PSYCHOLOGY MISCELLANY

No.97 Supplement - July 2017

Other Clinical Psychology Topics

Kevin Brewer

ISSN: 1754-2200

Orsett Psychological Services
PO Box 179
Grays
Essex
RM16 3EW
UK

orsettpsychologicalservices@phonecoop.coop

This document is produced under two principles:

1. All work is sourced to the original authors. The images are all available in the public domain (most from http://commons.wikimedia.org/wiki/Main_Page). You are free to use this document, but, please, quote the source (Kevin Brewer 2017) and do not claim it as you own work.

2. Details of the author are included so that the level of expertise of the writer can be assessed. This compares to documents which are not named and it is not possible to tell if the writer has any knowledge about their subject.

Kevin Brewer BSocSc, MSc

An independent academic psychologist, based in England, who has written extensively on different areas of psychology with an emphasis on the critical stance towards traditional ideas.

A complete listing of his writings at http://psychologywritings.synthasite.com/ and http://kmbpsychology.jottit.com.

CONTENTS

1. SCHIZOPHRENIA AND GENES

1.1. INTRODUCTION

Drug discovery occurs through serendipity ("fortunate accident") or systematic understanding of a disease. According to Dhindsa and Goldstein (2016), current treatments for schizophrenia "fall uniformly" into the former, and this is because of a lack of molecular basis of the illness (ie: full understanding of a genetic basis).

Ripke et al (2014), for example, found over 100 regions of the genome (loci) that could be linked to a risk of schizophrenia [1]. "However, finding a locus is not the same as identifying a causal gene - for instance, loci are often located in sequences that don't encode genes, and many different variations can occur in one region, making it hard to pinpoint what exactly is driving the risk signal. Indeed, these 108 risk factors were not traced to specific genes or variant sequences" (Dhindsa and Goldstein 2016 p162) (appendix 1A).

However, Sekar et al (2016) reported the role of genes on chromosome 6 related to acquired immunity, which play a part in "synaptic pruning" ("a developmental process in which the synaptic connections between neurons are continuously eliminated in the brain until early adulthood"; Dhindsa and Goldstein 2016 p163). Based on a mouse model [2], it is proposed that a particular gene (C4) leads to increased synaptic pruning in individuals who develop schizophrenia.

1.2. EARLY BRAIN DEVELOPMENT

"Disrupted in schizophrenia 1" (DISC1) is the name

---

[1] Finding biological-based diagnoses for mental disorders is a goal for psychiatry (Insel 2016). One recent example is Clementz et al's (2016) discovery of "biotypes" of psychosis. A selection of cognitive and physiological tests on 711 individuals with schizophrenia, bipolar disorder, and schizoaffective disorder distinguished three "biotypes".

[2] The mouse model is very popular in genetic research. For example, Mei et al (2016) presented evidence of restoring certain behavioural deficits that are autism-like in mice. Knock-in mice were genetically engineered to include a gene, whose absence is linked to anxiety, motor co-ordination deficit, social interaction deficits, and repetitive behaviour. Only the latter two behaviours were restored in adult mice. Human knockouts would be better (appendix 1B).

given to a protein, which, when mutated, predisposes the individual to major mental disorders like schizophrenia or major depression. It is active in neurons in the cortex (Greenhill et al 2015).

In a study with mice, Greenhill et al (2015) found that disrupting DISC1 after birth can have long-term effects for brain development (ie: on experience-dependent plasticity of the cortex). The persistent strengthening of synapses (ie: long-term potentiation) was affected, and the "changes may form the basis for the cognitive deficits associated with mutations in DISC1 and the delayed onset of a range of psychiatric symptoms in late adolescence" (Greenhill et al 2015 p424).

The researchers focused on the barrel cortex, which is an area that receives tactile information from the large whiskers (usually forty). All but one whisker were surgically removed from adult mice. Normally, cortical plasticity follows with the single-whisker information "taking over" the whole cortical territory. This process did not occur in mice where DISC1 was temporarily chemically disrupted in the first seven days of life. Transient disruption of DISC1 did not have this effect if it occurred after seven days old. "This indicates that a critical period exists in early development with long-lasting consequences for plasticity expressed much later in adulthood" (Greenhill et al 2015 p426). The consequences of the disruption could not be reversed.

Applying the findings to humans, the loss of plasticity in the adult brain could disrupt working memory function, which is common in schizophrenia (Greenhill et al 2015) [3].

Early onset schizophrenia (before age 18 years) is linked to disturbed development of the nervous system in the early years, and the involvement of the immune system. Recent research by Xu et al (2016) found differences in genes related to interleukin-18 (part of the immune response) in individuals with schizophrenia. The participants were 768 adults with schizophrenia and 1348 controls in northern China (including Beijing province) for the initial genome-wide search for genes. Then the findings were validated with 1957 adult cases and 1509 controls, and finally, with fourteen first-onset schizophrenia drug-naive adolescents and thirteen healthy adolescent controls [4].

---

[3] Bakken et al (2016) reported that 9% of genes had different effects on rhesus monkey and human brain development compared to 22% in rats and humans, and 25% in mice and humans.

[4] From the social point of view, Bateson (1978) introduced the idea of "double bind" to describe "a pathological sequence of circumstances, usually within a familial setting, whereby a maternal figure (or other family member) gives contactory messages to a child, but also fails to give the meta-communicative signals that would allow the child to distinguish and prioritise demands. As a result, the

1.3. EARLY-LIFE EXPOSURE TO DRUGS AND ALCOHOL

The developing cells in the brain can be negatively affected by early-life exposure to drugs and alcohol, but sex differences in the impact are also evident (Terasaki et al 2016).

1. Pre-natal - Exposure to drugs and alcohol here is due to the mother's intake, and higher testosterone levels in males will interact with this, for instance (Terasaki et al 2016).

In one study rats exposed to opiates (eg: methadone) in the womb had low birth weight, but males recovered sooner to the appropriate age-related weight after birth than females (Terasaki et al 2016).

Also mice exposed to heroin in the womb showed differences in memory after birth and in adulthood (eg: males had later recovery of cognitive abilities) (Terasaki et al 2016).

In relation to alcohol, though studies vary between rats and humans, males appear to be more vulnerable to the effects of foetal exposure than females (Terasaki et al 2016).

2. Post-natal - Exposure here occurs where babies born to drug-using mothers have a dependence and so the drug is given to the newborns to wean them off it slowly, or children in pain are given prolonged painkillers. Terasaki et al (2016) reported no sex differences in relation to opiate exposure, though the researchers noted that "future studies are still needed to explore potential sex differences in the effects or interactions of post-natal opiate exposure with environmental conditions (maternal care) or concomitant physiological conditions (pain or withdrawal) as these experiments will better inform our understanding of specific clinical

---

child does not develop an ability to differentiate between orders of meaning and may develop schizophrenia" (Morris 2007 p372). At a social level, Bateson (1978) used the term "schismogenesis" to refer to "the internal differentiation of societies into groups — sometimes becoming extreme enough to fracture the societies — which may be either symmetrical, as when rivalry produces a mutually inciting set of oppositions between subsets of the culture, or complementary, as when hierarchies marked by domination and submission develop between groups... The groups nonetheless remain relatively internally homogeneous" (Morris 2007 p372).

populations and potential sex differences in these populations" (p4).

1.4. APPENDIX 1B - ERA OF DATA

Levin (2014) described now as the "era of data" with large volumes of complex data needing analysis and interpretation [5]. Thus, statistical tools (and computer programmes) are used to "make sense of the myriad of information - in the form of thousands of data points" (Levin 2014). Techniques like multivariate statistics involve "the observation and analysis of may variables simultaneously" (Levin 2014).

Talking about the biomedical sciences, Levin (2014) emphasised "the centrality of data practices and quantification to the modern life sciences", such that there has "a shift from science practiced at the bench to science practiced at the computer". This is "data-centric science" (Levin 2014) [6].

Levin (2014) summed up her argument - "data practices are intertwined with ways of seeing and enacting the biological world, such that multivariate statistics both depend on and reproduce the notion that metabolism is complex" (p558). Furthermore: "Data practices are no different from other forms of scientific practice: they appear as objective and natural, but are caught up in socio-cultural, economic, and political networks" (Levin 2014 pp559-560).

Levin (2014) was studying metabolomics, which is the study of genes, molecules, and process in the body's metabolism, but her points about data practices can be applied to research into mental disorders that focus on analysing large amounts of genetic data in the search for causes. In popular terminology, the search for the gene for X.

Returning to Levin's (2014) work, she concluded: "...multivariate statistical practices give life to metabolism by generating non-linear, multivariate, and dynamic ways of engaging with the molecular biological world. It might seem, even, that through these practices 'life itself' is construed as something emergent, temporal, multiple, and dynamic (Rose, 2013). Emerging practices shift away from a linear and univariate view of biology, to embrace the 'multi-dimensional structures' and dynamic nature of biological data. In doing so, they allow researchers to grapple with complex metabolic

[5] In relation to the use of such data to predict behaviour, Siegel (2013) entitled a monograph, "predictive analytics: the power to predict who will click, buy, lie, or die" (Lane 2016).

[6] Pentland (2014) argued that informed consent to the use of data should include allow individuals to be able to delete their data at will (Lane 2016).

pathways and meanings. With multivariate statistical practices, 'the objects of molecular biology are becoming tangible and workable in new ways' (Myers 2008). Metabolism emerges as something multiple and interconnected, but still fundamentally informational and statistical" (p571).

In relation to the amount of data generally, Sarewitz (2016) coined the phrase "datageddon" as "big-data projects try to tackle complex problems with massive data-sets, creating an almost infinite number of possible hypotheses to test within that system. Those data sets therefore generate results that look meaningful but have no real application..." (Scudellari 2017 p453).

1.5. APPENDIX 1B - HUMAN KNOCKOUTS

Many techniques to study genes are not with humans. However, there rare groups of humans who lack functional copies of specific genes due to nonsense, frameshift and splice-site mutations. These mutations produce a copy of a gene that is non-functional because it has an abnormal protein, say (Plenge 2017).

Usually if one copy of the gene is non-functional from one parent, the other copy from the other parent is functional and the gene functions normally. But when both copies are non-functional, this is called a homozygous null mutation (Plenge 2017).

This mutation is more common among genetically closely related parents (eg: first cousins), and Saleheen et al (2017) studied first cousin marriages in Pakistan. These researchers studied 1843 individuals and found 1317 different non-functional genes (ie: 7% of known protein-coding genes; Plenge 2017). Relating these genes to health records, Saleheen et al (2017) found, for example, a particular protein that is linked to low-density lipoprotein in the blood stream.

Other large scale human knockout populations studied include British adults of Pakistani heritage (Narasimhan et al 2016), and Icelanders (Sulem et al 2015).

1.6. REFERENCES

Bakken, T.E et al (2016) A comprehensive transcriptional map of primate brain development Nature 535, 7612, 367-375

Bateson, G (1978) Steps to an Ecology of Mind Chicago: University of Chicago Press

Clementz, B.A et al (2016) Identification of distinct psychosis biotypes using brain-based biomarkers American Journal of Psychiatry 173, 4, 373-384

Dhindsa, R.S & Goldstein, D.B (2016) From genetics to physiology at last Nature 530, 7589, 162-163

Greenhill, S.D et al (2015) Adult cortical plasticity depends on an early post-natal critical period Science 349, 424-429

Insel, T.R (2016) Is there a better way to diagnose psychosis? Scientific American Mind March/April, 20-21

Lane, J (2016) Big data and anthropology: Concerns for data collection in a new research context Journal of the Anthropological Society of Oxford 8, 1, 74-88

Levin, N (2014) Multivariate statistics and the enactment of metabolic complexity Social Studies of Science 44, 4, 555-578

Mei, Y et al (2016) Adult restoration of Shank3 expression rescues selective autistic-like phenotypes Nature 530, 481-484

Morris, R.C (2007) Legacies of Derrida: Anthropology Annual Review of Anthropology 36, 355-389

Myers, N (2008) Molecular embodiments and the body-work of modelling in protein crystallography Social Studies of Science 38, 2, 163-199

Narasimhan, V.M et al (2016) Health and population affects of rare gene knockouts in adult humans with related parents Science 352, 474-477

Pentland, A (2014) Social Physics: How Good Ideas Spread - The Lessons from a New Science London: Scribe

Plenge, R.M (2017) Human genes lost and their functions found Nature 544, 171-172

Ripke, S et al (2014) Biological insights into 108 schizophrenia-associated genetic loci Nature 511, 421-427

Rose, N (2013) The human sciences in a biological age Theory, Culture and Society 30, 1, 3-34

Saleheen, D et al (2017) Human knockouts and phenotypic analysis in a cohort with a high rate of consanguinity Nature 544, 235-239

Sarewitz, D (2016) Saving science The New Atlantis 49, 4-40

Scudelleri, M (2017) Buzzwords and big dreams Nature 541, 450-453

Sekar, A et al (2016) Schizophrenia risk from complex variation of complement component 4 Nature 530, 7589, 177-183

Siegel, E (2013) Predictive Analytics: The Power to Predict Who Will Click, Buy, Lie, or Die Hoboken, NJ: Wiley & Sons

Sulem, P et al (2015) Identification of a large set of rare complete human knockouts Nature Genetics 47, 488-452

Terasaki, L.S et al (2016) An examination of sex differences in the effects of early-life opiate and alcohol exposure Philosophical Transactions of the Royal Society B 371, 20150123

Xu, Y et al (2016) Potential involvement of the interleukin-18 pathway in schizophrenia Journal of Psychiatric Research 74, 10-16

2. CAT OWNERSHIP AND PSYCHOSIS

There have been some studies which suggest the protozoan parasite Toxoplasma gondii (TG), carried by house cats, is linked to human psychosis. For example, individuals with schizophrenia were nearly three times more likely to have anti-bodies to this parasite in their blood than the general population in a 38-study meta-analysis (Torrey et al 2012). These anti-bodies are a sign of the presence of TG in the blood at some point. The mechanism of effect is proposed through the parasite impacting early brain development (Solmi et al 2017).

If there is a causal relationship between TG infection and later psychosis, then this will be seen in increased psychosis associated with cat ownership. The evidence is equivocal. Some studies found an association, while others did not (Solmi et al 2017), and Kapperud et al (1996) saw the link as handling soiled cat litter (as TG resides in cat faeces), particularly in pregnancy.

In the most recent study, Solmi et al (2017) used data from the Avon Longitudinal Study of Parents and Children (ALSPAC) for pre-natal and childhood cat ownership, and adolescent psychotic experiences (PEs). The ALSPAC began in 1991-2 with over 16 000 pregnant women in southwest England. By age 18 years, over 4500 offspring were still involved.

Pet ownership was self-reported by the mothers at the child's age of 8, 21, 31, and 47 months old, and PEs were based on information on unusual experiences (eg: depersonalisation) and perceptual abnormalities (eg: hallucinations) from clinic visits at 13 and 18 years old.

Cat ownership was not significantly associated with adolescent PEs after controlling for other variables.

Compared to past studies that found a relationship, Solmi et al (2017) had a number of strengths, including:

- Larger sample.
- The use of reports of PEs rather than formal diagnosis of schizophrenia.
- Cat ownership in the early years was used rather than the whole of childhood.
- Adjustment in the statistical analysis for several common potential confounders (eg: ethnicity, parental age, social class). Furthermore, the researchers stated: "We also adjusted for number of house moves in light of evidence of an association between residential mobility and PEs..., crowding index and housing type as

a proxies for both social class and greater possibility of contact with T. gondii-contaminated litter, and dog ownership as a possible confounder of the association between T. gondii infection (given an increased likelihood to contaminated soils outdoors) and psychosis risk" (Solmi et al 2017 p1665).

- Use of questionnaires asking about current cat ownership rather than depending on retrospective recall.

- The method used was a longitudinal study, whereas case control studies have been used elsewhere, and selection bias is a risk here (Solmi et al 2017).

The researchers commented: "Previous reports of positive associations between cat ownership and schizophrenia may therefore have been attributable to Type I error [7], particularly given the small sample sizes and lack of control for confounders inherent to some studies" (Solmi et al 2017 p1665).

REFERENCES

Kapperud, G et al (1996) Risk factors for Toxoplasma gondii infection in pregnancy: Results of a prospective case-control study in Norway American Journal of Epidemiology 144, 405-412

Solmi, F et al (2017) Curiosity killed the cat: No evidence of an association between cat ownership and psychotic symptoms at ages 13 and 18 years in a UK general population cohort Psychological Medicine 47, 9, 1659-1667

Torrey, E.F et al (2012) Toxoplasma gondii and other risk factors for schizophrenia: An update Schizophrenia Bulletin 38, 642-647

[7] This is where a finding is presented as statistically significant when it is not. This can happen, for instance, with a small sample and certain statistical tests.

3. ANTI-PSYCHOTICS AND DIABETES

Foley et al (2015) analysed data from the Australian National Survey of Psychosis, which is a population-based cross-sectional study of 18-64 year-olds at seven sites in five states. The study concentrated on 1155 individuals with psychosis, of whom 140 had type 2 diabetes. Current type 2 diabetes was diagnosed from current fasting blood glucose reading or use of anti-diabetic medication. Anti-psychotic medication use was calculated for the previous month based on inspection of pill bottles or medication charts in hospital.

After adjusting for other variables, individuals aged 35-64 years old taking any anti-psychotic medication were over four times more likely to have type 2 diabetes if they had no family history of diabetes ($p = 0.04$). The relationship was not significant with a family history of diabetes. Aripiprazole was the highest risk with olanzapine the lowest (which is contrary to other studies of the these two anti-psychotics; Rico-Villademoros and Calandre 2016).

Rico-Villademoros and Calandre (2016) suggested two biases that might explain the findings:

a) Survival or prevalence-incidence bias - Individuals showing side-effects are more likely to stop taking a drug, and these individuals might be classed as not taking the drug in a study.

"This form of survival bias could have reduced the difference in the diabetes prevalence between those who were taking anti-psychotics upon entering the study and those who were not, especially in patients at increased risk of developing metabolic side-effects (eg: those with a family history of diabetes)" (Rico-Villademoros and Calandre 2016 p104).

Foley and MacKinnon (2016) pointed out, in their defence, that practically a comparison group of currently not taking anti-psychotics will include past users because it is difficult to find "a large group of people with psychosis... who have never been treated with anti-psychotic drugs (a group that, to our knowledge, does not exist", and "Rico-Villademoros and Calandre do not suggest a more appropriate comparison group than those not currently taking anti-psychotic drugs. Use of another antipsychotic drug as a reference group does not change the effect of past drug use on present status, and use of another drug as the reference group provides only a relative comparison that is arguably more difficult to interpret" (Foley and MacKinnon 2016 p104).

b) Confounding by indication - Doctors may have prescribed different anti-psychotics to patients based on

risk of diabetes and perceived risk of each type of drug.

"Therefore, for patients with a raised risk of diabetes (eg: those who are overweight or obese, or those with a family history of diabetes), physicians might be more prone to prescribe an anti-psychotic with a low likelihood of causing diabetes (eg: aripiprazole) and avoid one associated with a substantial risk of this metabolic side-effect (eg: olanzapine), reducing the occurrence of diabetes in this high-risk sub-population. This bias could also have attenuated the association between olanzapine and diabetes and amplified the association between aripiprazole and diabetes" (Rico-Villademoros and Calandre 2016 p104).

REFERENCES

Foley, D.L & MacKinnon, A (2016) Authors' reply Lancet Psychiatry 3, 104-105

Foley, D.L et al (2015) Effect of age, family history of diabetes, and anti-psychotic drug treatment on risk of diabetes in people with psychosis: A population-based cross-sectional study Lancet Psychiatry 2, 1092-1098

Rico-Villademoros, F & Calandre, E.P (2016) Risk of bias in observational studies of interventions: The case of anti-psychotic-induced diabetes Lancet Psychiatry 3, 103-104

4. RECENT IDEAS ON AUTISM

4.1. INTRODUCTION

Autism spectrum disorders (ASD) covers a number of early-onset neurological conditions with social interactional problems (appendix 4A), and restricted and repetitive behaviours and focus, and the prevalence is about 1.5% of the population (Howsmon et al 2017).

Diagnosis is usually by observation of behaviours and psychometric tests by a multi-disciplinary team. Children are rarely diagnosed under two years old with these methods. A biological test based on an accepted biomarker would be very useful (Howsmon et al 2017).

Abnormalities in folate-dependent one-carbon metabolism (FCOM) and trans-sulfuration (TS) pathways, which are involved in triggering the genes related to brain development, is one area of exploration for a biomarker, as studies have shown differences between children with ASD and not (eg: Melnyk et al 2012).

Howsmon et al (2017) analysed data from the Arkansas Children's Hospital Research Institute's autism IMAGE study (Melnyk et al 2012) with 3-10 year-olds, including 83 ASD cases, 47 of their siblings, and 76 age-matched healthy controls. Seven metabolites of FCOM/TS were measured in the blood. Based on these measures, it was possible to correctly distinguish 98% of the ASD participants and 96% of controls as neurotypical (Howsmon et al 2017).

4.2. PSYCHIATRIC CO-MORBIDITY

Adults with ASD experience frequent co-morbidity of psychiatric disorders, particularly social anxiety disorder (eg: over half of ASD individuals; Spain et al 2016).

Spain et al's (2016) sample was adult males with ASD without low IQ living in south-east England, who were part of the Autism Imaging case-control Multi-site Study (AIMS) (Ecker et al 2012). Social anxiety was measured primarily by the Liebowitz Social Anxiety Scale-SR (LSAS-SR) (Liebowitz 1987), which has twenty-four items covering common social situations (eg: eating in pubic places; meeting strangers), each scored on a six-point scale. With a maximum score of 144, any score over 60 is

classed as social anxiety. Three other measures of social anxiety were also used.

The mean LSAS-SR score for the forty-six participants was 67 (range 9-124), while 65% of individuals with autism scored above sixty and 52% with Asperger's syndrome. These rates compare with 7-12% of neurotypical adults (Spain et al 2016).

In other studies with ASD individuals using the LSAS-SR, Bejerot et al (2014), for example, found that the mean score was 78, but only 28% of fifty participants scored higher than the cut-off.

4.3. APPENDIX 4A - SOCIAL INTERACTION DIFFICULTIES

Problems with social interactions in ASD are seen as related to social cognition deficits (eg: lack of theory of mind; inability to recognise emotions in facial expressions).

Though individuals with ASD appear not to be able to recognise emotions in other people's facial expressions, recent research suggests that it may be possible in the voice - ie: a difference between visual (or facial) emotion recognition (VER) and auditory (or voice) emotion recognition (AER).

One such study is by Tobe et al (2016). Nineteen individuals with high-functioning ASD, and 92 inpatients and outpatients with schizophrenia spectrum disorders at New York psychiatric institutions, and seventy-three matched healthy controls were tested. AER was measured by recordings of neutral sentences read with one of four emotions - anger, fear, happiness, or sadness - or no emotion. VER was tested with photographs of adult faces showing either happiness, sadness, fear, or anger, or a neutral expression.

In terms of group differences, the ASD individuals were poorer than controls at VER, but similar on AER. The schizophrenia group were poor on both VER and AER.

In a Japanese study, Lin et al (2016) found that twelve high-functioning ASD adults, like twelve matched IQ neurotypical (NT) controls, were faster to recognise the sound of human voices than the sound of string instruments, and, in fact, their reaction time (RT) was quicker (figure 4.1).

Participants were presented with auditory stimuli that included voices (a male singing vowels), strings (eg: cello), "chimeras" (the temporal features of a voice with the spatial features of strings, and vice versa), or a distractor (eg: trumpet).

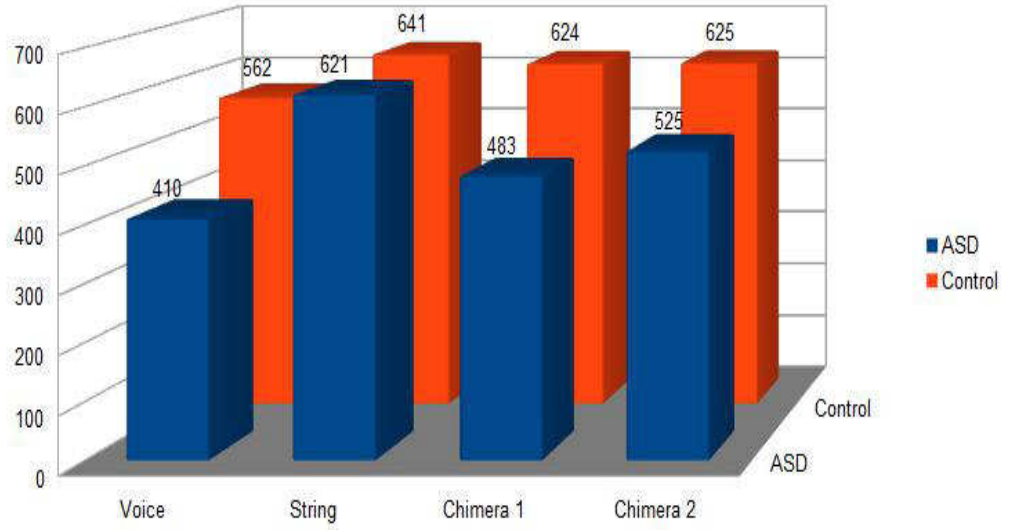


Figure 4.1 - Reaction time to different auditory stimuli (milliseconds).

The traditional view is that ASD children do not naturally attend to human voices as neurotypicals, and neuroimaging studies have found under-connectivity and/or less activity in voice-selective cortical areas when listening to voice sounds (Lin et al 2016).

Lin et al (2016) tried to explain their findings thus. The participants were told to pay attention to the auditory stimuli. In this situation, ASD individuals have low-level perceptual representation of voices (eg: is the sound a voice or not), which is similar to controls, but lack a high-level analysis, which extracts information about the mental state of the speaker as NT individuals do.

Interestingly, the ASD group were faster at recognition of the chimera stimuli. Lin et al (2016) stated: "This could be because they processed the sounds in an analytical manner, relying independently on the different acoustic cues to a voice. While such analytical listening may have favoured them in our RT task with chimeras, as they may have been oblivious to the conflicting cues present in the sounds, it could also be detrimental when listening to natural speech. Consistent with this speculation, it has been shown that when ASD individuals listen to complex sounds, their neural activity in primary auditory cortex is larger than those for NT individuals but their neural activity in non-primary auditory cortex is lower than those for NT individuals" (p6).

Both studies involved high-functioning individuals with ASD, small samples, and laboratory-based tasks

(Pycha 2016).

4.3.1. Parent-Mediated Training

Parent-mediated interventions with young children with ASD are behavioural programmes (eg: communication-focused) centred around the parents (ie: non-specialists). For example, the Pre-School Autism Communication Trial (PACT) (Green et al 2010) in the UK reported improvements in the children's communication when parent communication was adapted to the child.

Rahman et al (2016) reported a similar intervention called the parent-mediated intervention for autism spectrum disorder in south Asia (PASS) in Goa, India, and Rawalpindi, Pakistan. Parents of 2-9 year-old children with ASD were trained in 12 hone-hour sessions to adapt their communication style to the child's need (eg: reduce over-directive parental responses). Parent-child interaction in a naturalistic play situation was scored as the outcome measure eight months later.

There was evidence of improvements in the child's social interaction (eg: initiation of communication) in the PASS group as compared to a treatment-as-usual group. However, the study only involved sixty children (half in the PASS group).

Though PASS was based on PACT in the UK, there were differences in the findings between the two studies. Srinath and Jacobs (2016) suggested some possible reasons for the difference, including cultural beliefs: "Parental belief systems in Asian cultures might view autism as a punishment for previous sins, or a result of the mother's negligence of the child, or they might believe that complete recovery is possible with intervention" (p94).

4.4. REFERENCES

Bejerot, S et al (2014) Social anxiety in adult autism spectrum disorder Psychiatry Research 220, 1, 705-707

Ecker, C et al (2012) Brain anatomy and its relationship to behaviour in adults with autism spectrum disorder Archives of General Psychiatry 69, 2, 195-209

Green, J et al (2010) Parent-mediated communication-focused treatment in children with autism (PACT): A randomised controlled trial Lancet 375, 2152-2160

Howsmon, D.P et al (2017) Classification and adaptive behaviour prediction of children with autism spectrum disorder based upon multi-variate data analysis of markers of oxidative stress and DNA methylation PLoS Computational Biology 13, 3, e1005385 (Freely available at http://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1005385)

Liebowitz, M.R (1987) Social phobia Modern Problems of Pharmacopsychiatry 22, 141-173

Lin, I-F et al (2016) Fast response to human voices in autism Scientific Reports 6: 26336

Melnyk, S et al (2012) Metabolic imbalance associated with methylation dysregulation and oxidative damage in children with autism Journal of Autism and Developmental Disorders 42, 3, 367-377

Pycha, A (2016) An auditory component to autism Scientific American September, p13

Rahman, A et al (2016) Effectiveness of parent-mediated intervention for children with autism spectrum disorder in south Asia in India and Pakistan (PASS): A randomised controlled trial Lancet Psychiatry 3, 128-136

Spain, D et al (2016) Social anxiety in adult males with autism spectrum disorders Research in Autism Spectrum Disorders 32, 13-23

Srinath, S & Jacob, P (2016) Challenges in parent-mediated training in autism spectrum disorder Lancet Psychiatry 3, 93-94

Tobe, R.H et al (2016) Differential profiles in auditory social cognition deficits between adults with autism and schizophrenia spectrum disorder: A preliminary analysis Journal of Psychiatric Research 79, 21-27

5. GAMBLING, IMPULSIVITY, AND RISK-TAKING

5.1. AMERICAN INDIAN AND ALASKA NATIVE POPULATIONS

In the USA, rates of disordered gambling are higher among American-Indian/Alaska-Native (AI/AN) individuals than the general population - eg: in a general review, a problem gabling prevalence of 6-19% (2-5 times higher), and pathological gambling 7-22% (4-16 times higher) (Wardman et al 2001).

Specifically, in a study in New Mexico, pathological gambling was prevalent in 2.2% of AI individuals and 0.9% of non-AI residents in a sample of over 3000 people (Volberg and Bernhard 2006 quoted in Kong et al 2016).

Concentrating on military veterans, Westermeyer et al (2005) found pathological gambling among 10% of AI individuals compared to 4% of Hispanic Americans.

Disordered gambling can be co-morbid with other psychiatric problems, and AI/AN individuals may also suffer higher rates here. For example, 22% of AI/AN adults being treated for alcohol dependence were diagnosed with pathological gambling compared to 7% of non-AI/AN individuals (Elia and Jacobs 1993).

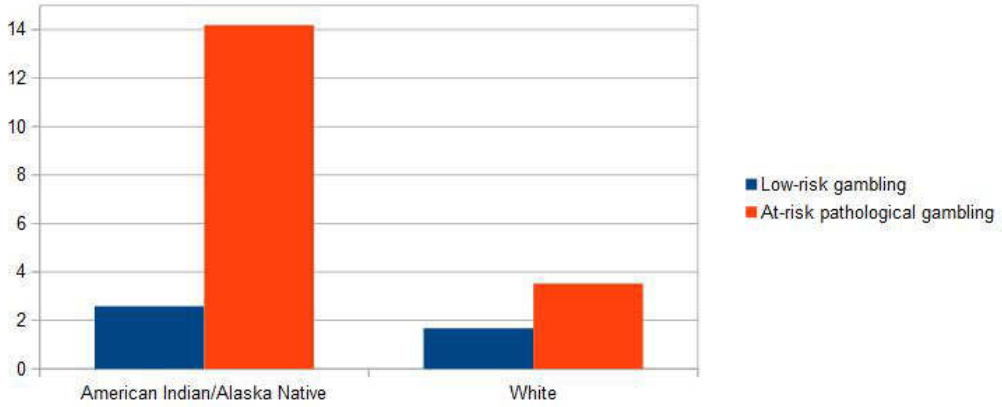
Kong et al (2016) confirmed this finding in a recent large-scale study. These researchers analysed data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) in 2001-2, which covered a nationally representative sample of over 40 000 US adults.

Problem gambling was classified into three groups based on ten questions - non-gambling/low frequency gambling (NG) (< 5 times per year), low-risk gambling (LRG) (> 5 times per year, and 0-2 symptoms of pathological gambling), and at-risk pathological gambling (ARPG) (3 or more symptoms of pathological gambling). Trained lay interviewers diagnosed other psychiatric disorders in formal interviews.

There were significant differences between AI/AN respondents and the general population in the three types of gambler, particularly LRG - 30% of AI/AN vs 27% (and 3.4% and 3.0% respectively for ARPG).

Co-morbidity of psychiatric disorders and LRG/ARPG

categories were found for all individuals, but the relationships were stronger for the AI/AN group. The odds ratio was fourteen times greater for "any psychiatric disorder" between ARPG and LG AI/AN individuals compared to three times greater among White respondents (figure 5.1).



(Data from Kong et al 2016 table 3 p60)

Figure 5.1 - Odds ratio for any psychiatric disorder based on gambling group (where no-gambling/low frequency gambling = 1).

Note that the researchers treated AI/AN respondents as a homogeneous group for convenience, and ignored "varying tribal affiliations with different cultural norms" (Kong et al 2016). Also the NESARC did not distinguish whether the AI/AN individuals lived on or off reservation, and living on a reservation has been linked to pathological gambling (Kong et al 2016).

Despite the large overall sample, the AI/AN sub-sample was quite small with 679 individuals (and only 28 classed as ARPG).

In terms of explaining the higher rate of disordered gambling among AI/AN individuals, Kong et al (2016) stated that it is "not inherently attributable to 'race' per se, but reflects a consequence of pervasive and systematic exposure to poverty, racism and discrimination, and historical and social trauma that members of this group have experienced through generations... Historical trauma includes multi-generational forced assimilation, such as removal from native lands, coerced placement of children into boarding schools, and laws prohibiting indigenous practices... These traumatic experiences may have both specific and cumulative effect on the mental health of AI/AN individuals" (p56).

Contemporary AI/AN communities often live alongside casinos, which are privileged on native lands by US law as a means to encourage economic growth among disadvantaged groups. "However, despite the opportunity for positive economic growth and social benefits, the outcome is controvertible. Economic analyses show that casinos on reservations increase employment and health benefits and decrease poverty rates; however, the increase in economic growth appears to be driven by non-AI/AN employment" (Kong et al 2016 p56).

5.2. SOCIAL CASINO GAMES AND PROBLEM GAMBLING

Social media gaming involves playing games via social networking sites. There are a variety of types of games, but social casino games (non-monetary gambling-style games) have "many similarities with traditional forms of online gambling activities" (Derevensky and Gainsbury 2016). Because there are no monetary prizes, such games avoid gambling regulations, though "freemium" versions encourage players to purchase additional elements to improve the game experience [8], and non-cash promotional prizes are offered to continue playing [9] (Derevensky and Gainsbury 2016) [10].

Free casino games, for example, (like poker) are offered as "practice" or "instructional" sites. "The extent to which young people are able to distinguish between social casino games, practice games and gambling has not been investigated" (Derevensky and Gainsbury 2016 p3). Nor the migration of players from social casino games to gambling in detail. "Although social casino games mimic gambling in many ways, players' expenditures and motivations may be significantly different. In free-play games, players reportedly focus on the competitive aspects, in contrast to 'beating the house'. Satisfaction from winning in this context appears to eliminate the need to win actual money" (Derevensky and Gainsbury 2016 p3).

---

[8] For example, "Runescape" involves the opportunity to "purchase spins" of a virtual wheel for prizes. "Such games may be promoting a perceived illusion of control by incorporating exaggerated odds of winning while [other games are] promoting favourable views of slot machines as a harmless entertainment activity" (Derevensky and Gainsbury 2016 p3).

[9] For example, "Jetpack Joyride" where primarily children are encouraged to fly Barry through bubbles and rainbows, and dodge hurdles. "Although not the core objective, players can acquire 'spin tokens' allowing them to play a slot machine where acquired rewards are used within the game. However, the slot machine does not appear randomly, rather it recognizes when users are likely leaving the game (eg: following a series of losses) and provides incentives to continue playing" (Derevensky and Gainsbury 2016 p3).

[10] Two developments in gambling availability are seen as key to problem gambling generally - the appearance of fixed odds betting terminals (eg: roulette machines) on the high street, and online sports betting (Gentleman 2016).

Early onset of gambling behaviour is a risk factor for later problem gambling (eg: Australian Internet gamblers; Gainsbury et al 2012 quoted in Derevensky and Gainsbury 2016).

Increased perceived illusion of control from social casino gaming could encourage gambling. For example, Sevigny et al (2005) reported greater payout rates on "practice" sites as compared to the "real" ones, with some former sites having a payout rate of 100%.

A survey of 11-16 year-olds in England and Wales found that around 15% had played social casino games in the past week, and half of them reported gambling for money (compared to 18% of non-players) (Ipso MORI 2011) [11] [12].

Gupta et al's (2013 quoted in Derevensky and Gainsbury 2016) study with university students found a general progression from social games to social casino games, to online gambling. "Facebook" was described as a "poker training ground" by some respondents (Derevensky and Gainsbury 2016).

King et al (2010) summarised the characteristics of social casino games that promote online gambling (Derevensky and Gainsbury 2016):

- Readily accessible and attractive;
- Promotes factually incorrect information about gambling (eg: likelihood of winning);
- Easy escape from life's problems;
- Encourages peer pressure to gamble;
- Easy transfer of parental attitudes towards gambling; "ultimately makes gambling more ubiquitous and socially acceptable" (Derevensky and Gainsbury 2016 p4).

However, Derevensky and Gainsbury (2016) admitted: "What still remains unclear is whether a causal relationship exists between playing simulated gambling games, actual gambling for money, and problem gambling" (p4).

"Pathological gambling" was added to official diagnostic categories by the American Psychiatric Association in 1980, and by DSM-5 in 2013 this had become

[11] Referrals to the National Problem Gambling clinic in London, for problem gambling generally, are mostly males, in their 30s and 40s, and working (though they have often subsequently lost their job through gambling) (Gentleman 2016).

[12] Establishing if problem gambling has increased is not easy. For example, the National Gambling Helpline reported an increase in calls by 18% in 2014-5 (Gentleman 2016), but this only measures help-seekers to that organisation.

"gambling disorder" (Fisher 2016).

The idea that behaviours can be addictive in the same way as substances has been argued over. "The grey area between clear disorders and unhealthy habits is rightfully controversial" (Fisher 2016 p48). Marks (1990) is reputed as the first to formally advocate behavioural addictions.

Potenza et al (2003), for example, found that individuals with gambling problems showed physiological signs in the form of decreased activity in the ventro-medial prefrontal cortex in fMRI scans. This area is associated with regulating impulses, and individuals with obsessive-compulsive disorder (OCD) show increased activity there (Fisher 2016).

Blaszczynski and Nower (eg: 2002) proposed three sub-groups of gambling addicts:

- Behaviourally conditioned gamblers (who get into the habit of chasing wins);
- Emotional vulnerable gamblers (who gamble to cope with anxiety and depression);
- Anti-social gamblers (who are highly impulsive in all areas of their lives) (Fisher 2016).

Concentrating on the middle group, gamblers who expected to gain relief from negative emotions or to maintain positive emotions were more likely to have gambling problems than gamblers who did not have these expectations (Quilty et al 2017).

The link between mood and gambling has been studied in different ways:

1. Laboratory experiments (eg: Hills et al 2001)

Sixty regular and sixty non-regular gamblers experienced a mood induction event (happy, sad, or neutral) before the opportunity to gamble. The induction of a sad mood reduced the gambling behaviour in non-regular gamblers compared to the other two mood conditions, whereas regular gamblers were not influenced by the mood condition.

Quilty et al (2017) highlighted the low ecological validity of this study.

2. Interviews/surveys (eg: Nower and Blaszczynski 2010)

The motivations of electronic gaming machine gamblers were self-reported most commonly as to avoid

problems, to increase excitement, and to make money by problem gamblers, and to have fun and enjoyment by non-problem gamblers.

This study, like many, "relied upon retrospective self-report measures, which assumes that individuals accurately recall and report their affective state, their reasons for gambling, and their level of gambling desire prior to past gambling activities" (Quilty et al 2017 p117).

3. Time sampling (eg: Gee et al 2005)

Regular gamblers rated their anxiety level before, during, and after a gaming episode via a mobile phone over a 7-14 day period. Participants were significantly more anxiety when they experienced a desire to gamble, and during and after a gambling activity than at baseline.

5.3. SCRATCH CARDS

"Scratch cards" (SCs), where gamblers uncover symbols, letters or numbers for an instant prize, are popular and appealing (Strange et al 2017).

They have been likened to slot machines, especially with small, unpredictable wins. Such wins are rewarded by the post-reinforcement pause (PRP). "Unlike losing spins, where gamblers tend to spin again immediately, following a win, gamblers pause before triggering the next spin, as though to internally celebrate the win. The length of such pauses varies directly with win size, and has been used to infer the different rewarding properties of these outcomes" (Strange et al 2017 pp48-49).

Strange et al (2016) observed gamblers playing custom-made SCs, and found evidence of the PRP.

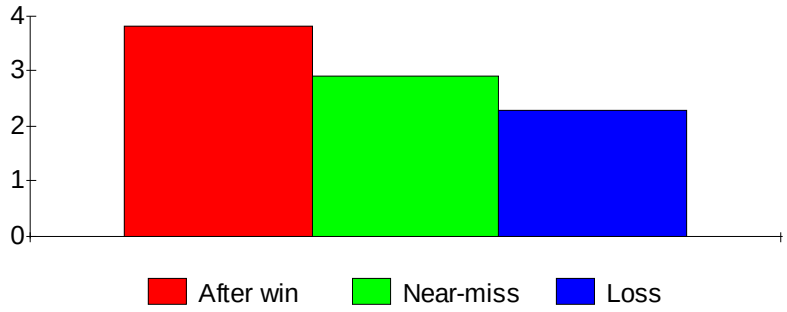
Another common characteristic between slot machines and SCs is the "near-miss", where the gambler is close to a winning combination. For example, when three "6s" are needed to win, and the gambler has "665". This is a loss, but psychologically it feels different to the gambler compared to "661", say. Near-misses have been found to arouse gamblers (due to frustration) more than small wins, and to increase the desire to continue playing (gambling urge) (Strange et al 2017). With their custom-made SCs, Strange et al (2016) found that near-misses produced arousal as measured by self-reports, and by skin conductance.

Strange et al (2017) developed this work in two similar experiments.

Experiment 1

Sixty-three undergraduates at a university in Canada were presented with custom-made SCs to play, while being video recorded, and their skin conductance and heart rate measured. The SCs involved matching three symbols, and each card contained nine symbols, and were designed as two types - loss/small win/loss, and loss/near-miss/loss.

Physiological measures of arousal were higher after small wins and near-misses than after losses, and subjective measures like gambling urge showed a similar pattern. PRP was significantly longest after a win (figure 5.2).

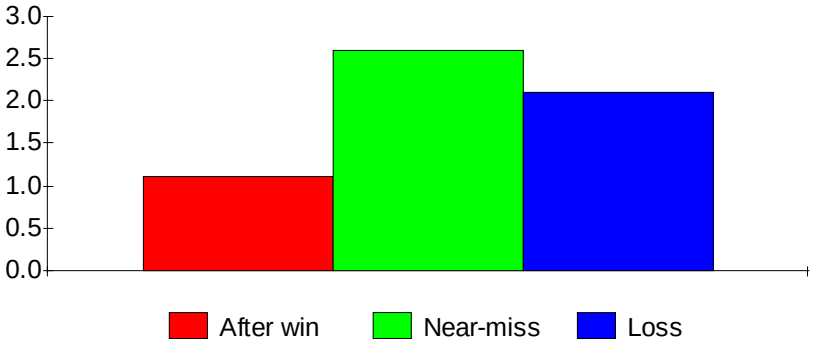


(Data from Strange et al 2017 table 1 p55)

Figure 5.2 - Mean PRP (seconds) in Experiment 1.

Experiment 2

Sixty-eight more undergraduates completed a similar experiment to above, but a subjective measure of disappointment was added. The findings were mostly similar to Experiment 1, and the disappointment was significantly highest after a near-miss, followed by after a loss (figure 5.3).



(Data from Strange et al 2017 table 2 p58)

Figure 5.3 - Mean rating of disappointment (where a higher score = greater disappointment) in Experiment 2.

Overall, these experiments replicated the findings of Strange et al (2016), and showed the similarity between SCs and slot machines. The most important finding for the researchers was the urge to gamble after a small win, and after a near-miss.

5.4. MOOD DISORDERS AND GAMBLING

There is evidence of an association between mood disorders and gambling behaviour. For example, individuals with gambling problems have mood disorders more than the general population, and individuals with mood disorders have gambling problems more often than in the general population (Quilty et al 2017).

"The nature of the association between mood disorders and problem gambling, however, remains unclear" (Quilty et al 2017 p116). For example, gambling problems develop as a means to relieve negative mood, or depression is a consequence of the negative effects of pathological gambling (Quilty et al 2017).

Quilty et al (2017) used a time sampling method to study moods, the desire to gamble, and gambling behaviour among thirty problem gamblers with depressive disorder or bipolar disorder in Canada. Participants were randomly prompted to respond to an electronic personal digital assistant (PDA) three times per day for thirty days.

At each prompt, participants completed the same ten-question survey covering current location and social context, mood, desire to gamble, most recent gambling activity, and motivation for gambling.

It was found that mood did not predict gambling behaviour, but it did predict desire to gamble, particularly sadness and arousal, and desire to gamble predicted actual gambling behaviour. Individuals with bipolar disorder were more likely to gamble to cope with negative mood, while participants with depression reported the motivation to gamble to improve or maintain a positive mood (as well as for social reasons).

It should be noted that half of the participants were in partial or full remission from their mood disorder, and individuals with severe mania or psychosis were excluded from the study.

5.5. ALCOHOL AND GAMBLING PROBLEMS

Tackett et al (2017) pointed out that "both problematic gambling and problematic alcohol use are fairly common problems, particularly among college student populations" (p132). For example, pathological gambling is estimated in 5% of college students in the USA compared to up to 3.5% of the general population, and

problematic drinking as twice the general population. Problem gamblers are three times more likely to have alcohol use disorders than non-gamblers, for instance (Tackett et al 2017).

Tackett et al (2017) investigated the common factors in problematic gambling and problematic alcohol use in a survey with nearly 5000 undergraduate students at a large northwestern US university. The survey included items from the following established questionnaires:

- South Oaks Gambling Screen (SOGS) (Lesieur and Blume 1987) - measures type and frequency of gambling, family history of gambling, and largest quantity of money lost.

- Gambling Motives Questionnaire (GMQ) (Neighbors et al 2002) - 48 items covering sixteen different gambling motivations.

- Alcohol Use Disorder Identification Test (AUDIT) (Saunders et al 1993) - ten items: eg: "How often during the last year have you found that you were not able to stop drinking once you had started?".

- Drinking Motives Questionnaire (DMQ) (Cooper 1994) - covers four types of drinking motivation (conformity, coping, enhancement, and social).

- Zuckerberg-Kuhlman Personality Questionnaire (ZKPQ) (Zuckerman et al 1993) - 99 items: eg: "I tend to start conversations at parties".

- Brief Symptom Inventory (BSI) (Derogatis and Melisaratos 1983) - 53 items covering nine mental health dimensions.

The responses to the different questionnaires were analysed for patterns, and 264 responses were examined in detail. A bifactor model was found (ie: the two behaviours of problematic gambling and alcohol use have an underlying common factor), but there were gender differences. For men, risk-taking was a common factor and with the importance of the social aspect of both behaviours, but this was not found for women. For example, heavy drinking men were more likely to gamble, but heavy drinking women were less likely to gamble. Thus, "these findings suggest that gambling and drinking are more strongly connected among men than women" (Tackett et al 2017 p144).

5.6. GAMBLING ADVERTISING

Gambling advertisements are increasing - eg: 234 000 on television in the UK in 2007 to 1.4 million in 2012 (Clemens et al 2017). Such advertising is "usually justified by suggesting that advertising does not affect the actual size of the market, but only the size of the shares of different companies in a fixed market. However, this statement is based on theoretical assumptions, not on empirical evidence" (Clemens et al 2017 p2).

The consequences are particularly important in relation to younger viewers. For example, Felsher et al (2004) found that of adolescents who recalled seeing advertisements for lottery tickets, two-fifths believed that the advertisements would motivate them to buy lottery tickets. While Lee et al (2008) reported that increased exposure to poker advertisements was associated with more positive attitudes towards gambling advertising, and such attitudes were also associated with stronger intentions to gamble in the future.

But these two examples of studies did not involve actual gambling behaviour. Clemens et al (2017) rectified this problem in their study in Germany. Over 4500 13-25 year-olds in two regions were surveyed about their gambling behaviour (lifetime, last 12 months and last week), self-rated exposure to 32 gambling advertisements (0 = never - 3 = more than 10 times), and other relevant variables (eg: average screen time).

Two-thirds of the sample, overall, had gambled at least once in their lifetime, 7% in the last week ("current gambling"), and 3% were classified as probable pathological gamblers. Gambling advertisement exposure was strongly associated with gambling behaviours. For example, current gamblers reported seeing 3.5 times more advertisements than "never gambled" individuals.

The researchers accepted the following key limitations to their study:

i) A cross-sectional design which measures behaviour once, so establishing cause and effect was not possible.

ii) Only selected gambling advertisements were used.

iii) Despite controlling for many variables, "the results may be biased by unmeasured confounding" (Clemens et al 2017).

iv) The data were self-reported.

5.7. ONLINE IMPULSIVITY

"Impulsive technology-mediated behaviours" (Aboujaoude and Starcevic 2016) or "online impulsivity" was furst described as the "online disinhibition effect" by Suler (2004). The online world has anonymity, invisibility, and "the absence of the offline trappings that communicate the power and hierarchy that help contain offline behaviour (eg: a police officer's uniform, a parent's or teacher's age)" (Aboujaoude and Starcevic 2016 p1014), and so the normal restraints on impulses are removed.

Online impulsivity is not in DSM-5, but "internet gaming disorder" is included as a condition for further study. Online impulsivity, though, is involved in this disorder, as well as in compulsive buying on the Internet, and "internet sexual problems". The prevalence of compulsive online buying has been reported at 16-17% (eg: students in Paris; Duroy et al 2014) (compared to 2-6% for non-internet buying; eg: USA; Koran et al 2014), while "internet sexual problems" are reported by 18% of men and 7% of women in a Swedish study (Ross et al 2012) (compared to 3-6% for compulsive sexual behaviour offlinein the general adult population) (Aboujaoude and Starcevic 2016).

Aboujaoude and Starcevic (2016) concluded: "It would seem as though, by facilitating access to opportunity (eg: to gamble, buy, meet sexual partners) or information, technology can make it more difficult to resist impulses and easier to pass to the act" (p1015).

5.8. DOPAMINE AND RISK-TAKING

Frank et al (2007) linked the ability to avoid losses (ie: risk aversion) to differences in a gene related to dopamine, while Dodd et al (2005) reported pathological gambling as a side effect of dopamine-related medications (as used in Parkinson disease).

In terms of animal studies, Zalocusky et al (2016) offered rats a choice between two levers, where one lever gave a safe intermediate-sized sugar reward, and the other a gamble between a small (75% of the time) or a large reward. Risk averse rats tended to press the safe lever, but if they gambled, certain dopamine receptors were more active after a losing gamble than a win or certain outcome. Risk-seeking rats, who chose the gamble lever more often, did not have the increased dopamine after a loss. But artificially increasing the dopamine in risk-seeking rats led to less gambles.

This study "identified specific neural signatures that predict risk preferences, and demonstrated that the activity of these neurons at specific time points contributes to animals' decisions" (Hollon and Phillips

2016 p589), but only specific dopamine neurons in particular areas of the brain (Hollon and Phillips 2016).

5.9. REFERENCES

Aboujaoude, E & Starcevic, V (2016) The rise of online impulsivity: A public health issue Lancet Psychiatry 3, 1014-1015

Blaszczynski, A & Nower, L (2002) A pathways model of problem and pathological gambling Addiction 97, 5, 487-499

Clemens, F et al (2017) Exposure to gambling advertisements and gambling behaviour in young people Journal of Gambling Studies 33, 1-13

Cooper, M.L (1994) Motivations for alcohol use among adolescents: Development and validation of a four-factor model Psychological Assessment 6, 117-128

Derevensky, J.L & Gainsbury, S.M (2016) Social casino gaming and adolescents: Should we be concerned and is regulation in sight? International Journal of Law and Psychiatry 44, 1-6

Derogatis, L.R & Melisaratos, N (1983) The brief symptom inventory: An introductory report Psychological Medicine 13, 595-605

Dodd, M.L et al (2005) Pathological gambling caused by drugs used to treat Parkinson disease Archives of Neurology 62, 9, 1377-1381

Duroy, D et al (2014) Characteristics of online compulsive buying in Parisian students Addictive Behaviour 39, 1827-1830

Elia, C & Jacobs, D (1993) The incidence of pathological gambling among Native Americans treated for alcohol dependence International Journal of Addiction 28, 659-666

Felsher, J.R et al (2004) Lottery playing amongst youth: Implications for prevention and social policy Journal of Gambling Studies 20, 127-153

Fisher, C.E (2016) Addicted to" Scientific American Mind January/February, 43-49

Frank, M.J et al (2007) Genetic triple dissociation reveals multiple roles for dopamine in reinforcement learning Proceedings of the National Academy of Sciences, USA 104, 41, 16311-16316

Gee, P et al (2005) Mood state and gambling: Using mobile telephones to track emotions British Journal of Psychology 96, 53-66

Gentleman, A (2016) The secret addiction Guardian 7/6, II 9-11

Hills, A et al (2001) Induced mood and persistence at gaming Addiction 96, 1629-1638

Hollon, N.G & Phillips, P.E.M (2016) Making risk-takers settle Nature 531, 7596, 588-589

Ipsos MORI (2011) Underage Gambling in England and Wales. A Research Study Among 11-16 Olds on Behalf of the National Lottery Commission London: National Lottery Commission

King, D et al (2010) The convergence of gambling and digital media: Implications for gambling in young people Journal of Gambling Studies 16, 175-187

Kong, G et al (2016) Problem-gambling severity and psychiatric disorders among American-Indian/Alaska native adults Journal of Psychiatric Research 74, 55-62

Koran, I.M et al (2006) Estimated prevalence of compulsive buying

behaviour in the United States American Journal of Psychiatry 163, 1806-1812

Lee, H.S et al (2008) Role of gambling media exposure in influencing trajectories among college students Journal of Gambling Studies 24, 25-37

Lesieur, H.R & Blume, S.B (1987) The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers American Journal of Psychiatry 144, 1184-1188

Marks, I (1990) Behavioural (non-chemical) addictions British Journal of Addiction 85, 11, 1389-1394

Neighbors, C et al (2002) Exploring college student gambling motivation Journal of Gambling Studies 18, 361-370

Nower, L & Blaszczynski, A (2010) Gambling motivations, money-limiting strategies, and pre-commitment preferences of problem versus non-problem gamblers Journal of Gambling Studies 26, 361-372

Potenza, M.N et al (2003) An fMRI Stroop task study of ventro-medial prefrontal cortical function in pathological gamblers American Journal of Psychiatry 160, 11, 1990-1994

Quilty, L.C et al (2017) A prospective investigation of affect, the desire to gamble, gambling motivations and gambling behaviour in the mood disorders Journal of Gambling Studies 33, 115-129

Ross, M.W et al (2012) Prevalence, severity, and correlates of problematic sexual Internet use in Swedish men and women Archives of Sexual Behaviour 41, 459-466

Sanders, J.B et al (1993) Development of the alcohol use disorders screening test (AUDIT). WHO collaborative project on early detection of persons with harmful alcohol consumption. II Addiction 88, 791-804

Sevigny, S et al (2005) Internet gambling: Misleading payout rates during the "demo" period Computers in Human Behaviour 21, 153-158

Stranger, M et al (2016) "I was that close": Investigating gamblers' reactions to losses, wins, and near-misses on scratch cards Journal of Gambling Studies 32, 1, 187-203

Strange, M et al (2017) Reinforcing small wins and frustrating near-misses: Further investigation into scratch card gambling Journal of Gambling Studies 33, 47-63

Suler, J (2004) The online disinhibition effect Cyberpsychology and Behaviour 7, 321-326

Tackett, J.L et al (2017) Co-morbidity of alcohol and gambling problems in emerging adults: A bifactor model conceptualisation Journal of Gambling Studies 33, 131-147

Wardman, D et al (2001) Problem and pathological gambling in North American aboriginal populations: A review of empirical literature Journal of Gambling Studies 17, 81-100

Westermeyer, J et al (2005) Lifetime prevalence of pathological gambling among American Indian and Hispanic American veterans American Journal of Public Health 95, 860-866

Zalocusky, K et al (2016) Nucleus accumbens D2R cells signal prior outcomes and control risky decision-making Nature 531, 7596, 642-646

Zuckerman, M et al (1993) A comparison of three structural models of personality: The Big Three, the Big Five, and the alternative Five Journal of Personality and Social Psychology 65, 757-768

6. MISCELLANEOUS SUBSTANCE MISUSE

Biology

(1) Substance use disorders generally have an estimated heritability of over 50%, with the figure being 68% in one study for stimulant use disorder (Heinzerling et al 2016). The increasing knowledge about the human genome can help in understanding which genes are involved.

For example, Heinzerling et al (2016) found a particular version of a gene was involved in methamphetamine dependence. Blood for genotyping was collected from over two hundred individuals at a Los Angeles hospital who were seeking treatment for methamphetamine problems. One version of the gene was found to be significantly associated with methamphetamine use before and during treatment in males, but not females.

(2) Changes in the brain are associated with drug addiction. In many cases, the changes are a product of the addiction (eg: enlarged putamen (dorsal striatum)). However, this enlargement has also been reported in unaffected first-degree relatives of stimulant addicts, and in individuals with obsessive-compulsive disorder, suggesting that ""putamen enlargement may partly represent a predisposing factor for compulsive behaviours" (Ersche et al 2017 p1).

Ersche et al (2017) described another change in the brain - disrupted iron regulation - which occurred in cocaine addicts due to the addiction. The study compared forty-four individuals with a chronic history of cocaine use and forty-four healthy controls using neuroimaging in the UK. The addicts had excessive iron accumulation in a particular part of the brain. There was no relationship between the iron accumulation and the enlargement of the putamen, which the researchers took as evidence for putamen enlargement as "a vulnerability factor to, rather than a consequence of cocaine addiction" (Ersche et al 2017 p6).

(3) Gender differences have been found in the patterns of substance abuse. For example, women are less likely to use substances than men, but become addicted faster (Locklear 2016).

The hormones involved in the menstrual cycle could play a role here, and in countering addiction, as shown in a recent animal study. Lacy et al (2016) allowed female rats to self-administer heroin, and measured the amount used across the oestrous cycle. It was found that heroin use was lowest when oestrogen levels were high and

progesterone levels were about to peak.

Measurement and Treatment

(4) Attempts by health professionals to reduce harm caused by substance misuse can be compromised by criminalisation of the substance. This has led health-related organisations to make recommendations about the legal position of substances - eg: the Royal Society for Public Health and Faculty of Public Health in the UK argued for decriminalisation of personal possession of "illegal" drugs (Kushlick 2016).

(5) Chemical analysis of urban wastewater for excretion products of illicit drugs is based on the assumption that traces of the drugs and/or metabolites can be detected in urine and/or faeces. It is a measure of drug use in a population (Castiglioni et al 2014).

This approach can show changes in the patterns of drug use over time, and the appearance of new chemical substances, but not the characteristics of users or the individual frequency of use (Castiglioni et al 2014).

(6) As the majority of substance users do not seek treatment, screening and brief interventions (SBIs) [13] have been proposed as an "opportunistic" solution in primary care. This involves dealing with the substance use when an individual approaches the health services for other reasons (Palfai et al 2016).

One example of a SBI is motivational interviewing (MI), which explores the individual's willingness to change their substance use and develops the self-efficacy to change (eg: "change talk") (Palfai et al 2016).

Palfai et al (2016) reported a randomised controlled trial on the skills of the providers of MI for illicit drug use and prescription drug misuse with data from the Assessing Screening Plus brief Interventions Resulting Efficacy to stop drug use (ASPIRE) study (Saltz et al 2014) in the USA. Drug use at baseline, six weeks, and six months was measured for 351 individuals.

In terms of the MI during primary care visits, the participants received the brief negotiated interview (BNI) or motivational intervention (MOTIV). The former is a 10-15-minute structured intervention from a health educator, which includes the "pros and cons" of drug use, while the MOTIV is a 30-45-minute intervention conducted

[13] Most SBIs are single session-based.

by a Master's degree level qualified provider. It is less structured. Based on recordings of the interventions, the researchers coded the intervention skills of the providers.

No relationship was found between the skills of the MI provider and lower frequency of drug use. The researchers suggested this possible interpretation of the findings - " a brief intervention may not be sufficient to change drug use no matter how competently it is delivered" (Palfai et al 2016 p12).

Medical Cannabis

(7) In the USA between 1999 and 2014, there were 165 000 opioid overdose deaths (Hsu 2016). Medical cannabis (MC) may offer an alternative to opioid painkillers. For example, Boehnke et al (2016) surveyed 244 patients at a MC dispensary in Michigan, USA, and found that many of these individuals had cut their opioid use for chronic pain by half. However, this was a retrospective study (Hsu 2016). While in an Israeli study, 44% of 176 chronic pain patients had stopped taking prescription opioids after six months of medical cannabis use (Haroutounian et al 2016).

But there are non-serious adverse effects to MC use, as shown in a study that compared cannabis and opioid users over one year, yet no differences in serious side-effects (Ware et al 2015).

REFERENCES

Boehnke, K.E et al (2016) Medical cannabis use is associated with decreased opiate medication use in a retrospective cross-sectional survey of patients with chronic pain Journal of Pain 17, 6, 739-744

Castiglioni, S et al (2014) Testing wastewater to detect illicit drugs: State of the art, potential and research needs Science of the Total Environment 487, 613-620

Ersche, K.D et al (2017) Disrupted iron regulation in the brain and periphery in cocaine addiction Translational Psychiatry 7, e1040

Haroutounian, S et al (2016) The effect of medicinal cannabis on pain and quality of life outcomes in chronic pain Clinical Journal of Pain 32, 12, 1036-1043

Heinzerling, K.G et al (2016) Single nucleotide polymorphism near CREB1, rs7591784, is associated with pre-treatment methamphetamine use frequency and outcome of outpatient treatment for methamphetamine use disorder Journal of Psychiatric Research 74, 22-29

Hsu, J (2016) Can medical cannabis break the painkiller epidemic? Scientific American September, 11-12

Kushlick, D (2016) Medical intervention New Scientist 26/11, 20-21

Lacy, R.T et al (2016) The effects of sex, oestrous cycle, and social contact on cocaine and heroin self-administration in rats Psychopharmacology 233: 3201

Locklear, M (2016) Menstrual cycle clue to addiction New Scientist October 1, p14

Palfai, T.P et al (2016) Is the quality of brief motivational interventions for drug use in primary care associated with subsequent drug use? Addictive Behaviours 56, 8-14

Saltz, R et al (2014) Screening and brief intervention for drug use in primary care: The ASPIRE randomised clinical trial Journal of the American Medical Association 312, 5, 502-513

Ware, M.A et al (2015) Cannabis for the management of pain: Assessment of safety study (COMPASS) Journal of Pain 16, 12, 1233-1242

7. MISCELLANEOUS ALCOHOLISM

7.1. INTRODUCTION

"Drug addiction is a chronically relapsing disorder characterised by compulsion to seek and take the drug, loss of control in limiting intake, and the emergence of a negative emotional state (eg: dysphoria, anxiety, irritability) when access to the drug is prevented" (Wscieklica et al 2016 p73).

Three stages of the cycle have been distinguished (Wscieklica et al 2016):

i) Binge/intoxication - dominated by positive reinforcement (ie: the reward of the "high");

ii) Withdrawal/negative affect - dominated by negative reinforcement (ie: the reward of not having the negative consequences of the "down");

iii) Preoccupation/anticipation - ie; addiction.

Alcohol use disorders (AUDs) have a lifetime prevalence of 30% in the USA, and an estimated seventeen million sufferers in a given year (O'Halloran et al 2016).

7.2. STIMULUS MATERIALS

In alcohol use disorders (AUDs), alcohol-related stimuli have been shown in experimental studies to activate addiction-related processes - behavioural, cognitive, and physiological and neurophysiological - which are different to controls (Fey et al 2017).

Finding appropriate stimulus material is important, and these include words, pictures, and videos. There are issues related to (Fey et al 2017):

a) Ecological validity - ie: represent real-life cues;

b) Standardisation of stimulus to be used - Objective stimulus characteristics relate to the physical stimulus parameters (eg: visual complexity) and subjective stimulus characteristics to the experience of

the viewer (eg: arousal);

c) Size of the database to avoid repetition.

Often pictures from advertisements or the Internet are used. Fey et al (2017) warned: "Such stimuli are insufficiently validated and are not adequately controlled with regard to their objective and subjective characteristics" (p2).

In terms of studies, Wrase et al (2002) used 32 alcohol-related pictures, eighteen non-alcohol beverage pictures, and ten neutral stimuli. Individuals with AUDs only differed from controls on the rating of the non-alcoholic beverages as more unpleasant and more arousing (Fey et al 2017).

Students are often used in the validation process, as in the Normative Appetitive Picture System (NAPS) (Stritzke et al 2004), which includes pictures of alcoholic and non-alcoholic beverages and other appetite cues, or the ninety-six-picture Amsterdam Beverage Picture Set (ABPS) (Pronk et al 2015).

Fey et al (2017) argued that pictures of non-alcoholic beverages "may unintentionally trigger associations with alcoholic drinks (ie: a picture of a Coke may evoke the cocktail whiskey and Coke)" (p3). So their database included only alcohol-related pictures (n = 444) and pictures of neutral stimuli (n = 387). Eighteen in-patients with AUDs in Switzerland and 18 healthy controls were asked a series of questions about each image, including emotion evoked (valence), strength of emotion (arousal), and urge to drink (craving).

The neutral pictures evoked significantly more craving and alcohol-relatedness in the AUDs group than the controls, and the alcohol-related pictures produced non-significantly more arousal and craving in the patients. This fits with the idea that individuals with AUDs perceive alcohol-relatedness in almost everything (ie: cognitive bias).

7.3. ANIMAL STUDIES

Animal studies allow researchers to deliberately manipulate levels of a substance, which would be ethically unacceptable with humans, and to study physiological changes in more detail (eg: via post-mortem).

One example of a study with alcohol is reported by Wscieklica et al (2016). Male rats were offered two bottles - water or sugared alcohol solution - over a 31-day period (or water or sugared water in the control condition). This is known as the two bottle paradigm.

The rats settled into a pattern of alcohol

consumption over the study period, and on the 31st day their behaviour was tested in a maze and an open field (an arena 60 cm x 60 cm), before being killed. There was no difference between the alcohol and control groups in the maze, but the alcohol group made significantly more crossings of the open field. this was taken as a sign of increased locomotor activity. Physiological changes were found in the brain areas related to reward, particularly changes in the Fos protein, in the post-mortem.

Alcohol has a biphasic effect - both stimulatory and depressant effects on behaviour, depending on the dose and the time since consumption (Karlsson and Roman 2016).

Karlsson and Roman (2016) explored the locomotor behaviour of rats in the multi-variate concentric square field (MCSF) test, which includes different environments (eg: sheltered and open; different illuminations).

A medium or large dose [14] of alcohol (or none in the control) was injected five minutes before the MCSF test. Distance moved in the arena and number of different environments visited in thirty minutes were scored. Rats receiving the highest dose of alcohol were least active overall. But rats with the medium doses were active in the first fifteen minutes of the test and less so in the second fifteen minutes compared to controls.

West et al (2016) selectively bred albino rats in their laboratory to vary in susceptibility to stress over fifty generations. After nineteen generations, it was found that easily stressed rats voluntarily consumed larger amounts of alcohol relative to stress-resistant albinos and normal rats. Differences in the locus coeruleus (LC) and linked to noradrenalin in the brain were found to be associated with the variation. The authors stated that "activity of LC neurons influence reward produced by alcohol and thereby LC affects the propensity to consume alcohol. We suggested that high levels of LC activity will decrease reward and inhibit consumption of alcohol and, conversely, low levels of LC activity will enable or facilitate reward and promote consumption of alcohol" (West et al 2016 p100).

The zebrafish (Danio rerio) has become popular to use in studies of alcoholism, particularly during pregnancy, and a number of physical consequences for the embryo have been reported (eg: enlarged body cavity, small eyes, organ failure, behavioural impairment) (Dewari et al 2016).

Dewari et al (2016) showed a reduction in reproductive capacity in their experiment. Over a nine-week period, zebrafish were given daily alcohol to mimic

[14] The large dose was equivalent to 125 mL/100g of body weight.

heavy drinking before being allowed to breed. There was a significant reduction in the number of eggs laid when either one or both parents were alcohol-exposed as compared to the controls. For example, the total number of eggs produced was 1300 in the control group (both parents no-alcohol) compared to 500 when both parents received alcohol (and 400-800 where one parent alcohol-exposed and one not).

Then a number of alcohol-exposed fish had a nine-week period of no alcohol, and there was "a complete restoration of fecundity to normalcy" (Dewari et al 2016 p85).

7.4. MISCELLANEOUS

(1) Budzynski et al (2016) looked for differences between alcohol dependent individuals who successfully abstained from alcohol for six months and those who relapsed in a sample of fifty-four males in Poland. All participants attended the same Addiction Therapy Unit in a hospital.

Twenty-three individuals abstained and 31 relapsed. No differences were found in demographic and clinical variables (eg: decrease in alcohol tolerance; history of delirium tremens (DTs)) between the two groups, but there was a significant biochemical difference. The blood concentration of nitric oxide metabolites was lower in relapsed individuals at baseline, and increased over the study period, while abstainers were the opposite. Nitric oxide metabolite concentration is linked to reactive oxygen species (FOS) production, which is part of the regulation of energy and food intake. This involves the hypothalamus which controls feeding behaviour, and "alcohol overuse may stimulate a vicious cycle of both fat and alcohol intake and ROS overproduction" (Budzynski et al 2016 p58) [15].

(2) AUDs are associated with increased pulmonary diseases (eg: bacterial pneumonia; acute respiratory distress syndrome) in frequency of illness and severity of symptoms. For example, AUD patients with bacterial pneumonia have longer hospitalisation, more intensive care unit admissions, greater need of mechanical ventilation, and poorer survival rates than the general population (O'Halloran et al 2016).

O'Halloran et al (2016) reported that AUDs were associated with negative changes in aspects of the immune

[15] Alcohol (or ethanol) withdrawal syndrome describes the symptoms that appear after an individual reduces or stops alcohol consumption after a long period of large intake (eg: alcohol withdrawal-induced anxiety-like symptoms). There is a risk that individuals return to drinking to stop these symptoms (Kumar et al 2016).

system than defend against bacterial pneumonia. Nineteen individuals with AUDs at a rehabilitation clinic in Denver, USA, were compared to twenty healthy controls.

(3) Alcohol consumption before and/or during sex is associated with a greater risk of acquiring HIV, via a greater likelihood of unprotected sex, group sex, and increased frequency of sexual activities, especially for men who have sex with men (MSM) (Fan et al 2016).

In Chongqing, where HIV among MSM is the highest of Chinese cities (Fan et al 2016), for example, a quarter of MSM drink alcohol before sex with a male partners (Liu et al 2014).

Fan et al (2016) explored this further in a survey of 391 MSM in Chongqing in 2011-12. Each participant was assigned a unique code number for the anonymous computer-assisted questionnaire, which included AUDIT, and the blood sample.

Overall, 20.5% of MSM reported alcohol consumption before or during sex in the last six months. MSM who drank alcohol (compared to MSM non-drinkers) were significantly more likely to:

- Have had anal sex with male casual partners in last six months (10 vs 2.7%);
- Not have used condoms due to alcohol in last six months (33 vs 18%).

Among the sample, 28 MSM (7% of total sample) were classed as heavy drinkers, and half of them drank alcohol before or during sex.

The link between alcohol use and HIV infection among MSM was not confirmed with the blood tests, but alcohol users were engaging in more HIV-risky related behaviours (eg: unprotected sex) than non-drinkers.

The sample were volunteers recruited via "local gay-oriented community-based organisations", and by individual snowball sampling. "Unlike MSM in Western countries, MSM in China not only have to deal with stigma and discrimination from families and society for their homosexuality identity..., but also have to marry a woman or find a girlfriend to hide their homosexual orientation and to carry on the family line... Additionally, the pursuits of sexual pleasure and the fear of making partners feeling mistrusted are continuously prevalent among Chinese MSM" (Fan et al 2016 pp1-2).

(4) Moderate alcohol consumption lowers blood pressure in younger women, but not in older women, which suggests an interaction between the components of alcohol and oestrogen (Yao et al 2016).

(5) Anxiety disorders and alcohol abuse are common together (eg: three-quarters of alcoholics having a current or previous diagnosis of an anxiety disorder) (Sharko et al 2016).

But what is the relationship between the two? Anxiety can motivate alcohol consumption as a means of relief, while chronic alcohol abuse can lead to anxiety disorders, or there may be a common factor in both conditions (Sharko et al 2016).

Sharko et al (2016) found support for the latter relationship in a study of overlapping neurobiological mechanisms in male rats.

(6) Mindfulness, which is defined as "an awareness of present moment experience and having a non-judgmental and accepting attitude toward the experience" (Schellhas et al 2016 p51), could be beneficial with addictive behaviours. For example, Zgierska et al (2008) reported a significant decrease in alcohol consumption in an eight-week pilot study with mindfulness-based meditation training, while Bowen et al (2014) found less alcohol consumption at twelve months follow-up with mindfulness-based relapse prevention.

If mindfulness reduces alcohol consumption, Schellhas et al (2016) wondered why. They applied action identification theory (Vallacher and Wegner 1987), which focuses on "the abstractness with which behaviour is represented in the mind" (Schellhas et al 2016). Behaviour can be represented as concrete (low-level) (eg: muscle movements) or abstract (higher-level) (eg: related to values and goals). So, alcohol consumption can be represented as, for example, swallowing the liquid (concrete) or relieving tension (abstract) (Schellhas et al 2016).

Alcoholic inpatients have been found to score higher on higher-level representations of alcohol and lower on low-level representations than the general population (Schellhas et al 2016). So, mindfulness may reduce alcohol consumption by focusing on the present moment (concrete representation).

(7) Alcohol consumption by undergraduates is more frequent than non-students, and may meet the criteria for "heavy drinking" (ie: five or more drinks on one occasion) more often (Foster et al 2016).

Foster et al (2016) focused on three psychological aspects of drinking in their study with around 350 heavy drinking students at a US university.

a) Drink refusal self-efficacy (DRSE) - the belief that individual can refuse alcohol, and it is negatively

associated with heavy drinking. The Drink Refusal Self-Efficacy (DRSE) Questionnaire (Young and Oei 1996) was used, with items like "I strongly believe that I can resist drinking".

b) Alcohol expectancies - the anticipated consequences (positive and negative) from drinking (eg: relaxation, changes in mood), and this interacts with DRSE. It was measured by items like "after a few drinks of alcohol, I would be more likely to be a better lover" on the Comprehensive Effects of Alcohol Questionnaire (CEOA) (Fromme et al 1993).

c) Drinking intention (DI) - this was measured by the Daily Drinking Questionnaire (DDQ) (Collins et al 1985).

It was found that DI moderated the association between DRSE and AE. The most alcohol was consumed by individuals with low DRSE (ie: unable to refuse) and high DI, followed by high DI and high DRSE individuals, while low DI scorers drank less (irrelevant of DRSE score) (table 7.1).

	Low DI/Low DRSE	Low DI/High DRSE	High DI/Low DRSE	High DI/High DRSE
Low negative AE	6	4	15	9
High negative AE	6	4	15	8

(Based on Foster et al 2016 figure 1 p69)

Table 7.1 - Mean number of drinks per week.

7.5. REFERENCES

Bowen, S et al (2014) Relative efficacy of mindfulness-based relapse prevention, standard relapse prevention, and treatment as usual for substance use disorders: A randomised clinical trial JAMA Psychiatry 71, 547-556

Budzynski, J et al (2016) Oxidoreductive homeostasis in alcohol-dependent male patients and the risk of alcohol drinking relapse in a six-month follow-up Alcohol 50, 57-64

Collins, R.L et al (1985) Social determinants of alcohol consumption: The effects of social interaction and model status on the self-administration of alcohol Journal of Consulting and Clinical Psychology 53, 189-200

Dewari, P.S et al (2016) Reversible loss of reproductive fitness in zebrafish on chronic alcohol exposure Alcohol 50, 83-89

Fan, W et al (2016) Alcohol drinking and HIV-related risk among men who have sex with men in Chongqing, China Alcohol 50, 1-7

Fey, W et al (2017) Development of stimulus material for research in alcohol use disorders International Journal of Methods in Psychiatric Research 26, e1527

Foster, D.W et al (2016) The road to drink is paved with high intentions: Expectancies, refusal self-efficacy, and intentions among heavy drinking college students Alcohol 50, 65-71

Fromme, K et al (1993) Comprehensive effects of alcohol: Development and psychometric assessment of a new expectancy questionnaire Psychological Assessment 5, 19-26

Karlsson, O & Roman, E (2012) Dose-dependent effects of alcohol administration on behavioural profiles in the MCSF test Alcohol 50, 51-56

Kumar, J et al (2016) The effects of acute ethanol administration on ethanol withdrawal-induced anxiety-like symptoms in rats: A biochemical study Alcohol 50, 9-17

Liu, Y et al (2014) Alcohol use among Chinese men who have sex with men: An epidemiological survey and meta-analysis BioMed Research International 2014, 414381

O'Halloran, E.B et al (2016) Alveolar macrophage inflammatory mediator expression is elevated in the setting of alcohol use disorders Alcohol 50, 43-50

Pronk, T et al (2015) validation of the Amsterdam Beverage Picture Set: A controlled picture set for cognitive bias measurement and modification paradigms Alcoholism: Clinical and Experimental Research 39, 10, 2047-2055

Schellhas, L et al (2016) How to think about your drink: Action-identification and the relation between mindfulness and dyscontrolled drinking Addictive Behaviours 56, 51-56

Sharko, A.C et al (2016) Ethanol-induced anxiolysis and neuronal activation in the amygdala and bed nucleus of the stria terminalis Alcohol 50, 19-25

Stritzke, W.G et al (2004) Assessment of substance cue reactivity: Advances in reliability, specificity, and validity Psychology of Addictive Behaviours 18, 2, 148-159

Vallacher, R.R & Wegner, D.M (1987) What do people think they are doing? Action identification and human behaviour Psychological Review 94, 3-15

West, C.H.K et al (2016) Reprint of: Locus coeruleus neuronal activity determines proclivity to consume alcohol in a selectively-bred line of rats that readily consumes alcohol Alcohol 50, 91-105

Wrase, J et al (2002) Development of alcohol-associated cues and cue-induced brain activation in alcoholics European Psychiatry 17, 5, 287-291

Wscieklica, T et al (2016) Alcohol consumption increases locomotion in an open field and induces Fos-immunoreactivity in reward and approach/withdrawal-related neurocircuitries Alcohol 50, 73-82

Yao, F et al (2016) Oestrogen receptor ER-alpha plays a major role in ethanol-evoked myocardial oxidative stress and dysfunction in conscious female rats Alcohol 50, 27-35

Young, R.M & Oei, J.P (1996) Grape expectations: The role of alcohol expectancies in the understanding and treatment of problem drinking International Journal of Psychology 28, 337-364

Zgierska, A et al (2008) Mindfulness meditation for alcohol relapse prevention: A feasibility pilot study Journal of Addiction Medicine 2, 165-173

8. MISCELLANEOUS SMOKING BEHAVIOUR

8.1. ANXIETY SENSITIVITY

Anxiety sensitivity (AS) is "the tendency to fear anxiety-related experiences" (Guillot et al 2016), and it is distinct from anxiety. AS is a risk factor for smoking, and high AS individuals have been found to be more likely to smoke for "the negative affect (NA) alleviating (ie: negative reinforcing) effects of smoking" (Guillot et al 2016 p31) (eg: loss of good mood).

AS is not a single dimension, but has three aspects (Guillot et al 2016):

- Physical concerns - fear that anxiety-related physical symptoms could be harmful;

- Cognitive concerns - fear that anxiety-related cognitive difficulties could be signs of mental abnormalities;

- Social concerns - fear that others will notice the anxiety symptoms.

Guillot et al (2015) found that the first two aspects of AS were associated with greater problems during tobacco abstinence, while Guillot et al (2016) confirmed the different relationship between the three aspects and smoking cessation.

Guillot et al (2016) recruited 473 treatment-seeking smokers in the USA, who had smoked an average of eight cigarettes or more for at least one year, and were highly motivated to quit.

AS was measured by the Anxiety Sensitivity Index (ASI-3) (Taylor et al 2007), which has eighteen items, each scored from 0 to 4. There are six items for each of the three aspects of AS:

- Physical concerns - eg: "it scares me when my heart beats rapidly", "it scares me when I am short of breath", "it scares me when I feel faint";

- Cognitive concerns - eg: "when my thoughts seem to speed up, I worry that I might be going crazy", "when I am nervous, I worry I am mentally ill", "it scares me when I am unable to keep my mind on a task";

- Social concerns - eg: "I worry that other people will notice my anxiety", "it is important to me not to appear nervous", "it embarrasses me when my stomach growls".

Other questionnaires were completed on nicotine dependence, smoking history, reasons for smoking, and smoking consequences. The latter was measured by the Smoking Consequences Questionnaire (SCQ) (Brandon and Baker 1991), which covers the outcome expected from smoking - sensory satisfaction (positive reinforcement) and reduction of NA (negative reinforcement).

AS physical and cognitive concerns were found to be associated with NA reduction, while AS social concerns was associated with both positive and negative reinforcement outcome expectancies on the SCQ.

These findings suggest that high AS individuals smoke because it reduces unpleasant physical and cognitive symptoms (eg: racing heart and thoughts) (negative reinforcement of smoking behaviour), and in doing so, reduces the fear that individual's anxiety symptoms will be noticed (negative reinforcement), while making the individual more relaxed in social situations (positive reinforcement). The authors recommended smoking cessation programmes that take these findings into account for high AS smokers.

8.2. SMOKING ONSET

Understanding the reason for the onset of smoking in adolescence will help in stopping that behaviour. Smoking susceptibility factors include low smoking refusal skills when offered a cigarette by a friend, peer or adult, and having the intention to smoke in the future. These factors are influenced by personal characteristics, norms, and self-efficacy, for example (Memetovic et al 2016).

Key relevant personal characteristics are sensation seeking, impulsivity, AS, and NA (hopelessness), which are all included in the Substance Use Risk Profile Scale (SURPS) (Woicik et al 2009). It has 23 items, each rated "strongly disagree" (1) to "strongly agree" (4) (table 8.1).

Among 1352 adolescents in the British Columbia Adolescent Substance Use Survey in Canada, Memetovic et al (2016) found that higher scores on hopelessness, impulsivity, and sensation seeking were associated with a greater intention to smoke (as measured by, "do you think you might try smoking in the future?"). AS had no significant association.

- I am happy
- I have faith that my future holds great promise
- It frightens me when I feel my heart beat change
- I feel that I'm a failure
- I am interested in experience for its own sake even if it is illegal

(Source: Woicik et al 2009)

Table 8.1 - Sample items from SURPS.

8.3. POINT OF SALE ADVERTISING

The tobacco industry "aggressively markets its products to consumers" at the point of sale (POS) mainly to increase brand awareness and loyalty. Colours, for example, are used to create associations (eg: green suggests fresh, cool, natural, and less harmful) (Nonnemaker et al 2016).

POS advertisements also act as a cue to smoke (ie: stimulate purchases among those not intending to smoke), and influence relapse among quitters (Nonnemaker et al 2016).

One policy response is "plain packaging" - ie: tobacco product packs with black and white text only - which was introduced in Australia in 2012. Subsequently, Wakefield et al (2013) found that Australian adults viewed cigarettes as lower quality and less satisfying in plain packaging, and were more likely to quit than with branded packaging.

In studies in countries with branded packaging, plain packed cigarettes were perceived as less attractive (by French smokers), and less appealing (by younger women in the UK), and the health warning would gain more attention (at least for non-smokers) (Nonnemaker et al 2016).

In experiments with a virtual store with no tobacco POS advertisements, Kim et al (eg: 2014) found a reduced urge to smoke compared to a control group.

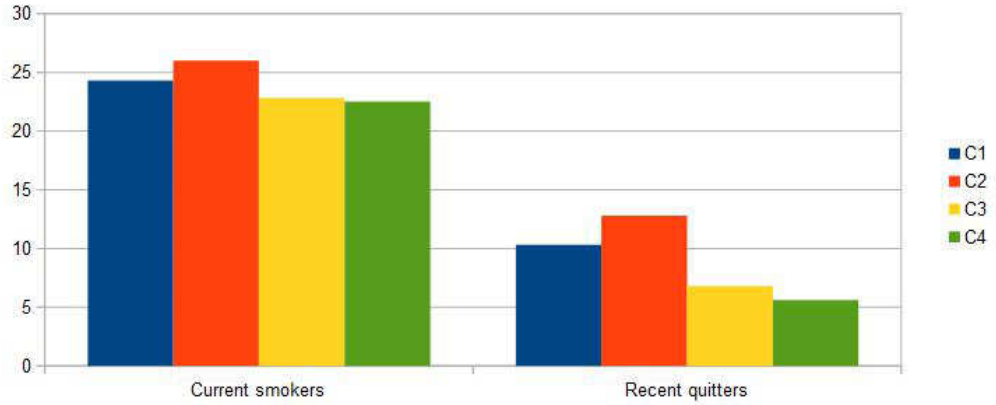
Nonnemaker et al (2016) reported the benefits of plain packaging in a virtual store. Over 1300 smokers or recent quitters in the USA performed a shopping task in a 3D interactive convenience store. The participants "visited" one of four stores that varied in the tobacco advertising and packaging:

- C1: Coloured packs and POS advertisements with full display shelves of cigarettes;
- C2: Plain packaging and advertisements with full display shelves;
- C3: Coloured packs and advertisements, but cigarettes on hidden shelves;
- C4: Plain packaging and advertisements and hidden

shelves.

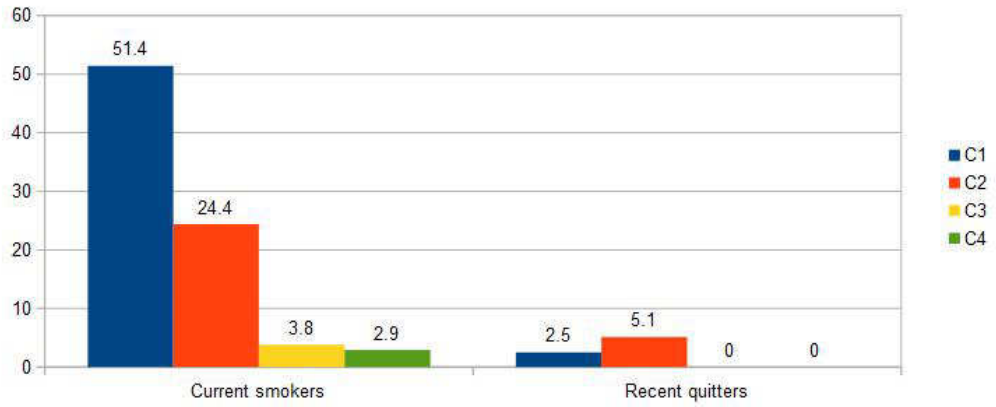
The main outcome measures were the urge to smoke after the virtual shopping task (rated 0 to 100), and the choice to purchase tobacco in the virtual store.

The urge to smoke did not vary significantly between conditions for current smokers, but it was significantly lower for recent quitters in C4 (figure 8.1). Significantly fewer participants purchased tobacco products when they were hidden (C3 and C4) (figure 8.2).



(Data from Nonnemaker et al (2016) figure 3 p19)

Figure 8.1 - Mean urge to smoke (out of 100) based on condition and smoking status.



(Data from Nonnemaker et al (2016) figure 3 p19)

Figure 8.2 - Percentage purchasing tobacco products in virtual store based on condition and smoking status.

Nonnemaker et al (2016) summed up: "Our results suggest that enclosing the tobacco product display or

mandating plain packaging and ads might lead to fewer tobacco purchase attempts among current smokers. Among recent quitters plain packaging and ads might result in lower levels of urge to smoke" (p20).

The major limitation of this study was the virtual store rather than real-life. The average time to complete the shopping task was thirty-five seconds, which is half the time calculated in real-life stores for the same task (Nonnemaker et al 2016).

8.4. REFERENCES

Brandon, T.H & Baker, T.R (1991) The Smoking Consequences Questionnaire: The subjective expected utility of smoking in college students Psychological Assessment 3, 484-491

Guillot, C.R et al (2015) Differential associations between components of anxiety sensitivity and smoking-related characteristics Addictive Behaviours 40, 39-44

Guillot, C.R et al (2016) Anxiety sensitivity facets in relation to tobacco use, abstinence-related problems, and cognitions in treatment-seeking smokers Addictive Behaviours 56, 30-35

Kim, A.E et al (2014) Influence of point-of-sale tobacco displays and graphic health warning signs on adults: Evidence from a virtual store experimental study American Journal of Public Health 104, 5, 888-895

Memetovic, J et al (2016) Examining the relationship between personality and affect-related attributes and adolescents' intentions to try smoking using the Substance Use Risk Profile Scale Addictive Behaviours 56, 36-40

Nonnemaker, J et al (2016) Influence of point-of-sale tobacco displays and plain black and white cigarette packaging and advertisements on adults: Evidence from a virtual store experimental study Addictive Behaviours 56, 15-22

Taylor, S et al (2007) Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index-3 Psychological Assessment 19, 176-188

Wakefield, M.A et al (2013) Introduction effects of the Australian plain packaging policy on adult smokers: A cross-sectional study BMJ Open 3, 7, e003175

Woicik, P.A et al (2009) The Substance Use Risk Profile Scale: A scale measuring traits linked to reinforcement-specific substance use profiles Addictive Behaviours 34, 1042-1055