ from those who do not seek treatment in terms of the severity of their gambling problems, levels of psychopathology, personality traits, coping resources and the ability to benefit from treatment. Treatment seeking gamblers may also be characterized by greater levels of psychopathology, which contributes to a greater level of distress and discomfort, motivating them to seek help.

Because treatment is usually voluntary, samples in treatment studies are generally self-selected convenience samples, as were the case in the present study. Obtaining a representative sample of gamblers would imply forcing treatment on gamblers who do not seek treatment. This would not be an ethical option. As the sample in the present study was a convenience sample we further do not know whether the gamblers were representative of the treatment seeking population of gamblers. However, at the time of initiating the recruitment process, there was no other official treatment offer for gamblers in the region (Hordaland). After 1/3 of the gamblers were recruited, another treatment

program for PG was offered in Hordaland, and in the following period there was a joint recruitment process where patients were randomly assigned between the two treatment programs. All general practitioners in the region and other potential referral instances were informed of these treatment projects. It is therefore likely that the sample was fairly representative, at least for treatment seeking gamblers in this region of Norway, and the results can hence probably be generalized to the treatment-seeking population of gamblers.

Participants in the contrast group in Study II also represented a self-selected convenient sample as they were recruited through newspaper advertisements. Therefore, they are probably not representative of non-pathological gamblers in general, and it is possible that those volunteering to participate in research differ in personality traits from the general population. One may assume that individuals volunteering to participate in research projects are healthier and of higher functioning than the population mean. When looking at the education level of the contrast group, nearly 73% had at least one year of higher education (University or College). Regarding education level they are therefore probably not representative of the general adult population of Norway. The contrast group in the present study comprised 1/3 females and was not representative of the general adult population in terms of gender which may have affected the generalizability of the findings. Using a contrast group representative of the general adult population would have allowed for also investigating the associations between gender and age with PG status. However, the aim of the present study was not to investigate the associations between gender and age with PG, but rather the associations between different personality characteristics and PG. Because we know from epidemiological studies that PG is unevenly distributed over gender and age (Petry, 2005) and personality traits have been

shown to differ across gender and age (González-Ibáñez, Mora, Gutiérrez-Maldonado, Ariza, & Lourido-Ferreria, 2005) it was considered important to try to control for these variables. The contrast group was consequently matched on sex and age (+/- 5 years) in order to try to limit the influence of these characteristics. Matching the control group on variables such as gender and age are common in studies investigating personality variables among PGs (e.g., Blanco, et al., 1996; Getty, Watson, & Frisch, 2000; Kaare, et al., 2009; Petry, 2001). To further control for the possible effects of gender and age, these variables were entered as predictors into the logistic regression analyses to ascertain that they did not significantly predict PG status.

The sample in Study III also consisted of a self-selected treatment seeking population of gamblers and, as already stated, treatment seeking gamblers are most likely not representative for the total population of gamblers. Although the aim of the present study was not to generalize to the total population of gamblers, it should be mentioned that as there is reason to believe that treatment seeking populations experience more distress and negative consequences of their gambling behavior compared to non-treatment seeking gamblers, there will probably be more elevated profiles among treatment seekers compared to non-treatment seekers.

4.2.3 Validity of Self-Report

Self-report questionnaires are prone to error and have been criticized for the possibility of distortion of the subjects' responses. Phenomena such as *social desirability* or the *need for social approval* may alter the subjects' responses. For example, patients may underreport symptoms of gambling urges after treatment in order to please the

therapist. Moreover, a non-intended biased memory may also result in incorrect self-reported gambling behavior because gamblers have a tendency to remember more wins compared to losses which affects the self report of net losses (Rachlin, 1990). Clinicians have also reported that clients do not report the full extent of their gambling-related problems until later in treatment, and Stinchfield et al. (2007) suggest that some gamblers may be motivated to withhold information that may be self-incriminating. It is also possible that some gamblers may be ”faking bad”, i.e. overreport their problems, in order to get access to treatment.

Although the validity of self-report could be questioned, other more objective verification of gambling is difficult to obtain because of the absence of both sensitive and specific physiological markers. Nevertheless, collateral reports have been used to validate self-reported gambling behavior. Hodgins and Makarchuk (2003) examined the reliability and validity of gamblers self-reports and found that the agreement between the gambler's and collaterals' reports was generally good, with no clear pattern of over- or underreporting by gamblers. In the treatment study 60% of the gamblers did not have a partner, hence collateral reports would have been difficult to obtain.

Chung et al. (2009) demonstrated changes in fMRI results following treatment with fluvoxamine in a case report of a pathological gambler, suggesting that fMRI may represent a more objective tool in evaluating treatment response in addition to subjective self report. Still, more research is needed to establish the usefulness of fMRI in evaluating treatment efficacy.

Attentional biases have also been suggested in PGs, and a recent study using a pictorial Stroop performance task revealed that PGs showed significantly longer reaction

times to win-related pictures compared to neutral pictures, and that PGs also had longer reaction times to win-related stimuli compared to a control group (Molde et al., 2010). Attentional biases are thought to promote gambling urges, and stimuli related to the addictive behavior may act as automatic and unwanted distractions and hence interfere with the individual's ability to control their behavior (Field & Cox, 2008). Previous studies have reported attenuation of attentional biases following successful treatment (Watts, McKenna, Sharrock, & Trezise, 1986), and measures of attentional biases may hence have the potential to serve as more objective measures of treatment effects.

4.2.4 Attrition

Attrition represents a potential problem in all treatment studies, and the attrition rate of gamblers in treatment has been reported being as high as 50% (Ladouceur, Gosselin, et al., 2001). High attrition rates may serve as a threat to the internal validity. When attrition rates are high and intent-to-treat analyses are not carried out, the effect of treatment may be inflated by the fact that those who did not respond to the treatment dropped out. For that reason, intent-to-treat analyses were performed in the present treatment study. Intent-to-treat analyses give a conservative estimate of the effectiveness of the treatment as a large proportion of pre-treatment scores are usually carried forward in the analyses, making it more difficult to obtain significant results. Considering the fact that 20% of the pre-treatment scores were carried forward and significant changes from pre-treatment to the various post-treatment assessments were still observed, strengthens the conclusion that the treatments had an effect.

4.2.5 Contrast/Control Groups

In the treatment study no time by group effects were found which indicates that the treatment modalities were equally effective. However, as no control groups were included (e.g., waiting list or placebo control), the improvements for both groups could be attributed to factors such as history, maturation, retesting, regression towards the mean or to nonspecific treatment influences which serve as a threat to the statistical conclusion validity. Since no placebo control group was included, the improvement of participants receiving medication could be attributed to placebo effects. Previous findings regarding the efficacy of SSRIs have been mixed (Sáiz-Ruiz et al., 2005). Although two previous studies have shown pharmacological treatments with SSRIs to be superior to placebos (Hollander et al., 2000; Kim, Grant, Adson, Shin, & Zaninelli, 2002), one study showed that the placebo and the active drug were equally effective in the short term (the first eight weeks; Hollander, DeCaria, et al., 2000). Further, other placebo-controlled studies have failed to demonstrate statistical superiority of the active drug (Blanco, et al., 2002; Grant et al., 2003; Sáiz-Ruiz, et al., 2005) and high placebo response rates (59%) have been demonstrated (Hollander, DeCaria, et al., 2000).

However, the use of control groups is also an ethical question. As stated in the Declaration of Helsinki: “In any medical study, every patient – including those of a control group, if any – should be assured of the best proven diagnostic and therapeutic method” (World Medical Organization, 1996, p. 3). For that reason, it would be unethical to use placebo as a control when a proven effective therapeutic method exists. The effectiveness of CBT for PG has been demonstrated in numerous studies (Gooding & Tarrier, 2009), but much of the gambling treatment research is characterized by methodological flaws and design weaknesses which have prevented the treatments for PG

from obtaining status as empirically validated (Toneatto & Ladouceur, 2003). At present, no treatment for PG has yet obtained status as empirically validated treatment but CBT has shown promising results and are close to meeting this status (A. Blaszczynski, personal communication, January 20, 2010). As pharmacological treatment is not as close to obtaining status as empirically validated, a pharmacological placebo group could have been included in the present study. This issue will be further elaborated in a subsequent chapter when discussing the design of the present studies.

The problems with representativeness of the contrast group in Study II are already discussed. Because gamblers have shown differences in personality and psychopathology variables as well as in treatment response depending on age (González-Ibáñez, et al., 2005), it was considered important to try to limit the influence of age when investigating personality variables as predictors of PG. Matching according to categorical variables (e.g., gender) is straightforward, but when matching is conducted according to continuous variables (e.g., age), a matching range is usually defined (Szklo & Nieto, 2007). For the present study a range of plus or minus 5 years were selected, as suggested by Szklo and Nieto (2007). One may argue that the matching procedures for selecting the contrast group (+/- 5 years) were not stringently enough. However, as long as the groups were not statistically different in terms of age, an age span of +/- 5 years was considered acceptable.

The third study of MMPI-2 profiles among PGs did not include any patient contrast group, and one cannot rule out the possibility that these characteristics are not specifically characteristics of PGs, but rather of all those seeking psychotherapy in general. A former study also found that pathological gamblers and alcoholics showed similar MMPI profiles (Ciarrocchi, et al., 1991), hence a suggestion for future research

would be to compare different types of addictive behaviors and investigate whether they display similar or different MMPI-2 profiles.

4.2.6 Treatment Adherence

In the treatment study, there was only one therapist conducting both the CBT and delivering the pharmacological treatment. Thus, there is the possibility that a therapist effect could have affected the treatment outcomes and that outcomes can be an effect of non-specific treatment effects, such as therapist alliance (Kazdin, 2003). However, it may also be regarded as a strength rather than a limitation of the present study that both group and all treatment conditions had the same therapist, so that potential differences between the groups could not have been attributed to the use of different therapists. However, because it is likely that treatment effects vary as a function of the patients, the therapist, and the context in which the patient functions (e.g., extent of family support), it is important to examine factors that may moderate the treatment effect (Kazdin, 2003). One such factor could be the therapist. Therefore, future studies should investigate the disseminability of the treatment approach and try to replicate the results using different therapists to test whether the treatment effects can be generalized to other therapists.

Both a patient and a therapist manual were utilized in the present treatment study to maximize the likelihood that all patients would receive the same treatment. However, no adherence or competence ratings were conducted and further larger scale studies including several therapists should be conducted to better assess the effectiveness of the present CBT program.

4.2.7 Maximum Dosage of Escitalopram

The maximum dosage of escitalopram in the present treatment study was 20 mg/day because the Norwegian Medicine Agency would not allow for a larger dosage since the tolerance of a larger dosages were not established at the time of initiating the treatment study. However, other pharmacological studies of SSRIs in the treatment of PG have utilized a larger dosage than what is normally prescribed for depression, so it is possible that larger dosages would have yielded greater effects of escitalopram.

4.2.8 Post-treatment Effect of Escitalopram

Participants in the escitalopram + CBT group were offered to continue the treatment with escitalopram throughout the 6-months follow-up period in order to investigate whether escitalopram in combination with CBT would yield a better effect than CBT on a long-term basis. Consequently, there is no measure of whether the pharmacological treatment effect persisted after these participants ended treatment with escitalopram as there were no assessments conducted after the discontinuation with escitalopram. Even so, based on clinical experience it has been suggested that 6-12 months of treatment with SSRIs should be sufficient in order to re-establish the balance of serotonin in the brain (J. E. Grant, personal communication, January 6, 2010). Hence, at the 6-months follow-up it would be expected that the effects of escitalopram should have reached its therapeutic potential.

4.2.9 Overall Designs of the Studies

The design of the first study comprised one between-subject factor (Group; CBT vs. escitalopram + CBT) and one repeated within-subject factor (Time) with 5 levels: pre-treatment (T1), 8-weeks post-treatment (T2), 16-weeks post-treatment (T3), 3-months follow-up (T4), and 6-months follow-up (T5). The randomized controlled design represents a strength of the first study. Randomization was conducted in order to decrease the chances that the two groups were different at pre-treatment which could be a threat to the internal validity. The randomization does of course not guarantee that the groups are not different. However, the statistical analyses (ANOVA and chi square tests) showed that there were no statistical significant differences between the two groups on any of the demographical or clinical characteristics at pre-treatment. Because pharmacological treatment with escitalopram has not yet received status as effective in treating PGs, it was (in line with the Declaration of Helsinki) considered unethical not to deliver CBT, which is the best known treatment, to this group. Hence there was only one assessment of the effects of single treatment with escitalopram (T2), thus it was not possible to investigate the long-term effects. This is a weakness of the present design, because any initial effect of the SSRI may be explained by a placebo effect.

The design of the treatment study would possibly have been improved by using a 4-armed design, investigating the differences between CBT, attention placebo control group, escitalopram, and a medication placebo group. However, this design would need a much larger sample in order to have sufficient power to detect possible significant differences between the four conditions. Obtaining such a large sample was not considered realistic for the present study, and the problems with recruiting a sufficient sample size for a 2-armed design confirmed this assumption. Since CBT has shown

efficacy in several well-controlled studies and is close to obtaining status as empirically documented treatment, pharmacological treatment with escitalopram was compared with CBT which is considered the "gold standard" (A. Blaszczynski, personal communication, January 20, 2010). Still, it is acknowledged that the lack of a placebo/control group represents a limitation of the design. Another limitation of the present design is that there was no measure after the escitalopram + CBT group discontinued treatment with escitalopram. Hence we do not know whether the treatment effect lasted after discontinuing medication.

In the second study the aim was to investigate which personality variables can predict PG status. The issues related to the contrast group and the representativeness of the sample have already been discussed elsewhere in this thesis. As this study had a cross-sectional design, we do not know whether the PGs' scores on the different personality variables contributed to (made them vulnerable to) developing PG, or if they have changed as a result of the consequences related to the problem. In a prospective longitudinal design one could have measured whether personality variables assessed at one time point would predict later PG status. This type of design would allow for making hypothesis about cause and effect. However, it was beyond the scope of this four year PhD project to carry out such a longitudinal design.

The third study also had a descriptive design and included only a treatment seeking sample. As some personality traits (e.g., impulsiveness) may be related to addictions in general, this study could have included another patient contrast group, such as another addicted sample. However, one former study compared the MMPI profiles of PGs, alcoholics and dually addicted gamblers and found that the only differences between the groups was that the PGs had higher education level and higher socioeconomic status

compared to the alcoholics (Ciarrocchi, et al., 1991). Few studies have investigated the extent to which treatment seeking gamblers resemble the total population of gamblers in terms of personality characteristics and levels of psychopathology. Including both a treatment seeking and a non-treatment seeking sample of gamblers would have allowed for making hypothesis of how treatment seekers differ from the non-treatment seeking population. However, the focus of the present study was to describe the treatment seeking population in order to generate hypothesis related to adapting treatment interventions.

4.2.10 Choices of Instruments

A variety of standardized outcome measures (with already established good psychometric properties) were included in the treatment study, including a symptom measure (G-SAS) and a measure of urges (PGVAC) to assess perceived control, a behavioral measure of problem gambling (PGBS) assessing frequency and money and time spent gambling, and a secondary measure of depression (BDI-II) to measure negative consequences of the disorder. Unfortunately no gold standard exists for measuring changes in gambling behavior after treatment interventions, and researchers (and clinicians) are confronted with the challenge of selecting from a wide array of instruments (Stinchfield, et al., 2007). The spectrum of measurements chosen for the present study is however in line with the recommendations that Nower and Blaszczynski (2008) put forward. They acknowledged the lack of conceptual clarity in the gambling field, making it difficult to assess actual efficacy of interventions and compare different treatment approaches, and suggested that the nature and extent of improvement should be measured along a spectrum including measurements of decrease in frequency of and the

time spent gambling, abstinence or controlled gambling that meets financial obligations, absence of symptoms of impaired control and cross-addicted behaviors, and absence of negative consequences and improved quality of life over time (Nower & Blaszczynski, 2008).

Pallesen et al. (2005) pointed to a measurement problem in the gambling field as many studies included in their meta-analysis used outcome variables comprising single items, and in some instances the researchers had made up their own items and scales. A strength of the present treatment study was that the outcome assessment was based on a variety of formerly validated scales, comprising several items (the PGBS was the only instrument comprising only three single items). The G-SAS measures urges, thoughts, and behavior related to gambling during the last week. It is therefore sensitive to measure recent changes in symptoms. Most outcome measures have a longer time frame, such as the SOGS-R, making it difficult to assess recent changes. The PGVAC is a visual analog scale measuring current urge or craving for gambling. This measure was considered a good supplement to the more general symptom measure G-SAS. Behavioral measures also constitute important additional information to the aforementioned measures. In opposition to the PGVAC which captures internal states (urges), the PGBS represents a behavioral measure of frequency of gambling and amount of time spent gambling and amount wagered during the last week. These three measures (the G-SAS , the PGVAC and the PGBS) were considered to complement each other, and are well established measures in the gambling literature (Stinchfield, et al., 2007). As it is well established that gambling often co-occur with other disorders, e.g. depression, the BDI-II which measure symptoms of depression was included as a secondary outcome measure. Furthermore because SSRIs are generally prescribed for depressive symptoms it was considered important to include a measure of depression.

A potential measurement problem associated with the PGBS is that it is based on a one week timeframe, and not on a one month timeframe as recommended by Walker et al. (2006). Many gamblers are known to gamble periodically and for example spend more money on gambling after receiving salaries. In Norway, salaries are usually paid once a month, and as a consequence the behavioral measure may be compromised in terms of reliability. Furthermore, 9 of the 30 clients (30%) had not spent any money on gambling during the last week at pre-treatment. This does not necessarily indicate that they had controlled their gambling behavior, but may rather be a result of not having any money left to gamble with. Hence, for these individuals it was not possible to improve on this measure.

A weakness of the present study is that it did not include a measure of the mechanisms of change, e.g., cognitive distortions. However, initially the Informational Biases Scale (Jefferson & Nicki, 2003) was included as an outcome measure. The instrument has been shown to have good psychometric properties and suggested to be useful in clinical settings with EGM gamblers (Jefferson & Nicki, 2003). At the time of initiating the study, almost all gamblers seeking treatment in Norway had problems with EGMs, and this instrument was therefore considered a good instrument to measure changes in cognitive distortions following treatment. However, changes in the gambling marked in Norway (such as the banning of EGMs, increasing popularity of poker and opportunities to gamble on the internet) have resulted in a more heterogeneous treatment-seeking population of gamblers over the last years. Consequently, only 33% of the gamblers in the present study gambled primarily on EGMs, hence the IBS were not appropriate for the rest of the sample and this instrument was consequently not included in the analyses. In retrospect, I realize that choosing a more general measure of cognitive distortions, e.g., the Gamblers Belief Questionnaire (Steenbergh, et al., 2002), which is

not restricted to EGMs only would have been a better option and would have allowed for investigating whether the CBT group improved relatively to the SSRI group on this measure.

Collateral reports of gambling behavior could also have been included in the present study in order to have more objective measures of gambling behavior. However, as already mentioned, former studies have found generally high correlations between gamblers self report and collateral reports (Hodgins & Makarchuk, 2003). Furthermore, because many gamblers do not have a partner (60% in the present study) this type of measure is problematic. Another option would be to include an independent expert (clinician) evaluation to supplement the self-report. However, this would have demanded much more resources, and was not possible within the framework for the present study. Measures such as fMRI and Stroop task performance to measure attentional biases have also been suggested as possible indicators of treatment effects which do not rely on self-report, however, the usefulness of these measures in assessing treatment effects for PGs still remain to be validated in large-scaled studies.

In Study II a measure of the FFM of personality was included in addition to specific measures of impulsivity and sensation seeking. The FFM is a comprehensive model of personality and has received considerable empirical support (Piedmont, 1998). Bagby et al. (2007) noted that because previous studies have utilized a variety of dimensional personality models resulting in inconsistencies among outcomes there is a need for a single overarching model when investigating traits related to PG. Few studies have used measured based on the FFM in assessment of PGs, and Bagby et al. (2007) suggested that the FFM is an ideal platform to assess traits related to PG. The short version NEO-FFI was chosen in favor of the longer version NEO-PI-R in the present study, because these data was collected as part of the screening procedure for the

treatment study, and the clients also had to fill out a large battery of other instruments. Using the long version could hence have resulted in test fatigue, and possibly resulted in more missing data. An advantage of using the NEO-PI-R is that it also includes facet traits (six subscales for each of the five traits). However, as this instrument consists of 240 items and the short version comprises only 60 items, the latter was considered more useful in this setting.

The narrow impulsiveness subscale of the EIS is regarded as a good measure of pathological impulsiveness and has increasingly become the gold standard for measuring impulsivity (Blaszczynski, et al., 1997) and shows good inter-correlations with other measures of impulsivity (Dickman, 1990). However, as the EIS-nI is a unidimentional measure of impulsivity with dichotomous answer categories, the BIS-11 with its three subscales (motor impulsiveness, nonplanning impulsiveness and attentional impulsiveness) rated on a 4-point likert scale was considered a good supplementary measure of impulsiveness.

There has been a controversy in the field regarding the measure of sensation seeking (as already described in the Introduction of this thesis). Based on the critiques of the widely used Zuckerman's SSS-V (Zuckerman, et al., 1978) regarding the validity of SSS-V in the area of research concerning pathological gambling (Arnett, 1994; Hammelstein, 2004), the AISS which differentiates between two different aspects of the concept of sensation seeking (need for novelty and need for stimulus intensity) was considered a better alternative. The AISS has been shown to have good psychometric properties and has been found to be more strongly related to risk behavior compared to the SSS-V (Arnett, 1994). Because the AISS conceptualizes sensation seeking as a need, it hence offers a possibility of measuring sensation seeking as independent of impulsiveness which is related to the (lack of) control over the behavior (Hammelstein,

2004). The SSS-V has been shown to be moderately to highly correlated with impulsiveness (Zuckerman, 1994), whereas the AISS has not been correlated with measures of impulsivity (Hammelstein, 2004)

Regarding the third study of MMPI-2 profiles among pathological gamblers, much of the previous research in the field has utilized the former version MMPI. However, this version has been criticized for not being up-to-date and for having a number of nonworking items (Butcher, 2005). Furthermore, the norms were considered overly narrow, partly because they consisted on a small, somewhat provincial sample from Minnesota. The instrument underwent a major revision during the 1980s (Butcher, 2005), hence there is a need for updating the knowledge of MMPI profiles among gamblers. Furthermore, the gambling market has also changed considerably since the 1980s and 1990s when the former studies have been conducted which may also have affected the heterogeneity of the gambling population. As the MMPI-2 is a comprehensive tool for assessing both psychopathology and personality characteristics which has gained considerable psychometric support throughout the years (Butcher, 2005) and the research in this field was somewhat out-of-date, a study of MMPI-2 profiles among gamblers was considered a relevant contribution to the field.

An alternative to using the MMPI-2 in Study III could have been to use more specific symptom measures, such as the Symptom Check List-90 Revised (SCL-90-R; Derogatis, 1994) to assess pathology among the treatment-seeking gamblers. However, SCL-90-R profiles among gamblers have not been as systematic and extensively studied as MMPI profiles, and would hence not have allowed for comparisons with previous findings in the field. Although moderate correlations between corresponding standard MMPI scales and SCL-90-R scales have been found (r ranging from .42 to .64; Degoratis, Rickels, & Rock, 1976), the SCL-90-R is less comprehensive in assessing general

psychological distress. The MMPI have accordingly been suggested a more useful instrument when detailed information about the patient's psychological condition is needed (Kinney, Gatchel, & Mayer, 1991).

4.2.11 Choices of Methods/Analyses

Intent-to-treat analysis is often recommended because it gives a more conservative estimate of the treatment effects, and it also takes the issue of attrition into account. Because a significant proportion of the individuals dropped out in the present study, intent-to-treat analysis was considered the preferred analyses. Furthermore, because this study had 5 assessment points (pre-treatment, 8-weeks post-treatment, 16-weeks post-treatment, 3-months follow-up, and 6-months follow up) and the measurement of the dependent variables were repeated, repeated measures ANOVA was chosen for the present analyses. Using standard ANOVA in this case is not appropriate because it fails to model the correlation between the repeated measures; hence the assumption of independence is violated (Pallant, 2005). Furthermore, when there is a great deal of variation between sample members the error variance estimates from standard ANOVAs are large. In repeated measures ANOVA, the error variance is reduced because the repeated measures for each sample member provides a way of accounting for this variance. Univariate ANOVA make the assumption of sphericity (that the variance of the population difference scores for any two conditions are the same as the variance of the population difference scores for any other two conditions), an assumption that is often violated (Pallant, 2005). Multivariate statistics do not require sphericity (Pallant, 2005), and was hence used in the present analyses.

When comparing the two single treatments (CBT vs. SSRI) it is not only important whether they score differently on the outcome measures at T1, but the change from T0 should also be taken into account. Because repeated measures ANOVA models the correlations between the repeated measures, inherent in the analysis when comparing the two groups by the interaction term at T1 (8-weeks post-treatment), the pre-treatment scores (scores at T0) are controlled for. When testing the second hypothesis (whether SSRI + CBT yield better outcome than CBT) one could argue that the scores of the CBT group at T1 should be compared with the scores of the SSRI + CBT group at T2 (when both groups had just finished the CBT). However, then assumption of equal time interval between the time points of measurement would have been violated, and time could be a possible confounder. In the present analyses, the time interval between each point of measurement is equal for both groups and the second hypothesis is therefore tested by comparing both groups at T2. Changing behavior is a process that may take time, hence it was considered important to use equal time intervals to avoid time as a confounding factor.

Another alternative to the analyses conducted in the present study would be to conduct multi-level modeling analyses. Within longitudinal data, observations within the same individual are more alike than two random observations from two different individuals because the measurements within the same individual are typically interdependent or correlated in a systematic way. Within multi-level models this pattern of intra-individual correlation can be accounted for. Individuals within a treatment group may respond differently to the same treatment, and within traditional analytic approaches these individual differences are attributed to sampling or measurement error instead of meaningful individual variability in change. Contrary to traditional ANOVA-based forms of analysis (e.g., repeated measures ANOVA) which assumes that everyone changes in

the same way (has the same treatment effect), multi-level models allow for simultaneously modeling of both intra-individual change and individual differences in intra-individual change (Laurenceau, Hayes, & Feldman, 2007). With a multi-level modeling approach it is possible to explain the impact of inter-individual variability at the different time points using covariates which may predict individual variation in both the rate and shape of change (H. Goldstein, 2003). An advantage of using multi-level modeling is that the specific tests for each dependent variable are more powerful as the standard errors become smaller. This method also allows subjects to have missing time points (B. T. West, Welch, & Galecki, 2007). A multi-level modeling analysis of the data was conducted, using the SPSS (version 15.0). However, the results showed that there was a significant effect of time, but no significant group effect or time by group interaction effect. Because both approaches (repeated measures ANOVA and multi-level modeling) yielded significant time effects but not significant group effects or interaction effects, repeated measures ANOVA was chosen when presenting results from the present study as this type of analysis is considered the traditional analytic tool for randomized controlled treatment designs (Laurenceau, et al., 2007). Furthermore, as more people are familiar with traditional repeated measures ANOVA it was presumed that this form of analysis would better communicate with the clinical reader. Furthermore, the inclusion of measures of ES was considered an important complement to the inferential statistics as this measure is independent of sample size and measures the strength of the relationship between two variables. Reporting of ES is recommended by a prominent task force in the field of psychology (Wilkinson & the Task Force on Statistical Inference, 1999).

In the second study hierarchical regression analyses were considered, with the total scores on the SOGS as the continuous dependent variable. However, as most of the PGs scored in the upper end of the SOGS-R and the contrast group in the far low end of

the scale, the assumption of normal distribution was seriously violated and logistic regression analyses was considered to be a better alternative. Using only bivariate logistic regression analyses would have been problematic because some of the scores on e.g. the subscales of the BIS-11 or on the AISS may be interrelated and it would therefore yield an inaccurate estimate. Using adjusted (multivariate) logistic regression analyses allows for controlling for the common variance among the different predictor variables. As education level was no longer significant in the adjusted analyses this indicated that some of the variance in this variable was already accounted for by other variables. Still, the collinearity diagnostics indicated that multicollinearity (high correlations between the predictors) was not a problem in the present data set.

In order to investigate whether there existed subgroups in the sample in the third study we decided to use LCA which has several advantages over the more traditional cluster analyses, such as *k*-mean analysis. Traditional clustering techniques have some limitations that LCA does not have. For example, traditional cluster techniques often rely on the assumption that the responses are measured on an interval level and that they are normally distributed; assumptions which are seldom met. Further, in *k*-mean cluster analysis you would have to decide on the number of clusters in advance where as in LCA you let the structure of your data determine the number of clusters. Also, *k*-mean analysis does not provide fit statistics like the LCA does. An advantage of LCA is that it is based on a statistical model that can be empirically tested, and the determination of number of classes is therefore less arbitrary than when using traditional cluster methods. LCA has been suggested to be more optimal to detect the number of clusters compared to *k*-mean (e.g, Magidson & Vermunt, 2002a, 2002b).

Sub-grouping of the gamblers in the third study was initially investigated using hierarchical cluster analysis (between-groups linkage, with squared Euclidian distance measure). The procedure was computed for 2-10 clusters, and discriminant analyses revealed that a two-cluster solution resulted in 96% correct classification of the cases whereas a four-cluster solution resulted in a 100% correct classification of the cases. However, in the latter solution one cluster included only one case and the two-cluster solution was hence considered to be more clinically meaningful and chosen on the basis of parsimony. The first cluster, comprising 75% of the sample had no elevated mean scales, whereas the second cluster, comprising 25% of the sample, had elevations on 8 of the 10 standard scales and was characterized by an 8-7 profile type. Considering the fact that more than 30% of the sample scored above a T score of 65 on six of the ten scales, characterizing 75% of the sample with mean scores < 65 probably give a too simplistic description of the gamblers. The present latent cluster analysis approach, which also had a higher rate of correct classification (98.4%), probably gives a more adequate description of the sample.

4.3 Clinical Implications

Study I showed promising results for the effectiveness of escitalopram, although more research is needed to establish the relative effectiveness of CBT and treatment with escitalopram. Pharmacological treatments have the advantage of avoiding long waiting lists and not being dependent on a skilled therapist to deliver the treatment, and it can hence be more accessible in smaller places and more remote areas. As anxiety is often co-morbid with gambling problems (Petry, et al., 2005), high levels of anxiety may keep these individuals from seeking treatment for their gambling problems, especially group

treatment which is the most commonly offered treatment format for gambling problems in Norway (Norwegian National Helpline, 2010). The fact that the pharmacological treatment is less time consuming for the patient and require less effort may contribute to lowering the thresholds for treatment seeking. However, a possible disadvantage of pharmacological treatment is that the patient will attribute the change to the medication and not credit him/herself for the change process. This may make them more susceptible for relapse when they quit medication. Hence, as CBT aims at changing cognitive distortions one may speculate that this treatment would have more long-lasting effects Future studies should therefore investigate the long-term effects of pharmacological treatment of PG, including measures after the pharmacological treatment is discontinued, and investigate whether relapse rates are comparable to that of CBT.

Another issue is the possible negative side effects associated with the use of pharmacological agents. The most common side effects (incidence > 5%) associated with escitalopram include nausea, insomnia, fatigue, diarrhea, dizziness, dry mouth, somnolence, and ejaculation failure (Baldwin, Reines, Guiton, & Weiller, 2007). However, most patients do not experience any side effects, and if a side effect does occur, it is usually mild to moderate in severity, and passes within the first 2-3 weeks. The possibilities of experiencing negative side effects may off course make some clients reluctant to try medication. It is therefore important that the patient is well informed of possible side effects, and that they most often are temporarily and will decrease after 2-3 weeks. Sudden discontinuation may cause unpleasant reactions, and it is therefore important to gradually increase or decrease the prescribed dosage. Few adverse effects were reported in the present treatment study. Most adverse effects were only temporary (mostly within the first 3-4 weeks), but one participant was advised to discontinue

medication after 10 weeks because of development of manic tendencies. Escitalopram has been associated with less negative side-effects than older SSRIs (Huska, et al., 2007), and studies have shown that the drug is well tolerated (Black, et al., 2007).

An advantage of CBT compared to pharmacological treatment is that it is non-evasive and avoids the problem of possible negative side effects of the medication. However, as already mentioned, CBT demands more therapist resources and there is often a shortage of skilled therapist and long waiting lists. A possible combination of the two treatments would be to offer pharmacological treatment in the initial treatment phase while waiting for CBT.

Study II found that pathological impulsivity was a significant predictor of PG. High levels of impulsivity in PGs have previously been found to be related to drop-out from treatment (Leblond, et al., 2003) and has been suggested to lead to lower effectiveness of treatment interventions (Goudriaan, et al., 2008). An assessment of impulsivity prior to treatment could therefore be beneficial in order to provide adapted treatment interventions for impulsive gamblers. Treatment of impulsive gamblers may benefit from emphasizing motivational enhancement which may avoid high drop-out rates. Furthermore, Ledgerwood and Petry (2005) suggested that impulsive gamblers may benefit more from CBT than other approaches because CBT offers a structured approach and focuses on distorted thinking about gambling and the restructuring of the environment to make gambling less accessible. Helping clients to identify and handle personal high risk situations may also be important to improve the effect of treatment interventions for impulsive gamblers.

Openness was to be negatively associated with PG in the second study, and low scores on this domain are associated with a preference for the familiar, unanalytic thinking and being unappreciative of intellectual challenges. One may speculate that the challenging of cognitive distortions and teaching of problem-solving skills is especially important in therapy with these gamblers.

The results of the third study showed that gamblers are heterogeneous, and this may have implications for designing effective and targeted treatment interventions. In other areas of addictive behaviors sub-typing individuals on the basis of personality and underlying motivations for substance use has led to the development of motivation-matched treatment programs which have been shown to be effective (Conrod et al., 2000). Similarly, motivation-matched treatment for PGs could be developed and presumably enhance treatment efficacy. Gamblers who gamble primarily for the thrill or sensation seeking could be encouraged to find other less harmful ways of fulfilling their need for stimulation, while gamblers who engage in gambling activities in order to cope with stress or other negative emotions may benefit more from training of coping skills and learning how to deal with their underlying pathology. High levels of psychopathology were found in approximately half the gamblers in Study III, and one may assume that for these individuals, the gambling served a function of escaping from non-gambling related problems. As such, there may exist an underlying co-morbid disorder that also needs to be targeted in therapy. However, half of the gamblers in Study III showed personality profiles within the normal range, and they may gamble for reasons other than coping with psychopathology or being predisposed by certain personality characteristics. As this group seemed to resemble the behaviorally conditioned problem gamblers in Blaszczynski and Nower's model, one might expect that these subjects have adequate resources to benefit

from minimal interventions or standard short-term cognitive behavioral therapy as suggested by Blaszczynski and Nower (2002) while those individuals with other co-morbid disorders may need longer and more supportive treatment.

4.4 Contributions to the Field

This thesis showed that pharmacological treatment with SSRIs have great potentials in treating PGs and may be as effective as the established CBT. This can significantly reduce clinicians' time and reduce waiting-lists, and may also have the potential for lowering thresholds for treatment seeking. However, as this was a small scaled pilot study more research is need to establish the relative effectiveness of the different treatment modalities and whether pharmacological treatment with escitalopram is equally effective for all gamblers independent of underlying co-morbidity.

As there has been a controversy regarding whether gamblers are characterized by impulsivity and sensation seeking, this body of research contributed to the field in supporting the notion of pathological impulsivity in pathological gamblers. Pathological impulsivity was found to be a significant predictor of PG status even when controlling for other personality variables and other measures of impulsivity, such as motor impulsiveness, attentional impulsiveness, and non-planning impulsiveness. This further indicates that impulsivity is a complex construct, and underlines the importance of including different measures of impulsivity. Goudriaan et al. (2008) found that self-reported impulsivity, as measured by the BIS-11, did not significantly predict relapse in pathological gamblers, while neurocognitive measures of disinhibition did predict relapse. The difference between the self-reported and the neurocognitive indicator of impulsivity

may be a result of that the BIS-11 do not capture the type of impulsivity that is characteristics of gamblers. Hence, the shorter EIS-nI may more readily capture the type of pathological impulsivity that is characteristics of gamblers in particular. In the present study, sensation seeking was measured as both need for novelty and need for stimulus intensity, however only the latter type of sensation seeking remained a significant predictor of PG when controlling for the other variables. This underscores the need for differentiating between different aspects of impulsivity and sensation seeking.

Although some studies have investigated MMPI profiles among pathological gamblers, few studies have used the revised version, MMPI-2, which has undergone considerable changes from the first version. This study therefore contributed to up-dating the field in this area. Further, this line of research confirmed earlier proposals that gamblers are a heterogeneous population, and this may have consequences for designing adapted treatment interventions. As half of the gamblers showed quite normal personality profiles and low levels of psychopathology, this group may benefit from standard short-term treatment interventions. However, the other groups with elevated profiles may need more adapted treatment interventions which also take the underlying co-morbidity and pathology into account. One may assume that this group would hence need longer treatment.

4.5 Directions for Future Research

Mediators of treatment or processes of change have been overlooked in the treatment literature of pathological gambling (Blaszczynski & Steel, 1998; Pallesen, et al., 2005). When certain treatments have established efficacy, one should put efforts into

investigating what are the effective mechanisms of change. It is often the assumption that the gambling behavior has changed as a result of the specific techniques. However, research has shown that common or non-specific treatment factors, such as alliance, client resources and expectations (placebo effects), account for more than the specific techniques (Lambert, 1992). When process evaluations are lacking and mediators of behavioral change are not assessed, there is a threat to the internal validity and one cannot make accurate conclusions concerning what works in therapy. Future research should therefore focus on identifying components of treatment that are responsible for behavioral change and assessing mediators of change in the different treatment approaches to pathological gambling.

Limited empirically validated data on effective treatments for PGs with co-occurring disorders exist (Hollander, et al., 2004). It may well be that the response to pharmacological treatments for PG is conditioned by the presence/absence of co-morbidity which is targeted by a particular drug. For example, patients with co-morbid depression may respond better to treatment with escitalopram than non-depressed PGs since escitalopram also targets the underlying depressive symptoms. In a similar manner, patients with co-morbid substance abuse/dependence may benefit more from opioid antagonists than those without co-morbid substance abuse. As a consequence, future studies should therefore take co-morbidity into account when assessing the effectiveness of pharmacological treatments for PGs. Future studies of pharmacological treatment for gamblers should include large samples that allow for sub-grouping gamblers based on possible underlying psychopathology when investigating the effect of different pharmacological agents.

PGs have been shown to be a heterogeneous population (e.g., Myrseth, Pallesen, Molde, Havik, & Notelaers, 2010; Stewart, et al., 2008; Vachon & Bagby, 2009), resulting in a need to differentiate between subgroups of gamblers. As Myrseth, Pallesen, Molde, Johnsen and Lorvik (2009) stated, there is a need to put a greater emphasis on gamblers' preference for what type of gambling they prefer when investigating the antecedents and maintenance of problem gambling. Gambling preference has been shown to be related to differences in cognitive distortions (Myrseth, Brunborg, et al., 2010), sensation seeking (Bonnaire, Bungener, & Varescon, 2006; Coventry & Brown, 1993), personality traits (Adkins, et al., 1987), and neurobiological functioning (van Holst, et al., 2009). Thus, information regarding subgroups of gamblers who display different personality characteristics may be important as far as designing more effective and adapted treatment and prevention interventions. Including different sub-groups of gamblers in studies assessing the effectiveness of treatment or prevention interventions may introduce excessive variance, and hence be a confounding factor. Future studies should consequently include large samples which allow for a differentiation among subgroups of gamblers rather than treating all problem gamblers as a homogenous population.

Within epidemiological research the use of different instruments to identify problem and pathological gamblers have resulted in different prevalence rates in the same population (e.g., Götestam & Johansson, 2009). Further, treatment outcome studies use a variety of different measures to assess outcomes, making the comparison of effectiveness between different studies difficult (Pallesen, et al., 2005). There is consequently a need to validate existing measures and develop more uniform and universal measures for evaluating treatment outcomes in the gambling field. Shaffer, LaBrie, et al. (2004) argue

that diagnostic immaturity characterizes the gambling field. Firstly, different screens often measure different dimensions (behavior, attitudes, or cognitions) and the way the measure them are often arbitrary. Secondly, different screens also use different time frames and have consequently different sensitivity for recent changes. They further question the accuracy of self-report (due to e.g., memory bias and self-presentation bias) and suggest that more time and research should be spent on developing advanced unobtrusive diagnostic tools that do not rely on self-report, such as fMRI, event-related potential, neurochemical analysis, psychophysiologcal assessment, and reaction time measures (Shaffer, LaBrie, et al., 2004).

Because most gamblers do not seek treatment, it would be of great value to identify vulnerability factors that can be targeted through prevention programs. Most of the research on vulnerability factors and personality characteristics among gamblers has been cross-sectional; hence there is a need for more longitudinal and prospective research to draw conclusions about cause and effect. Longitudinal studies also enable the investigation of whether personality characteristic among gamblers will be stable over time, or if such characteristics are subject to change in conjunction with treatment. Future studies should also attempt to investigate whether treatment seeking gamblers differ from non-treatment seeking gamblers in terms of personality traits and levels of psychopathology. Knowledge concerning vulnerability factors and characteristics of non-treatment seeking gamblers may allow for targeting the non-treatment seeking population through prevention intervention.

5. Conclusions

Pathological gambling is associated with substantial negative consequences, and there is a need for cost-effective treatments. The present thesis endeavored to investigate whether pharmacological treatment which has the potential of lowering thresholds for treatment seeking and improving access to treatment would yield comparable effects to the well-established CBT for problem gambling. The present research suggests that both pharmacological treatment and CBT are associated with improvement in the short term. Further, CBT alone and in combination with pharmacological treatment seem to be associated with long-term effects in a 6-months perspective. Further studies are warranted to establish the relative effectiveness of these various treatment modalities.

Furthermore, the present research suggested that there are differences in personality variables and education level between pathological gamblers and non-pathological gamblers, and evidence supporting heterogeneity among gamblers were found. Hence, one cannot talk about a general "gamblers personality" as such. The results indicated that approximately half of the gamblers show quite normal personality profiles and are characterized by low levels of psychopathology, while the other half is characterized by considerable pathology, especially symptoms of anxiety and depression. The existence of different subtypes of gamblers confirms previous findings, and may have implications for designing effective and more targeted treatment interventions.

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Appendix A

Diagnostic criteria for 312.31 pathological gambling

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 experiences, 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 of 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 significant relationship, job, or educational or career opportunity because of gambling

(10) relies on others to provide money to relive a desperate financial situation caused by gambling

B. The gambling behavior is not better accounted for by a Manic Episode.

American Psychiatric Association (1994). Diagnostic and Statistical Manual for mental disorders, 4th ed. Washington, DC: American Psychiatric Association.

Appendix B

Overview of gambling assessment instruments

Instrument	Content area	Number of items	Administration	Scoring
National Opinion Research center DSM-IV Screen for Gambling Problems (NODS; Gerstein et al., 1999)	DSM-IV criteria for PG, including both life-time and past year gambling problems	34	Interview/ questionnaire	1 point for each scored item Range 1-10 Score of 5+ indicates PG
South Oaks Gambling Screen – Revised (SOGS-R; Lesieur & Blume, 1987)	Type and frequency of gambling, signs and symptoms of PG, negative consequences of gambling	20	Questionnaire/ self-report	1 point for each scored items Range 0-20 Score of 5+ indicates probable PG
South Oaks Gambling Screen Revised for Adolescents (SOGS-RA; Winters et al., 1993, 1995)	Signs and symptoms of PG, negative consequences of gambling	12	Questionnaire/ self-report	1 point for each item Range 0-12 Score of 4+ indicates problem gambling
Gambling Symptoms Assessment Scale (G-SAS; Kim et al, 2001)	Gambling symptoms, urges and thoughts related to gambling during the last week	12	Questionnaire/ self-report	Each item scored 0-4 Range 0-48 Scores > 30 indicate sever problems Scores 21-30 indicate mild symptoms Scores < 21 indicate mild symptoms
Lie/Bet Questionnaire (Johnson et al., 1997)	Lying about gambling, and betting more and more money	2	Questionnaire/ self-report	Answering yes to one or both items indicates problem gambling
Massachusetts Adolescence Gambling Screen (MAGS; Shaffer et al, 1994)	2 subscales; one 14 item measure of signs and symptoms of PG and one 12 item measure of the DSM-IV criteria for PG	26	Questionnaire/ self-report	8 items are scored 1 point and 4 items are scored ½ point Range 0-10 Score of 5+ indicates PG
Pathological Gambling Behavioural Self-Report Scale (PGBS; Hollander et al., 2005)	Net losses, number of gambling occasions, and total time spent gambling during the last week	3	Questionnaire/ self-report	Open-ended questions
Gamblers Self-Efficacy Questionnaire (GSEQ; May et al., 2004)	Gambling self-efficacy (control) in specific situations	16	Questionnaire/ self-report	Each item scored 1-100 (increments of 20)

				Overall score calculated by the mean response to all items
Pathological Gambling 100 mm Visual Analog Craving Scale (PGVAC; Hollander et al., 2005)	Gambling urges and control	5	Questionnaire/ self-report	Each item scored 0-100 Range 0-500
Addiction Severity Index adapted for Gambling (ASI-G; Lesieur & Blume, 1992)	Modification of ASI by adding items of gambling frequency and problems associated with gambling Measure 7 domains; physical health, employment status, alcohol abuse, drug abuse, criminal behavior, interpersonal relationships, mental health, and gambling	7 domains	Interview	Domain specific composite score Range 0-1
Yale-Brown Obsessive-Compulsive Scale Modified for Pathological Gambling (PG-Y-BOCS; Hollander et al., 1998)	Measures gambling urges and behavior. 2 subscales; Gambling Urge/Thought and Gambling Behavior	10	Questionnaire/ self-report	Each item scored 0-4 Range 0-40
Canadian Problem Gambling Index (CPGI; Ferris et al., 2001)	3 sections; gambling involvement, problem gambling assessment, and correlates of PG	31	Questionnaire/ self-report	9 items cored 0-3 Range 0-27 8+ indicates problem gambling
Structured Clinical Interview for PG (SCI-PG; Grant et al., 2004)	Diagnostic criteria for PG	10	Interview	Diagnosis present/absent
Information Biases Scale (IBS; Jefferson & Nicki, 2003)	Cognitive distortions in VLT players	25	Questionnaire/ self-report	Each item scored 1-7 Range 25-175
Gamblers Belief Questionnaire (GBQ; Steenberg et al., 2002)	Thought and cognitive distortions about gambling. 2 subscales; Luck/perseverance and Illusion of control	21	Questionnaire/ self-report	Each item scored 1-7 Range 21-148
Gambling Passion Scale (GPS; Rousseau et al., 2002)	Passion toward gambling. 2 subscales; obsessive passion and harmonious passion	10	Questionnaire/ self-report	Each item scored 1-7 Range 10-70 (5-35 for each subscale)
Gambling Urge Scale (GUS; Namrata & Oei, 2004)			Questionnaire/ self-report	
Scale of Gambling Choices (SGC; Baron et al., 1995)	Measures loss of control regarding gambling behavior. Includes 3 factors; ability to control gambling, intention to limit gambling, and loss of control over gambling	18	Questionnaire/ self-report	Each item scored 1-5 Range 18-90

Readiness to Change Questionnaire (RCQ; Rollnick et al., 1992)	Readiness to change according to the stages-of-change model. 3 subscales; pre-contemplation, contemplation and action stage	12	Questionnaire/ self-report	Each item scored 1-3 Range 12-36
Time-Line Follow Back (TLFB; Hodkins & Marachuk, 2003)	Calendar to measure days with gambling and money spent on gambling	Past 4 weeks, 3 months, 1 year etc.	Interview	Counting days of gambling and summing up amount of money lost due to gambling
Diagnostic Interview for Gambling Severity (DIGS; Winters et al., 2002)	Demographics, gambling involvement, treatment history, onset of gambling, gambling frequency, amounts of money bet/lost, sources of borrowed money, financial and legal problems, mental health screen, other impulse control disorders, medical status, family and social functioning, and diagnostic symptoms (lifetime and past year)	20 diagnostic symptoms to measure 10 criteria	Interview	10 diagnostic items scored 0/1 Range 0-10 5+ indicates PG
Gamblers Anonymous 20 Questions (GA-20)	Signs and symptoms of compulsive gambling; negative consequences	20	Questionnaire/ self-report	Each item scored 0/1 Range 0-20 7+ indicates compulsive gambler
Gambling Assessment Module (GAM; Cunningham-Williams et al., 2005)	Assesses diagnostic criteria according to DSM-III and DSM-III-R; social-, psychological-, and financial consequences of gambling etc.	27 item demographics 40 items gambling section 7 items interviewer observation	Interview/ Questionnaire	10 diagnostic items scored 0/1 Range 0-10 5+ indicates PG
Gambling Treatment Outcome Monitoring System (GAMTOMS; Stinchfield et al., 2007)	The Gambling Treatment Admission Questionnaire includes measure of diagnostic criteria, measures of gambling problem severity, including the SOGS, gambling frequency, gambling-related financial problems, and legal problems	142 item Gambling Treatment Admission Questionnaire, 10-item measure of diagnostic criteria	Questionnaire/ self-report	10 diagnostic criteria scored 0/1 Range 0-10 5+ indicates PG

Appendix C

A brief overview of the content in the manualized treatment sessions

Session 1

Session 1 provides an overview of the treatment program, and includes a general introduction to CBT and to the treatment manual. Instructions regarding self-monitoring of one's own gambling behavior are given, and the therapist presented the "think-aloud-method." In addition to weekly self-monitoring and maintaining a written record of gambling episodes, the clients are given homework assignments between each session which are reviewed during the next session. The first homework assignments are directed at investigating the positive and negative aspects of the client's behavior, the reasons for stopping/continuing gambling, and what is difficult about giving up gambling.

Session 2

Exposure with response prevention is conducted in Session 2. After an introduction with an explanation of the rationale for the technique, the client is exposed to a gambling situation. Clients are then instructed to use the "thinking aloud method", and the therapist follows the client's gambling-related thoughts and urges to gamble through the use of guided dialogue. The rationale for this technique is the potential reactivity of hearing your own thoughts expressed out loud (Griffiths, 1993). Clients are encouraged to continue exposure training on a weekly basis throughout the treatment, either alone or assisted by a friend/family member. Homework assignments in which clients are asked to identify the triggers of gambling behavior (events, days/times, people, or emotions) are provided. Clients are also asked to increase pleasant activities and create a list of alternative activities to gambling.

Session 3

At the beginning of Session 3 and each subsequent session, the therapist starts by examining gambling episodes or high risk situations from the previous week. Clients are encouraged to reward him or herself for non-gambling days or for successfully handling triggers or high risk situations. The therapist helps the gambler to better understand the pattern of his or her gambling behavior and to identify the triggers of gambling. The central mechanisms in gambling, such as return rates, are examined through "the funnel diagram" (Coman, 2003) and "Coman's factory roof line graph" (Coman, 2005). Homework assignments focusing on stimulus control (avoiding gambling venues and gambling related stimuli), coping with urges (developing alternative responses to temptations and cravings) and increasing behavioral and social reinforcement (engaging in other leisure activities and socializing with people who do not gamble) are given.

Session 4

After reviewing homework assignments and the past week's gambling episodes, a documentary is shown which gives information about the gambling industry and how electronic gaming machines work. The effect of structural characteristics such as light, sound, near-wins and operant behavioral principles behind payback rates is discussed, and assignments regarding the role of cognitive distortions are given.

Session 5

This session focuses on cognitive distortions such as the illusion of control, the selective memory of wins, the perception of personal luck, overestimating the odds, superstitious behaviors and the gambler's fallacy. Exercises such as throwing dice are used to challenge cognitive misbelieves and erroneous perceptions about the chance aspect of

gambling. A homework assignment is provided in which the client is asked to identify which thoughts "justify" further gambling when they are losing/winning.

Session 6

Session 6 focuses on the motivation to quit gambling. The therapist uses motivational interviewing to amplify the *importance of* giving up gambling and the *confidence* the client has that he/she can actually do so. Decision-making and common excuses used to postpone decision-making are discussed. Clients are encouraged to make a written statement about further gambling and exercises on motivational enhancement are provided.

Session 7

This session focuses on identifying triggers and risk situations, and finding alternative strategies for handling such triggers. Clients are also trained in challenging cognitive distortions, thoughts and feelings that commonly lead to gambling. Exercises related to relapse prevention are provided as homework.

Session 8

The "stages of change model" (Prochaska & DiClemente, 1982; 1983; 1984) is presented, and clients are invited to reflect over the different stages of the change process. Strategies to avoid lapses and relapses in the future, as well as how to continue to support change are discussed.