Report to the Ministry of Health
Feedback to MOH re Emerging Trends in National & International Literature

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Literature	Findings	Comment
Disordered Gambling: etiology, trajectory, and clinical considerations. Authors: Shaffer HJ & Martin R (2011) The Annual Review of Clinical Psychology, 7, 483-510 doi: 10.1146/annurev-clinpsy-040510-143928	• This article reviewed the history, etiology and trajectory of pathological gambling, and research into diagnosis, treatment, and public health issues. • Pathological gambling (PG) interest and research has rapidly advanced during the past 20 years and we have better understandings of excessive gambling, its biopsychosocial factors, and its development as a problem. There has been a dramatic increase in PG research since the latter half of last century, and particularly recently • Physical neuro-adaptation (tolerance and withdrawal effects) compound with psychosocial effects (shame, guilt, depression, debt and loss of control), and may despite these problems, crave and continue gambling • Despite DSM defining PG as a progressive, preoccupying behaviour with increased	• The substantial review paper provides an important summary of recent findings that (mostly) accord with directions that NZ is currently moving in respect of PG. • Its description of PG as neither progressive nor stable emphasises similarities between PG and other addictions. Many people affected by either sub-clinical (described as displaying some symptoms or 'shadow syndrome') or pathology may move in or out of these categories in many cases with or without treatment. This instability may be contributed to by both the influence of changeable bio-psycho-social factors (one, albeit increasingly acceptable, model of addictions) and the fluid categorisation of PG under the current DSM4 diagnostic manual. • However, somewhat counter-intuitively, the authors describe the prevalence rate of PG

	gambling of money despite the negative costs, it is now possible, from longitudinal studies, that it is not either progressive or stable • Prevalence range of problem gambling has remained constant over time despite increased exposure to gambling opportunities (but may increase temporarily as a novelty effect) because people adapt to new gambling. Research indicates rates of 0.7% in 1976 and similar rates of 0.6% in 2005, three decades later. • Although gambling machines are described as addictive because of their variable ratio of reinforcement, a better predictor of problem gambling is the number of gambling games participated in (i.e. gambling involvement), rather than any one game. They cite research supporting this change of focus (Welte et al 2009; LaPlante et al 2009). • Self-reported gambling behaviour (much of earlier research) is unreliable, while recent research is based upon more reliable actual gambling (e.g. Internet, lottery). • Theory describing how problem gambling develops is sparse, but suggests it is complex and due to many factors. Addictions seem to be bio-psycho-social in both development and their being maintained. There may also be an underlying addiction syndrome with different addictions being an	as being stable over the past three or more decades. They further describe that this has occurred despite growth in gambling opportunities, attributing this 'stability' to adaptation by the public to novel gambling, at most, spiking in PG prevalence following new gambling, which then reduces. This is similar to a hypothesis tentatively raised by the Australian Productivity Commission (2011) which acknowledges the influence on prevalence rates upon screening tools used. If PG is difficult to describe, is unstable in its course, and few longitudinal studies currently exist, then snapshots of prevalence may be somewhat unreliable, and due to the stigma of PG (and possible increase in this perception over time), be under-estimating the prevalence level of PG. Between 2000 and 2007, four NZ surveys identified prevalence rates of sub-clinical PG and PG ('Level 2 and 3 gambling') of 1.35% (2000), 1.9% (2003), 9% (2007) and 2.6% (2007) indicating at least a doubling of prevalence, and perhaps demonstrating the problems in such surveys. • The PG syndrome model of an underlying 'addiction risk' across addictions, with the expression or object of addictions (e.g. gambling, drugs, excessive shopping) and influenced by the biopsychosocial issues specific to individuals, provides a credible

	expression of that syndrome. Risk factors that exist (and protective factors may counter these) may decide whether the underlying syndrome is expressed in the presence of the addictive object (e.g. PG). • Neurotransmitter variables appear to be associated with many addictions (increased dopamine with PG, sexual behaviours) while decreased activation in impulse regulation areas of the brain. • Studies show genetics have an important role in PG with 50%-60% of the variation in PG risk due to genetics (Lobo & Kennedy 2009). The existence of PG family members signals higher likelihood of PG, and is supported in twin studies. The authors conclude that genetics may increase addiction risk generally. • Women appear to develop PG later (but more rapidly) than men do, and women may seek help earlier. However, gender may be less a risk factor for PG than other factors. • PGs are more likely to have mental health or substance use disorders (SUD) than non-PGs. Those with psychiatric disorders may be 17 times more likely to develop PG (Kessler et al 2008). Nearly half PGs will have had a mood or anxiety problem, 75% an alcohol disorder, and over 60% a personality disorder.	perspective that may assist treatment, yet also inform a public health model. • The emphasis upon similarities across addictions, biological and genetic factors that receive tentative support from new scanning methods (PET and fMRI), and in particular, the focus upon addressing co-existing issues with PG, appear to fit within the NZ co-existing problems approach, and broad competencies initiatives. • This paper is a comprehensive and timely review of the current state of a quickly developing addiction. Although changes to definition of PG in DSM5 scheduled for release in 2013 highlighted in the paper may result in some of the recent research being less informative, the alignment of PG with alcohol and drug addictions within a new category ‘addiction and related disorders’ signals a greater acceptance of PG within the more mainstream mental health fields.

	• Social factors for risk include early life gambling, early wins after gambling, and lower socio-economic status. • Adaptation may explain why PG prevalence hasn't increased over time, and one study cited showed long-term casino employees had lower PG prevalence than recent employees (Shaffer et al 1999). Internet gambling appears to be lower than expected, suggesting exposure to gambling may not be the main factor in PG prevalence or intensity. • Much of PG research is with PG treatment seekers who may not be representative of all PGs. • Treatment of PG may be influenced by the DSM5 signalled re-categorisation of PG from an impulse disorder to a substance-related disorder under a new category: 'addiction and related disorders' and an assumption that PG has aspects in common with substance use disorders (e.g. clinical expression, etiology, comorbidity, physiology and treatment). The current threshold in DSM4 of 5 out of 10 criteria may also be lowered. • PGs are reluctant help-seekers for gambling issues, although in one study, although none had sought PG help, 49% of PGs had sought help for other mental disorders (Kessler et al 2008). Neither PGs nor their	

	health professionals recognised the need for such help. In another study 5.5% of PGs had sought help for their gambling (Slutske et al 2006). The authors concluded: ○ 'it is imperative that clinicians learn about gambling-related disorders so that patients can integrate their care without having to enter specialised treatment' p498 • Because PGs change more rapidly than expected as well as being in treatment for other problems, it is imperative that brief public health screens, guides for self-directed change, and e-health resources be available, with an emphasis on self-help. • Approximately 98% of PGs have a co-existing disorder, with this either pre-existing or emerging at the same time as PG in 76.5% of cases (Kessler et al 2008). The PG can exacerbate the earlier disorder, or if the PG developed first (23.5% of cases), then it may stimulate the new disorder, which in turn, exacerbates the PG. Identifying these co-existing disorders and sub-clinical syndromes may produce better clinical outcomes. • Because of co-existing disorders, the variability of the PG and the course of the PG may be chronic, with intermittent treatment needs throughout their lifetime.	

	• Treatment post-screening for PG is similar to other addictions, with CBT and motivational enhancement being effective. Medications such as naltrexone and SSRI antidepressants have also assisted, as they have with other addictions. • Self-exclusion programmes have shown some efficacy, but self-exclusion may not be the cause of improvement but rather, that the act may reflect pre-existing improved motivation. • PG may be best treated with a 'cocktail' treatment to address PG and co-existing problems, and the use of unobtrusive diagnostic tools.	
Gaming machine addiction: the role of avoidance, accessibility and social support. Authors: Thomas A, Allen F & Phillips, J (2011). Psychology of Addictive Behaviours, 25(4), 738-744	• This research addressed gambling machines (EGMs) and the process from social to problem gambling. An overarching addiction framework was used (similar to the above Shaffer et al model of an underlying addiction syndrome, with different expressions or objects of addiction – in this case, gambling). • A dominant theory in many models of addiction is that a desire to avoid stress drives the addiction, with the behaviour or the drug/alcohol providing a temporary cognitive and/or behavioural escape. The environment (the gambling venue) provides a break from stressors, with the continuous games, lights and music providing a	• Although excessive gambling can produce problems that may produce a desire to escape from, other research (Kessler et al 2008) has identified that PGs in the majority of cases have co-existing problems that pre-date their gambling problems. • This research describes how EGMs can provide a drive to gamble that may be independent from winning money, and which provides a reward or reinforcement whenever gambling occurs, win or lose. Escape from dysphoria may be effected through the repetitive nature of EGM gambling, and accessibility (geographically, and at most times) may become an important issue to address.

	cognitive distraction from stressors. Access is important, as the person must be regularly exposed to the gambling in order to learn that gambling can provide avoidance of stress. • Geographical accessibility as well as being available when necessary (temporal availability), is associated with both gambling uptake and problems, as was the absence of social support (or presence of social contact if the relationship was detrimental to the gambler). This geographical and temporal accessibility was more important than the social environment (e.g. welcoming). • The process appeared to be experience of stressors for the gambler, then an avoidance-motivated gambling behaviour to cope (albeit dysfunctionally), then leading into addiction to the gambling. • The authors noted that the combination of non-skill games, flashing lights and comfortable EGM venues make a more attractive option for avoidance than gambling that requires more involvement (than EGMs). • They finally concluded from the research that accessible gambling (EGMs) were in turn associated with both how frequently the gamblers gambled, and also how severe their gambling problems were, just as	• This research provides support for restricting both numbers of venues and opening times for venues, if prevalence of PG risk is a goal preferred over public choice. • The research also is support for treatment that addresses underlying stress and development of new more functional coping behaviours. • These findings provide further evidence for the co-existing problem approach adopted in NZ, as well as providing support for therapy that develops functional coping behaviours to substitute for the gambling (avoidance-motivated) learned behaviour.

	accessibility to alcohol will influence consumption.	
Drinking patterns of pathological gamblers before, during, and after gambling treatment Authors: Rash C, Weinstock J & Petry, N (2011). Psychology of Addictive Behaviours, 25(4), 664-674	• PG and alcohol disorders commonly co-occur (45%-73% PGs have an alcohol problem sometime during their lifetime), with treatment seeking PGs also high (59%; Kausch 2003). Research indicates that such alcohol and PG problems co-occur and are experienced at the same time (i.e. gambling and drinking sessions also co-occur) and result in more risky gambling and greater negative consequences. • The majority of non-pathological gamblers are also risky drinkers (4 or more drinks per day for males, 3 for females) • The authors noted that research indicated that the quantity of alcohol use appeared to be related to the severity of gambling problems, with the strength of the relationship increasing as alcohol use increased. Repeated pairing of alcohol and gambling may result in gambling becoming a conditioned cue for drinking – if so reduced gambling may result in reduced drinking. • Reduced drinking may also occur through gamblers addressing their gambling may become open to other changes that improve their health. • This may counter the possibility that switching addictions may be less likely to occur when receiving treatment for PG. Two	• Alcohol and gambling are often available in the same venues, and addiction-switching appears to be a common outcome, both indicating that the findings of this research may be important in informing the content of treatment for problematic levels of both behaviours. • A surprising finding of the study was that severity of alcohol and gambling problems when they co-occurred, were inversely related – low PG severity correlated positively with higher alcohol problem severity and vice versa. This appears to be contrary to the Te Ariari trends of addictions and other mental health problems (including other addictions) and NZ findings (Sullivan & Steenhuisen 2006), where alcohol or other drugs and PG severity were positively associated. • An important caveat to this research may be the time period measured (although the focus was on alcohol use during PG treatment), with 12 weeks post-treatment period being a relatively brief period to draw (possibly enduring) conclusions from. DSM4 and other evidence suggests that PG is a recurrent and unstable condition, while outcomes are starting to be measured over

	studies (Toneatto et al 2002 & Stinchfield et al 2005) studying alcohol use post-treatment, identified lower drinking levels and support that switching addictions/symptom substitution may not usually occur. The current authors noted that both studies measured drinking frequency rather than quantity and therefore did not measure risk for binge drinking. The current study measured alcohol use during the three 12 week periods prior, during and after PG treatment, and analysed data from two previous studies (n=333). • Of the participants (help-seeking gamblers), 26% of all participants were at-risk drinkers, and of those who drank alcohol, 47% were at-risk drinkers. • Although research indicated an overall reduction in alcohol use for those entering treatment for PG, 50 of the 76 drinkers who were risky-drinkers at some time, remained risky-drinkers both during and in the following 12 weeks of PG treatment, suggesting the need for further focus on alcohol use. • Reduced alcohol use did not appear to be a result of the PG treatment because the reductions occurred either before PG treatment, or within the first PG treatment session. The cause may have due to one or more of three possibilities: 1) the reduced conditioned cues for alcohol use (less	considerably longer periods (e.g. 2 years) to allow for this and other factors. • Nevertheless, the importance of incorporation of alcohol issues within a treatment plan to address conditioned cues (continued use of alcohol triggering thoughts of gambling following PG treatment), or to enhance motivation to address alcohol issues during an opportunistic self review of wellbeing, may be timely and best practice. The other suggestion that financial restraints may reduce alcohol use appears counter-intuitive, as if gambling is reduced during PG treatment, more funds would appear to become available for alcohol, rather than less. • The study acknowledges that although drinking sessions may reduce, the level of binge drinking may become (or still be) a problem, and that further research is warranted; this aligns with the concerns that two-thirds of PGs who have risky alcohol use remain within that category, and that integration of alcohol issues within a PG treatment plan is supported. This is a current approach within the NZ strategy to address problem gambling and the study provides further support for this.

	gambling paired with alcohol), 2) financial restraints from gambling reducing available money for alcohol, or 3) decision to change gambling spreading to or motivating other health decisions (e.g. reducing alcohol use, eating more healthily, or exercising). Multiple health targets did not appear to interfere with the PG treatment and may improve overall outcomes. • A finding was that although the link between alcohol and gambling was high, the severity was inversely related, with less severe PGs drinking more alcohol than more severely affected PGs. An explanation offered was that less problematic PGs may be less motivated to change either their gambling or their alcohol use. • Within treatment content, those PGs participating in non-gambling social activities as part of treatment had less escalation in alcohol use, not incorporating alcohol use into their social activities. • The authors concluded that although alcohol use reduced when PGs entered treatment, a substantial number (two-thirds) of PGs continued to drink at risky levels, concluding that (additional) interventions targeting alcohol use may be warranted to reduce such risk further. • Limitations to the study acknowledged by the authors were that the conclusions could only	

	be drawn in respect of PG and alcohol use and not PG and other drugs, and that there wasn't any 'no-treatment' control group.	