

Alcohol and the Developing Adolescent Brain: Evidence Review



SHAAP

SCOTTISH HEALTH ACTION ON ALCOHOL PROBLEMS
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1. Introduction and guide to this report

Scottish Health Action on Alcohol Problems (SHAAP) aims to provide a co-ordinated, coherent and authoritative medical and clinical voice on alcohol-related harm.

On 27th September 2013, SHAAP hosted a seminar where policymakers and experts from the fields of genetics, neuroscience and social psychology came together to review and discuss new and emerging medical and scientific research about the impact of alcohol on the adolescent brain and implications for policy and practice.

Introducing the day, Dr John Wilson (Vice-President of the Royal College of Physicians of Edinburgh (RCPE)) reminded the audience that clinicians see the consequences of alcohol misuse every day in their working lives:

'The medical profession now sees serious alcohol related illnesses occurring at younger ages and there is a worrying trend of harm to women in their thirties – none of this was evident 25–30 years ago. Thanks to new technology it is now possible to see how the brain develops and the effect of alcohol on the brain. It is great to have an opportunity to come together, hear from the experts and review the evidence base.'

For the purposes of this report, adolescence can be defined as:

*'the period of physical, psychological and social transition between childhood and adulthood.'*¹

The term can broadly be used to describe young people, from early teens to mid twenties. In this report, we have tried to make clear when our meanings are intended to be more specific than this.

Our event was organised as follows: First, experts presented papers. There was then a round table discussion, led by four key discussants, about the findings and implications for policy and practice. Two papers which provided background data about drinking patterns and alcohol-related harm amongst young people in Scotland were also presented. These are included in this report as Appendices One and Two. All these papers and the discussion have been summarised and form the basis of the report.

Structure of this report

Section Two: In the first paper, Dr. Iroise Dumontheil summarises the processes of development that occur during adolescence, the links between adolescence and mental health, and findings from research into peer influences on behaviour.

Section Three: In the next paper, Dr. Killian Welch reviews evidence on detectable brain abnormalities in young people with alcohol use disorders.

Section Four: Professor Gunter Schumann then goes on to discuss what advances in genetic research can teach us about different responses to alcohol by individuals.

Section Five: Professor Anne Lingford-Hughes makes the case for the importance of understanding the neuropsychobiological mechanisms for early-onset alcohol use and escalation of drinking so as to inform prevention and treatment.

Section Six: Dr. Gordon Fernie reviews the available evidence to determine alcohol's impact on impulsivity and control processes.

Section Seven: This aims to capture the main themes of our round table discussion. This process was facilitated by the discussants; Professor Stephen Lawrie, Dr. Peter Rice, Dr. Evelyn Gillan and Eric Carlin.

Appendix One: Dr. Deborah Shipton discusses findings of national surveys of self-reported alcohol consumption provide insight into adolescent drinking in Scotland.

Appendix Two: Dr. Lesley Graham examines the evidence on harms to young people from alcohol consumption.

References

- 1 Christie D & Viner R (2005) *Adolescent Development*. British Medical Journal 330:301–304

2. Brain development in adolescence

A summary of the processes of development that occur during adolescence, the links between adolescence and mental health, and findings from research into peer influences on behaviour.

Dr. Iroise Dumontheil, Department of Psychological Sciences, Birkbeck, University of London

2.1 How does the brain change during adolescence?

Until recently it was widely believed that the brain was anatomically mature from childhood. Changes in social behaviour during the teens were attributed to hormones, social experience or the changing social environment. However, advances in the field of magnetic resonance imaging (MRI) have shown that the structure of the brain undergoes continuous and prolonged development until early adulthood.¹

Brain development in adolescence is of particular significance when considering that the leading causes of deaths for 15–24-year-olds in the USA were not linked to disease, but to behaviour². Similar patterns are also evident in the United Kingdom, according to data released in 2012.³

We now know that synaptic density – the number of connections (synapses) between neurons – increases drastically in the first few months and years of life. After this the brain undergoes a period of “pruning”; systematically discarding parts that are inactive and strengthening connections that are frequently used. During childhood, this pruning mainly occurs in the areas responsible for sensory (e.g. vision) and motor processes, whereas in adolescence pruning occurs in the prefrontal cortex, the area responsible for planning, inhibitory control, decision-making and social interactions, and in the temporal cortex, which includes regions supporting social cognition.

2.2 What are the key differences between the adolescent and the adult brain?

Recent MRI studies have shown that, in addition to the changes in brain structure observed during adolescence in the temporal and frontal cortex, there are also changes in brain activity and behaviour. Brain activity differences

between adolescents and adults have mainly been reported in two types of situations.

First, adolescents show differences in the activation of “the social brain”, during the processing of social information, e.g. when adolescents are asked to think about their own or other people’s thoughts, or to look at and interpret emotional expressions on the faces of other people. These changes in social and affective processing – the means by which we make sense of social and emotional information – may be crucial to understanding adolescent vulnerabilities. For example, adolescents and adults show differences in response to social distress; experiments involving ostracism suggest that adolescents’ brains become more aroused in response to social exclusion than do adults’, and their mood is consequently more affected.⁴

Structural changes in white matter during adolescence are thought to underlie the establishment of long-range connections that support top-down modulation of behaviour.⁵ Adolescents show differences in activation of the frontal cortex in situations that require reasoning, decision-making, or inhibitory control. As a result, it is suggested that adults are better at mentally comprehending the longer term impact of behaviour and that, therefore, appreciation of adverse consequences plays a greater role in decision-making for adults than young people.

2.3 How do peers influence decision-making?

One key difference in terms of behaviour between adolescents and adults lies in situations where decisions have to be taken in a social context. This is particularly apparent when looking at the influences of peers on adolescents’ decision-making.

During adolescence, relationships with peers often become more elaborate, personal and emotional. This activity is linked to the development of the prefrontal cortex, the region responsible for regulating behaviour and social interactions. The frequency of interactions with peers increases as young people become independent and spend more time with their friends, reducing the time spent with family members.

When adolescents are with their peers they take more risks; this is evidenced through numerous trials and experiments. For example, in simulated driving situations young people engage in riskier driving behaviour if being watched by peers; the same young people are more cautious (at adult-levels) when participating in simulated driving tests on their own.⁵ Similarly, young offenders’ crimes are typically committed when they are in the company of peers.⁶

Adolescents’ increased risk-taking in a social context may result from differences in the development of two key systems. The limbic system – which controls basic emotions and processes reward information – matures faster than the prefrontal cortex (the region responsible

for planning and self-regulation).⁷ This difference in risk sensitivity may be an evolutionary device, in that it potentially prepares young people to leave the protection of their home and make their own way in the world.

Peer influence can also have positive impacts. For example, academic performance and motivation can improve when students associate with high-achieving peers. There can also be an increase in pro-social behaviour when young people spend time with peers engaged in positive activities.

2.4 What do we know about the adolescent brain and mental health?

A range of mental health conditions commonly emerge in adolescence; these include anxiety disorders, bipolar disorder, depression, eating disorders, psychosis and substance use experimentations.

The link between adolescence and the onset of psychopathology may emerge from the fact that “moving parts get broken”. Adolescence is characterised by changes in the neural systems that support reasoning, social interactions, cognitive control of emotions, risk and reward evaluation. When these changes are too extreme or take place at the wrong time, and interact with certain psychosocial factors (e.g. school or relationships) and/or biological environmental factors (e.g. pubertal hormones or psychoactive substances) there is an increase in the risk of cognitive, affective and addictive disorders.⁸

2.5 Summary of key points

- It is clear that there are significant differences between adult and adolescent brain functioning. As well as anatomical and functional variations, social drivers such as peer influence and being excluded from peer/social groups can have a stronger impact on young people, compared to adults.
- Given these variances in key aspects of brain function and responses, there are good grounds for further research into the different impacts that alcohol may have on young people in comparison to adults.
- There is scope to build on the findings that young people are less responsive to long-term consequences of behaviour than adults. For example, this may inform the design of public health messages and interventions designed to influence decision-making.

References

- 1 Giedd JN and Rapoport JL (2010). *Structural MRI of pediatric brain development: what have we learned and where are we going?* *Neuron*, 67:728–734.
- 2 Borse NN, Gilchrist J, Dellinger AM, Rudd RA, Ballesteros MF, Sleet DA (2008). *CDC Childhood Injury Report: Patterns of unintentional injuries among 0-19 year olds in the United States, 2000–2006*. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.
- 3 Office of National Statistics Office (2013). ‘Leading cause of death in England and Wales’ and General Register Office for Scotland (2011). Births, deaths and other vital events: Causes of death.
- 4 Sebastian CL, Tan GCY, Roiser JP, Viding E, Dumontheil I, Blakemore, S-J (2011). *Developmental influences on the neural bases of responses to social rejection: implications of social neuroscience for education*. *NeuroImage*, 57:686–694.
- 5 Luna B, Padmanabhan A, O’Hearn K (2010). *What has fMRI told us about the development of cognitive control through adolescence?* *Brain and Cognition*, 72:101–113.
- 6 Gardner M and Steinberg L (2005). *Peer influence on risk taking, risk preference, and risky decision-making in adolescence and adulthood: an experimental study*. *Developmental Psychology*, 41:625–635.
- 7 Casey BJ, Jones RM, Hare TA (2008). *The adolescent brain*. *Annals of the New York Academy of Sciences*, 1124:111–126.
- 8 Goddings A-L, Burnett Heyes S, Bird G, Viner RM, Blakemore S-J (2012). *The relationship between puberty and social emotion processing*. *Developmental Science*, 15:801–811.

3. The effects of alcohol on the developing adolescent brain

A review of the available evidence on detectable brain abnormalities in people up to the age of 40 with alcohol-use disorders.

Dr. Killian Welch, Robert Ferguson Unit, Astley Ainslie Hospital, Edinburgh

3.1 How does the brain develop during adolescence?

Brain development continues through adolescence: synaptic pruning discards synapses that are inactive, heavily used networks are strengthened and myelination, (the 'insulating' of neurones), increases the speed of neural communication. However, different brain regions reach maturity at different times. In adolescence, though the brain regions that control desires and motivation are well developed, the frontal regions (responsible for planning, impulse inhibition and abstract thought) are still maturing. As a result, adolescents are less able to resist urges and also less able to conceptualise the adverse consequences of behaviour, particularly future problems. Their potential for mood volatility, conflict, and risky behaviour is often understandable.

3.2 What do the studies suggest about the longer-term impacts of alcohol misuse by adolescents?

Research demonstrates that adolescent animals are more susceptible than adults are to the acute effects of alcohol and that the consequences of alcohol exposure persist into adulthood. Studies of adolescent humans suggests that they are also more susceptible than adults to alcohol-induced memory impairment. Animal data suggests however that adolescents may be less susceptible to the motor effects of alcohol.¹ This would mean that the signs that adults often recognise as indicating intoxication (clumsiness, unsteady walking) would not occur in an adolescent until higher levels of blood alcohol were reached. There is also evidence that adolescent heavy drinking increases the probability of alcohol disorders developing in later life.²

If adolescent alcohol use can be associated with persistent effects in adulthood, this suggests that it may be interacting with adolescent brain development. To help us understand

this process, a number of studies have compared brain structure in adolescents who drink heavily with those who do not (cross-sectional studies). Some of the differences seen in adolescent heavy drinkers include:

- Reductions in the hippocampal volume (the central area of the brain that controls spatial navigation and short-term and long-term memory). This suggests that the adolescent hippocampus may be especially vulnerable to alcohol-related damage.
- Decreases in prefrontal cortex (that part of the brain responsible for executive function) of adolescents, although only consistently observed in females.
- Reduced integrity of white matter regions throughout the brain. White matter integrity is important to how the brain learns and functions.
- Reduction in a major white matter tract, the corpus callosum, in young adult women with alcohol-use disorders. The corpus callosum has a role in thinking processes, including problem solving.

The most useful studies follow up adolescents over time to see if the brain develops differently in those who drink heavily compared to those who do not; these are called longitudinal studies. This gets around the problem of not knowing if differences seen when drinkers are compared to non-drinkers are a cause or consequence of use of the substance. Only one study has done this to date, and it reported that alcohol use was associated with reduced integrity in the superior longitudinal fasciculus, a tract which regulates motor behaviour.³

3.3 Limitations of the data

At present the data is limited, and some findings contradict each other. For example, in contrast to the single longitudinal study³, one cross-sectional study found increased integrity of the major white matter tract in adolescents with alcohol-use disorders compared to the control group.⁴ Also, while some of the earlier studies of adolescents with alcohol use disorders reported reductions in the hippocampal volume (the central area that controls spatial navigation and short-term and long-term memory) another study with a similar sample group found no reduction. Such contradictions are normal when there are few studies, but the trend (and strongest data we have to date) seems to suggest that adolescent alcohol use is associated with non-trivial brain structural differences. Whether these are definitely due to alcohol only (though studies control for their effects, young alcohol users are often using other drugs too), and whether they are associated with other influences need to be explored further.

3.4 Summary of key points

- There is some evidence that alcohol-associated brain structural effects are more pronounced in adolescents than in young adults.
- Mid-to-late adolescence may be an age of particular vulnerability for the hippocampus. Female adolescents may be more susceptible to white matter effects.

References

1. White A M; Bae J G; Truesdale M C; et al (2002). *Chronic-intermittent ethanol exposure during adolescence prevents normal developmental changes in sensitivity to ethanol-induced motor impairments*. *Alcoholism: Clinical and Experimental Research* 26:960–968.
2. Welch KA, Carson A, Lawrie SM (2013) *Brain structure in adolescents and young adults with alcohol problems: Systematic Review of Imaging Studies Alcohol and Alcoholism*. 48:433–444.
3. Bava S, Jacobus J, Thayer RE et al. (2012) *Longitudinal changes in white matter integrity among adolescent substance users*. *Alcoholism: Clinical and Experimental Research*. Jan 2013; 37(Suppl 1): E181–E189.
4. De Bellis MD, Van Voorhees E, Hooper SR et al. (2008). *Diffusion tensor measures of the corpus callosum in adolescents with adolescent onset alcohol use disorders*. *Alcoholism: Clinical and Experimental Research* 32:395–404.

4. The role of genetics in alcohol consumption

Advances in genetic research can teach us about different responses to alcohol by individuals.

Professor Gunter Schumann MD, Chair in Biological Psychiatry, King's College, London

4.1 How does neuroimaging and genomic research link to alcohol and addiction?

Most of our current understanding about adolescent behaviour is based on evidence gathered at the population or subgroup level. While environmental and social factors play a role in influencing behaviour, geneticists are also investigating what can be determined at the individual level.

A greater understanding of impulsivity – the interplay between inhibitory control and reward anticipation – is important because it is a factor in risky behaviours such as harmful or hazardous alcohol use and smoking. The ability to identify individuals who are most at risk of harmful behaviours may enable us to improve the effectiveness of prevention and/or early intervention programmes.

Genomic research also contributes an understanding of heritability in alcohol use, abuse and dependence. These factors, combined with behavioural traits and environmental influences may contribute to the development of addictions.

4.2 Evidence from the IMAGEN research study

The IMAGEN study is a European research project investigating mental health and risk-taking behaviour in teenagers. This longitudinal genetic-neuroimaging study involves a cohort of 2000+ adolescents. The research, carried out by 17 participating institutions across Europe, explores the neurobiological basis of individual differences in brain responses to reward, punishment and emotional cues at 14, 16 and 19 years. The information presented in this document relates to the first tranche of research conducted with 14-year-olds.

This research involves identifying the distinct neuropsychological processes involved in reward anticipation. A range of experiments on participants' responses to stimuli, combined with brain scanning, has established that three clusters in the brain play a significant role in reward. The vision cluster links to emotional anticipation, the sensory and motor cluster

relates to reward sensitivity and the reward cluster relates to response preparation.

New tools and techniques help scientists to learn more about these markers. They test their predictive value by comparing genetic profiles against observed behaviours, neuroimaging of the brain and blood markers. Other tools include self-report questionnaires, behavioural assessment and interviews.

4.3 What does the research tell us about adolescent behaviour?

Young people with potentially problematic substance use showed greater risk-taking and lower activation of specific brain areas than their peers. This may suggest that some young people are more predisposed to problematic substance use than others; substances affect individual brains at varying speeds and there are different dopamine reactions to psychoactive substances.¹ This may imply that some people need to consume more than their peers do in order to achieve the same effect from a substance.

The research also suggests that certain markers are associated with different manifestations of impulsive behaviour. Neuroimaging shows that distinct areas of brain activity relate to inhibitory control and reward anticipation. Activity and neural density in key areas of the brain are associated with a likelihood of impulsiveness and initiating substance use in early adolescence.^{1,2}

Genome research with mice has also explored the reinforcing properties of addictive substances. Those animals with a specific gene which regulates dopamine neuronal activity showed an increase intake and preference for ethanol and higher levels of dopamine activity. Mice without the gene consumed less, were less responsive to ethanol and had lower dopamine activity. This is important because the reinforcing properties of substances seem linked to their dopamine effects. Dopamine is a neurotransmitter that helps control the brain's reward and pleasure centres. It suggests that the young people with this gene are likely to have an increased response to, and preference for, addictive substances.¹

4.4 Summary of key points

- The research provides insight into the processes that occur when addictive substances are used and suggests that some young people are predisposed to be more susceptible to the impact of substances than others.
- Emerging findings around the reinforcing properties of substances also suggest there is scope to reconsider the current research focus, whereby studies typically focus on one substance at a time (for example, alcohol). It would also be useful to conduct investigations that would examine the interplay between addictive substances.

References

- 1 Whelan, R et al. (2012). *Adolescent impulsivity phenotypes characterized by distinct brain networks*. *Nature Neuroscience* 15,920–925 doi:10.1038/nn.3092.
- 2 Schilling, C et al (2013). *Cortical thickness of superior frontal cortex predicts impulsiveness and perceptual reasoning in adolescence*. *Molecular Psychiatry* 18, 624-630 | doi:10.1038/mp.2012.56.

For further details on the work of this project see: <http://www.imagen-europe.com>

5. The clinical implications of alcohol's effects on the brain

Understanding the neuropsychobiological mechanisms for early-onset alcohol use and escalation of drinking is critical to inform prevention and treatment work.

Professor Anne Lingford-Hughes, Centre for Neuropsychopharmacology, Imperial College London

5.1 What does neuropsychobiological research tell us about the impact of alcohol?

Scientists know that the adolescent brain is malleable and differs from the adult brain. For example, areas that control cognition and planning are not fully developed until adulthood, and throughout adolescence a process of pruning eradicates unused synapses while frequently used connections are reinforced. We also have an understanding of the brain regions affected by alcohol-related disease in adults.

There is now a need to determine if the effects of substances on a young person's brain differ from those on a mature adult brain and result in lasting changes.

5.2 Key differences in the impact of alcohol on the adolescent and adult brain

Research with rats has explored the impact of repeated alcohol exposure on the brain at different ages. The regions of the brain susceptible to alcohol-induced harm in rats were found to differ between adolescents and adults; adolescent binge drinkers showed more damage in frontal brain regions, whereas in adult rats the damage was greater in posterior regions.¹

Rodent research has also shown that younger animals are less sensitive to the sedative and motor-impairing effects of alcohol. This means they can stay awake for longer and move more than adults – both these factors facilitate increased alcohol consumption.

There are also differing effects on memory. Adult rats perform better than adolescent rats on memory tasks and their spatial memory is not affected by alcohol. This suggests that adolescent binge drinking may lead to enhanced vulnerability to alcohol-induced spatial memory

impairments in later life. Although this finding is based on animal experiments, work by Townshend and Duka has shown that student binge drinkers do perform worse in tests of cognitive performance compared to lighter drinkers.²

Human adolescents who became heavier drinkers showed more activation in the reward areas of the brain than those who were non- or minimal drinkers, suggesting increased impulsiveness during response inhibition tasks such as the Go-No-Go test. This test requires participants to switch between alternative actions in a simulated experience (for example, stop the car, start the car).

Given that the brain is maturing during adolescence, alterations in chemistry might result in enduring deficits in control of emotion, logical thinking and inhibition of impulsivity. In turn this lack of executive control could exacerbate addictive tendencies.³

5.3 How does alcohol affect the brain?

Much of our understanding about alcohol's impact on brain chemistry comes from research with adults, although evidence for the impact on adolescents is growing.

Alcohol's effects on the brain include anxiety, sedation, memory impairment and chronic heavy consumption, which may result in tolerance and withdrawal symptoms such as seizures.

The effects of alcohol have been linked to GABA, the main inhibitory mechanism of the nervous system. In the short-term, alcohol increases GABA activity, leading to reduced anxiety, slurred speech, sedation, disinhibition and reduced conscious.

Over the longer term alcohol blunts GABA functioning, which is associated with a lower level of response to alcohol. This means that people require increasing doses of alcohol to achieve an effect (tolerance). The implications of alcohol use depend on an individual's initial GABAergic functions – suggesting those with lower levels are more likely to require greater levels of alcohol to achieve an effect, and therefore are at higher risk of developing alcohol problems than those who react more intensely to alcohol at the outset.⁴

Simultaneously, alcohol also blocks the excitatory glutamatergic system. In the short term, alcohol blocks glutamate excitation. Chronic alcohol consumption results in a compensatory increased activity in the glutamatergic system. Such hyperactivity is toxic to the brain and can involve withdrawal symptoms such as seizures and brain damage. In addition, glutamate plays a key role in neuroplasticity (brain growth and development) and it is suggested that acute and chronic alcohol consumption in adolescence could result in long-term changes in brain circuitry by altering connections.³

5.4 Can the research help us to improve pharmacological interventions?

Until recently, research has focussed on gaining a greater understanding of how dopamine affects the brain so as to develop pharmacological treatments that might alter the dopaminergic reward pathways.

However, more metabolic pathways in the brain are now being investigated – such as GABA, and opioids – and these are resulting in medications that have potential to improve our success with treatments for substance misuse. Increased knowledge about which cognitive processes are dysregulated will also support efforts to use psychosocial approaches. Such intervention may prevent long-term changes in brain plasticity, reduce reinforcing potential, prevent brain damage/cognitive impairment and ultimately reduce substance misuse.

5.5 Summary of key findings

- The adolescent brain has a different and largely greater sensitivity to alcohol than the adult brain, which may lead to enduring changes. At present the evidence is restricted to short-term effects, but ongoing research will soon begin to identify more lasting impacts.
- There remain questions around attempting to study the impact of alcohol in isolation – because many young people use more than one substance at a time; for example alcohol and cigarettes, or alcohol and stimulants.
- Larger studies are required to understand the complexities of individual responses to alcohol. There are several areas for further exploration, including genetic vulnerability, gender differences, and comorbidity of factors such as depression and anxiety.
- By understanding more about the underlying neurobiology, informed development of pharmacological and cognitive prevention and treatment strategies will occur.

References

- 1 Markwiese BJ, Acheson SK, Levin ED, Wilson WA, Swartzwelder HS (1998). *Differential effects of ethanol on memory in adolescent and adult rats*. Alcoholism: Clinical and Experimental Research; 22: 416–421.
- 2 Townshend J M & Duka T (2005). *Binge Drinking, Cognitive performance and mood in a population of young social drinkers*. Alcoholism: Clinical and Experimental Research 29(3): 317–25.
- 3 Carpenter-Hyland EP, Chandler L J (2007). *Adaptive plasticity of NMDA receptors and dendritic spines: implications for enhanced vulnerability of the adolescent brain to alcohol addiction*. Pharmacology Biochemistry and Behaviour; 86: 200–208.
- 4 Hu X, Oroszi G, Chun J, Smith TL, Goldman D, Schuckit MA (2005). *An expanded evaluation of the relationship of four alleles to the level of response to alcohol and the alcoholism risk*. Alcoholism: Clinical and Experimental Research. 2005;29(1):8–6.

6. The effects of alcohol on psychological and cognitive function

A review of the available evidence to determine alcohol's impact on impulsivity and control processes.

Dr. Gordon Fernie, Division of Applied Medicine (Psychiatry), University of Aberdeen

6.1 What are the key cognitive changes during adolescence?

During adolescence, increases in novelty-seeking, sensation-seeking, risk-taking and reward sensitivity are observed prior to the maturation of regulatory or control systems.¹

Sensation-seeking and reward sensitivity peak during mid-adolescence whereas cognitive control processes such as planning and response inhibition increase gradually and linearly with age. These cognitive changes correspond with developments in neuroanatomical systems that undergo similar developmental trajectories throughout adolescence.^{2, 3}

Crucially, all these systems and processes are implicated in the development of addictions. Adolescence is both a critical stage of development and a period of potential vulnerability to exposure to addictive substances like alcohol. Alcohol use, therefore, has the potential to delay or disrupt cognitive control development or desensitise reward processing, establishing patterns and processes which leave young people predisposed to riskier behaviour or substance misuse.

6.2 How does alcohol affect adolescents?

There are challenges associated with observing the acute effects of alcohol on adolescents due to the ethical concern of giving alcohol to underage participants. Observations are typically restricted to research with animals, while studies involving adolescents generally rely on self-reported behaviour.

Research shows that in rodents the adolescent response to alcohol differs from adults; they are more sensitive to alcohol's rewarding effects and show increased social facilitation.^{4, 5}

Adolescent rats also have decreased sensitivity to alcohol's aversive effects such as sedation, motor

impairment, disruption in locomotion, and 'hangover' effects. These negative consequences also serve as cues, which may help to regulate intake. The differences in adult responses to alcohol may reflect the neurobiological changes occurring in adolescence.

Research with young people has shown that drinking episodes may increase the likelihood of experiencing aggressive or troublesome behaviours, e.g. getting into an argument or a fight. This behaviour may be understood as being related to a failure in response inhibition.⁶

6.3 Why do some young people drink more than others?

Cross-sectional studies in adolescents have revealed robust differences in personality traits between adolescents who drink regularly and those who do not.⁷ Greater impulsivity and sensation seeking are found in heavy drinking adolescents; these young people are also more likely to experiment with other substances. Cognitive impairment is found more often in adolescents and young adults with alcohol-use disorder.

Adolescent heavy drinkers show increased 'delay discounting' – the preference for immediate reward over long-term gain – compared to their peers. This impaired response inhibition is associated with increased frequency of alcohol consumption; it also means that this group is less likely to give consideration to any long-term negative consequences of behaviour.⁸

Young people from families with a history of alcohol or substance misuse are more likely to have elevated levels of impulsivity. As explained previously, impulsivity is linked to higher levels of drinking and drinking at an earlier age.⁹

6.4 Is there a connection between impulsivity and alcohol consumption?

The relationship between impulsivity and alcohol is complex and there are questions around cause and effect. While there are links between alcohol consumption and behavioural disinhibition, higher levels of impulsivity may pre-date alcohol involvement and serve as a risk factor for the development of heavy drinking and alcohol problems once individuals begin to experiment with alcohol.

Elevated impulsivity is a strong predictor of persistent alcohol use disorders into⁹ adulthood. Individual differences in the rate of increase in risk-taking (but not absolute levels of risk-taking) during early adolescence are predictive of subsequent alcohol involvement.¹⁰

Impulsivity has also been identified as a risk-factor for early adolescent alcohol use. Children identified as relatively impulsive and inattentive by their schoolteachers at age 11 are more likely to have experimented with alcohol by the age of 14.⁹

Large cohort studies have shown that in adolescents with an alcohol- or substance- use disorder, cognitive deficits are found eight or ten years later. These differences exist despite controlling for baseline performance and indicate that prolonged adolescent alcohol use caused these deficits. However, it is possible that individual differences precede the onset of these disorders in the first place, qualifying the nature of the causal relationship.^{11, 12}

References

- 1 Geier C F and Luna B (2009) *The maturation of incentive processing and cognitive control*. *Pharmacology, Biochemistry and Behavior*, 93(3), 212–221.
- 2 Galvan A, Hare TA, Parra CE, Penn J, Voss H, Glover G, et al (2006). *Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents*. *Journal of Neuroscience*;26(25):6885–92.
- 3 Strang NM, Chein JM and Steinberg L (2013). *The value of the dual systems model of adolescent risk-taking*. *Frontiers in Human Neuroscience* 7:223. doi: 10.3389/fnhum.2013.00223
- 4 Spear LP. (2013). *Adolescent neurodevelopment*. *Journal of Adolescent Health*, 52:S7–S13.
- 5 Goldstein R Z, Volkow N D (2002). *Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex*. *American Journal of Psychiatry*, 159, 1642–1652.
- 6 Fuller E (2013). *Smoking, drinking, and drug use among young people in England in 2012*. Care NICfHaS, editor. London.
- 7 De Wit H (2009). *Impulsivity as a determinant and consequence of drug use: a review of underlying processes*. *Addiction Biology*, 14:22–31
- 8 Colder C R, O'Connor R (2002). *Attention biases and disinhibited behavior as predictors of alcohol use and enhancement reasons for drinking*. *Psychology of Addictive Behaviors* 16, 325–332.
- 9 McGue M, Iacono W G, Legrand L N, Malone S and Elkins I (2001). *Origins and Consequences of Age at First Drink. I. Associations With Substance-Use Disorders, Disinhibitory Behavior and Psychopathology, and P3 Amplitude*. *Alcoholism: Clinical and Experimental Research*, 25: 1156–1165 .
- 10 MacPherson L, Magidson J F, Reynolds E K, Kahler C W, Lejuez C W. *Changes in sensation seeking and risk-taking propensity predict increases in alcohol use among early adolescents*. *Alcoholism: Clinical and Experimental Research*. 2010;34(8):1400–8.
- 11 Tapert S F, Granholm E, Leedy N G, Brown S A (2002). *Substance use and withdrawal: Neuropsychological functioning over 8 years in youth*. *J Int Neuropsychol Soc* 8:873-883.
- 12 Hanson K L, Medina K L, Padula C B, Tapert S F, Brown S A (2011). *Impact of adolescent alcohol and drug use on neuropsychological functioning in young adulthood: 10-year outcomes*. *Journal of Child and Adolescent Substance Abuse*. 20:135–154.

7. Discussion and recommendations

We do not attempt here to summarise the individual papers; rather we aim to capture the main themes of our round-table discussion. This process was facilitated by the discussants, Professor Stephen Lawrie, Dr. Peter Rice, Dr. Evelyn Gillan and Eric Carlin.

Based on the discussions, it was agreed that the following principles should inform policy and practice:

1 Protecting young people from alcohol-related harm has to be within the context of a ‘whole population’ approach to reduce overall alcohol consumption.

As well as focussing on the specific circumstances and needs of individual young people, reducing overall population alcohol consumption will have the most marked impact on reducing overall alcohol-related harms.¹

2 Protecting young people from alcohol-related harm should be embedded in national and local policy.

Public strategies need to prioritise action on price, marketing and availability, including paying specific attention to these in relation to young people. As the emerging evidence on alcohol and the brain indicates uncertainty about the about the risks posed by alcohol consumption at a young age, review of alcohol sales practices and alcohol legislation needs to focus not only on the age at which young people should be able to purchase alcohol but also the age at which it can legally be consumed. Deep concerns were also expressed in relation to advertising and specifically the role of social media in normalising the use of alcohol by young people.

There is a need to skill the workforce in all services which young people may come in contact with, including health service settings, schools, social and youth work and criminal justice services.

3 Emerging evidence that the adolescent brain may be especially vulnerable to alcohol harms should inform all health-promoting activities.

Brain development continues until the mid twenties. There is also clear evidence that suggests a likely causal association between alcohol use in adolescence and structural changes to the brain. Young people and those who have caring responsibilities, including parents, have

the right to have access to up-to-date, accurate information about this to enable them to make informed choices and to prevent or reduce harms. For parents, such new information might cause them to re-evaluate decisions they have made about permitting their children to drink in the family home, a supervised environment.

Impulsivity is a risk-factor for early adolescent alcohol use. Staff in schools and youth agencies need to be skilled in recognising where young people are likely to get into difficulties with alcohol or if they are already having difficulties. Services require back-up support and advice to enable them to intervene where appropriate.

Neurological research indicates physiological reasons why young people may be less concerned about long-term impacts of harmful health behaviours than adults. The ways in which information is communicated to young people, their families and carers is important. Consultation with young people can suggest ways of improving access to advice and support in relation to prevention, treatment and support. At all times care should be taken to ensure that information is accurate, up to date, honest and non-alarmist.

The merits of early recognition and brief interventions for adult individuals who are drinking in a harmful way are well established and data on the efficacy of this approach in younger people are promising. Interactions with health, education, social welfare and criminal justice services present opportunities for the delivery of alcohol-based interventions aiming to prevent and reduce drinking behaviours which harm young people and those around them.

4 Emerging evidence that the adolescent brain may be especially vulnerable to alcohol harms should prompt investment in further exploratory research.

This event focussed on bringing together clinical, biological, epidemiological and social scientific evidence on young people’s alcohol use and related harm. In this emerging field, future research priorities should be multi-disciplinary.

Brain imaging studies indicate that harms need to be replicated and extended in order to understand more about the likelihood of adolescent alcohol use causing lasting changes in brain structure and function. Emerging lessons suggest different impacts by sex and age; more research is therefore needed that stratifies subjects by sex and early, mid or late adolescence. It may also be useful for researchers to take account of pubertal stage of development rather than simply age. We also need to know more about the relative risks associated with the amount of alcohol consumed overall by young people and in individual sessions.

Longitudinal studies that examine large cohorts beginning in alcohol-naïve children and continuing across adolescence and into young adulthood would help us to

understand the relationships between alcohol exposure and patterns of alcohol use and long-term impacts on cognition and behaviour. Such studies would help clarify the degree to which it is alcohol exposure or trait characteristics which account for the differences observed between adolescent alcohol abusers and controls in existing studies.

The complexities of individual responses to alcohol also require closer analysis. There are several areas for further exploration, including genetic vulnerability, foetal alcohol exposure, social inequality, gender, and family history, alongside other influences such as personality, anxiety and depression.

As with adults, many young people who use alcohol also use other drugs. It would be useful to undertake investigations that explore the interplay between different substances.

More evidence is also required to establish the most effective and safest therapeutic interventions for young people with established alcohol related problems, distinguishing these from interventions that are designed for adults.

References

- 1 Scottish Government (2009) *Changing Scotland's Relationship with Alcohol: A discussion paper on our Strategic Approach*, Edinburgh.

Appendix 1: Putting young people's drinking into context

Understanding the trends: alcohol use by adolescents. National surveys of self-reported alcohol consumption provide insight into adolescent drinking in Scotland.¹

Dr. Deborah Shipton, Glasgow Centre for Population Health

At what age are young people drinking?

By the age of 13, just over half of young people in Scotland (56%) have consumed alcohol at some point. A minority (14%) self-report that they drank in the last week and 5% said they were drunk in the last week. At 15, most young people in Scotland (77%) have had at least one alcoholic drink, 34% say they drank in the last week and 19% reported getting drunk in the last week. Between 2008 and 2010 there was a slight increase in the numbers of 15-year-olds drinking alcohol in the previous week. It is not clear if this is a continuing trend or a fluctuation in the numbers. See Figure 1 below.¹

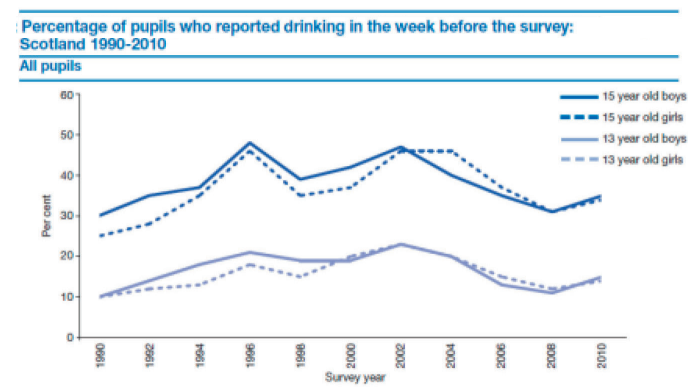


Figure 1: Percentage of pupils reporting drinking in the week before the survey: Scotland 1990-2010 (source: SALSUS 2010)¹

How much do young people drink?

In Scotland those 15-year-olds who drank consumed on average (median) 11 units of alcohol in the previous week, and 13-year-olds consumed on average 8 units. (The definition of one unit in the UK is 8 grammes or 10 millilitres of pure alcohol.²)

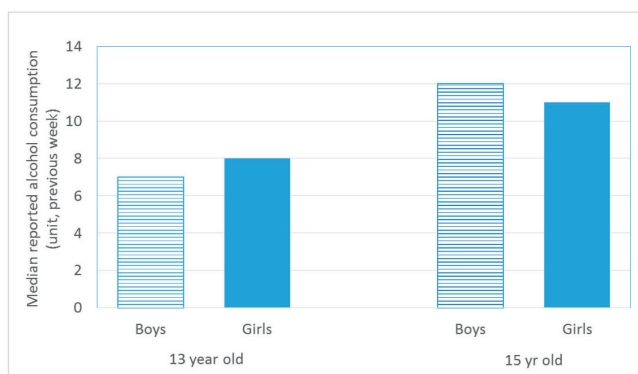


Figure 2: Median reported alcohol consumption for boys and girls in Scotland (Salsus 2010)

What type of drinks do young people consume and are there patterns by gender?

Over the past twenty years there has been a convergence in regular drinking behaviour across the sexes. The data from 1990–2002 show males at the ages of 13 and 15 drank slightly more regularly than females. Since 2008, the evidence indicates that young men and young women drink with the same frequency and consumed roughly the same amount of alcohol.

Survey findings indicate that boys mostly drink beer, larger or cider (87%) followed by spirits (52%) and alcopops (41%).¹ Girls consume stronger drinks and they are just as likely to have drunk spirits (68%) as they are beer, lager or cider (68%). They are also more likely to drink wine (40%, compared to 26% of boys) or alcopops (63%). This is reflected in the drink types that units of alcohol came from – most units consumed in the previous week came from strong beer, lager or ciders and from alcopops- see Figure 3.

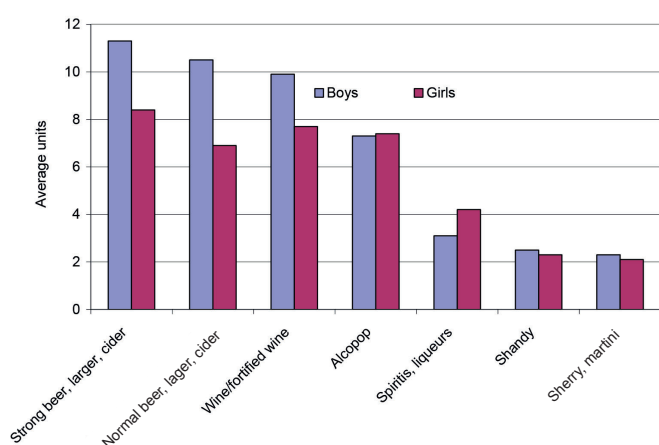


Figure 3: Average units of each type of alcohol drunk in the previous week by 15-year-olds (who had consumed that alcohol). Source: Graph created by author from Salsus 2010 data.

Which young people drank the most?

Young people who live in deprived areas are more likely to drink alcohol, drink at an earlier age, and to drink to excess. In deprived areas (as defined by the Scottish Index of Multiple Deprivation) 19% of 13-year-olds drank alcohol in the previous week compared to 11% in the least deprived areas. This relationship was stronger for young women than young men. The effects of higher alcohol consumption in areas of deprivation is likely to be compounded by inequalities which affect nutrition, exercise and emotional well-being¹.

Trends in older adolescents (16–24-year-olds)²

General consumption trends reduced between 2003 and 2011 for 16–24-year-olds. Evidence suggests the following:

- An increase in the numbers of non-drinkers and moderate drinkers (men and women who drink no more than the recommended weekly limits – 21 units for men and 14 for women).
- A reduction in number of harmful drinkers (men who consumed more than 21 units per week and women who drank more than 14 units per week) and decrease in reported mean units consumed per week (men and women).
- A reduction in the percentage of young men and women who reported heavy episodic drinking (over 8 and over 4 units on heaviest drinking day).
- A reduction in the number of reported days of alcohol drunk in the previous week (men and women).

Summary of key points:

- Just over a third of 15-year-olds in Scotland reported having consumed alcohol in the past week; on average those young people who do drink, consume an average of 11 units of alcohol per week.
- Evidence indicates that young people in Scotland are most likely to consume strong beers, lagers and ciders. Young women are more likely to drink spirits than their male counterparts.

References

- 1 Scottish Schools Adolescent Lifestyle and Substance Use Survey (SALSUS) National Report (2010): Smoking, drinking and drug use among 13 and 15 year olds in Scotland in 2010
- 2 Scottish Health Survey 2010 (Scottish Government, 2012)

Appendix 2: Understanding alcohol- related harm

The impact of adolescent drinking on health: The evidence on harms to young people from alcohol consumption.

Dr. Lesley Graham, Information Services Division, NHS National Services Scotland

What are the immediate health harms of drinking alcohol for young people?

The immediate (acute) effects of excess alcohol consumption include vomiting, injury, coma and hypothermia. Alcohol increases the risk of fatalities and injuries in young people. Alcohol is believed to have played a role in more than half of traumatic brain injuries and is linked to 80% of deaths from homicides, suicides and unintentional injuries.¹

In Scotland in 2010, of the children aged 13 and 15 that had drunk alcohol within the past year (60% of all 13 and 15 year olds), the following occurred as a result of drinking: almost one third (31%) had vomited, a minority (3%) had been admitted to hospital overnight and 2% had an injury that needed to be seen by a doctor.²

In 2007 an audit reviewed attendance at various A&E departments in Scotland by under 18 year olds, providing a profile of presentations by young people. Overall 1 in 50 (a total of 669) attendances were alcohol related and 22% of these were admitted to hospital. The median age of attendance was 16, with boys marginally more likely to attend (54%). The key medical complaint was either trauma (48%) or intoxication (42%), with self-harm identified in a minority (14%) of young people. Assault was the cause of trauma in over half of cases.³

In 2011/12 there were 3,440 general acute alcohol related hospital discharges (rather than individuals) of those under 25 years, with this group representing 9% of all alcohol related hospital discharges.

Over the three years between 2009/10 and 2011/12 there was a slight year-on-year decrease in general acute inpatient discharges for alcohol conditions in young people aged under 15 and those aged 15–19. The numbers of discharges has remained consistent for 20–24 year olds.³

In 2011/12 there were over 1,600 general acute alcohol related hospital discharges of young people under 25 years due to acute alcohol intoxication (see Figure 1).⁴

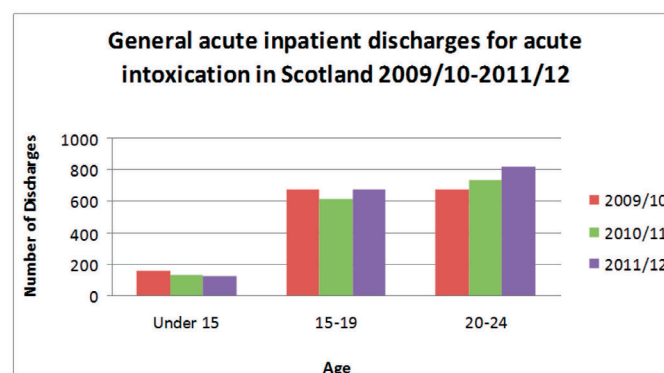


Figure 1: General acute inpatient discharges for acute intoxication in Scotland 2009/10 and 2011/12 Data analysis of SMR01 (Acute Hospital Admissions) over a three year period 2009–2012

What are the longer-term harms from alcohol consumption for young people?

Long-term (chronic) effects of excessive alcohol consumption include liver disease and alcohol dependence. In 2011/12 there were 80 hospital discharges for alcohol dependence of young people aged 20–24 and 30 for those aged 15–19-years-old.⁴ There are also links between poorer mental health and alcohol-misuse problems.¹

Alcohol-related mortality

In Scotland in 2003, 15.4% (66/429) of all deaths by 16–24-year-olds were attributable to alcohol (i.e. from conditions where alcohol is a contributory factor). Young males (17.5%) were almost twice as likely to die as a result of alcohol than young females (9.9%). For young males the main reasons for alcohol attributable death were road traffic accidents, intentional self-harm and assaults; in young females the main reasons were road traffic accidents and epilepsy.⁵

Across the population, the majority of alcohol-related mortality is due to alcoholic liver disease (ALD). Mortality rates for ALD rose between 1982 and 2002 but has followed a downward trend since then.² However, a recent study has shown that alcoholic liver disease is increasing amongst young people in parts of Scotland, particularly for young women.⁶

Alcohol and social harm

Several reviews of social harms associated with alcohol consumption by young people have discussed links between alcohol and unprotected sex, unplanned pregnancy and sexually transmitted infections.¹

What are the links between alcohol and offending?

There are strong, though complex, links between alcohol and offending. Alcohol-related crime spans a range of social harm and includes anti-social behaviour causing social nuisance; vandalism; drink driving; robbery; sexual offences; assaults and homicide. Consumption of alcohol leads to an increased risk for an individual of being both a perpetrator of violent crime and of being a victim. For example:

- Three quarters of young offenders (18–21) say they were drunk at the time of offence, compared to half of all other prisoners – suggesting that alcohol may have a greater impact for this age group.⁷

The 2009/10 Scottish Crime and Justice Survey reported that in two thirds (62%) of incidents of violent crime, victims believed their attacker was under the influence of alcohol. This varied by age with younger victims more likely to believe their attacker under the influence of alcohol (68% of 16–24-year-olds compared with 43% of over 45s) See Figure 2.⁸

A study by McKinlay et al. identified a rise in the proportion of young Scottish offenders who considered alcohol had contributed to their offending (from 48% in 1979 to 80% in 2007).⁹

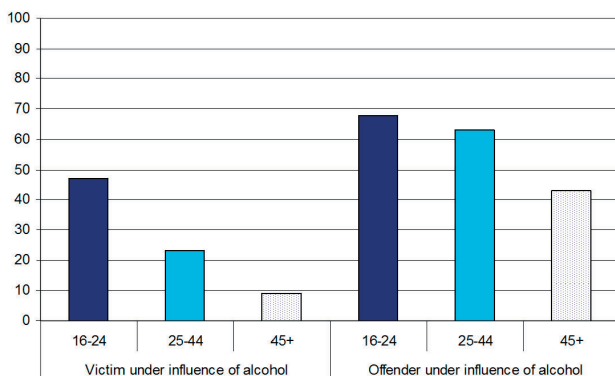


Figure 2: Proportion of violent crime where victim was under the influence of alcohol/ the victim perceived the offender to be under the influence of alcohol, by age group, 2009/10.⁸

Summary of key points

- Overall the evidence points to a considerable burden of health harm, ranging from acute medical needs (accidents and injuries) to longer-term conditions.
- Strong links between alcohol and offending are evident with data that suggest the problem has increasingly been implicated in offending in recent years.

References

- 1 *Impact of Alcohol Consumption on Young People A Systematic Review of Published Reviews* (2009). Dorothy Newbury-Birch, Janet Walker, Leah Avery, Fiona Beyer, Nicola Brown, Katherine Jackson, Catherine A Lock, Ruth McGovern and Eileen Kaner (Institute of Health and Society, Newcastle University) Eilish Gilvarry (Northumberland, Tyne and Wear Drug and Alcohol Service) Paul McArdle and Venkateswaran Ramesh (Northumberland Tyne and Wear NHS Trust) Stephen Stewart (Freeman Group of Hospitals NHS Trust) (Department of Children, Schools and Families, 2009)
- 2 *Monitoring and Evaluating Scotland's Alcohol Strategy (MESAS)* (NHS Health Scotland 2012)
- 3 *Scottish Emergency Department Alcohol Audit 2008* (ISD, 2008)
- 4 *Data analysis of SMR01 (Acute Hospital Admissions) over a three year period 2009–2012*
- 5 *Alcohol attributable mortality and morbidity: alcohol population attributable fractions for Scotland* (2009) Ian Grant, Anthea Springbett and Dr Lesley Graham (NHS National Services, 2009)
- 6 Shipton D, Whyte B, Walsh D (2013). *Alcohol-related mortality in deprived UK cities: worrying trends in young women challenge recent national downward trends*: Journal of Epidemiology and Community Health;67:805-812 doi:10.1136/jech-2013-202574.
- 7 *Scottish Prison Survey 2011 and Scottish Young Offenders Survey 2011* (Scottish Prison Service, Strategy Unit 2011)
- 8 *Scottish Crime and Justice Survey 2009/10* (Scottish Government, 2010)
- 9 *The McKinlay Report: Alcohol & Violence among Young Male Offenders (1979-2009)* (Scottish Prison Service, 2009)

Appendix 3: Speakers

Dr Iroise Dumontheil, Department of Psychological Sciences, Birkbeck, University of London:

Dr Dumontheil is a lecturer at the Department of Psychological Sciences at Birkbeck, University of London, and a member of the Centre for Educational Neuroscience (CEN) and of the Centre for Brain and Cognitive Development (CBCD).

Dr Gordon Fernie, Division of Applied Medicine (Psychiatry), University of Aberdeen:

Gordon Fernie works in the Division of Applied Medicine at the University of Aberdeen. He is currently running a CSO-funded clinical trial investigating ketamine as the anaesthetic for electro-convulsive therapy (ECT).

Dr Lesley Graham, Information Services Division, National Services Scotland:

Lesley Graham is an Associate Specialist in Public Health at NHS National Services Scotland.

Professor Anne Lingford-Hughes, Centre for Neuropsychopharmacology, Imperial College London

Professor Anne Lingford-Hughes is Professor of Addiction Biology at Imperial College and Consultant Psychiatrist with a particular interest in pharmacological treatments of alcohol problems and comorbidity.

Professor Gunter Schumann MD, Chair in Biological Psychiatry, King's College, London

Gunter Schumann is Professor of Biological Psychiatry at the Social, Genetic and Developmental Psychiatry Centre, and Honorary Consultant at South London and Maudsley NHS Foundation Trust where he leads the challenging behaviour programme of the National Psychosis Unit.

Dr Deborah Shipton, Glasgow Centre for Population Health

Deborah Shipton is a Public Health Research Specialist at the Glasgow Centre for Population Health.

Dr Killian Welch, Robert Ferguson Unit, Astley Ainslie Hospital, Edinburgh

Dr Killian Welch is a Consultant Neuropsychiatrist working in the Neurorehabilitation units of the Astley Ainslie Hospital and the Department of Clinical Neurosciences in Edinburgh.

Appendix 4: Workshop participants

Dr Anne-Marie Barry
Policy Officer, SHAAP

Eric Carlin
Director, SHAAP

Dr Catherine Chiang
Consultant in Public Health
Medicine

Prof Jonathan Chick
Queen Margaret University

Alison Ferguson
Scottish Government

Dr Evelyn Gillan
Alcohol Focus Scotland

Donald Henderson
Scottish Government

Prof Stephen Lawrie
University of Edinburgh

Sophie Macken
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Gina Martin
PhD Student CAHRU
St Andrews University

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Consultant Psychiatrist in
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Dr Garth Reid
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Lorraine Simpson
The Lines Between (note
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