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From the editor



Dr Dominique Florin

MCA Annual General Meeting

In November, the MCA's highly successful AGM took place at the Royal College of Physicians. The Max Glatt Memorial Lecture was given by Professor Sir Ian Gilmore, with customary brilliance. The title was 'The UK's troubled relationship with alcohol – a challenge for policy makers', and Professor Gilmore reviewed the trends in total consumption in the UK since the early 1900s, before comparing UK consumption with that in Europe and the wider world. It was clear that, within Europe, both the increase in overall consumption and our pattern of drinking ('bingeing') are matters of concern. Both have led to rising pressure on NHS resources and increased fatalities, the latter particularly evident in the fivefold increase in liver deaths over the last 30 years. Professor Gilmore put the potentially beneficial effects of moderate consumption – through reduction in ischaemic heart disease – into context, emphasising that any benefit was age-related, and did not outweigh the risks of even moderate consumption until mid-life. The main drivers of the changes in UK drinking patterns were then reviewed: price, availability and marketing; and the importance of tackling these through some form of regulatory framework. Differences in governmental approaches within the UK were noted; the current coalition government approach to alcohol suggests that local solutions rather than central regulation are likely to be more in favour. Following this lecture, Dr Brian Hore gave a vote

of thanks to Sir Ian Gilmore, and Professor Peter Brunt then presented the speaker with the Max Glatt Memorial Medal. We were particularly honoured that Mrs Glatt, widow of Dr Max Glatt, was able to be present for this event.

Seminar on dealing with fetal alcohol spectrum disorder

In the afternoon following the MCA's AGM, the MCA Seminar was held, on fetal alcohol spectrum disorder (FASD). This was introduced and chaired by a great friend of the MCA, Professor Moira Plant, who has devoted her professional career to the study of fetal alcohol exposure. We heard two excellent presentations, firstly by Dr Raja Mukherjee, a psychiatrist who runs a specialist service in Surrey for children and families affected by FASD, and a second by Dr Maggie Watts, a public health physician from Ayrshire who has been working for 20 years in this area. Articles based on both of these talks are reproduced in this issue of *Alcoholis*. The questions and discussions which followed the talks were very lively, and could have gone on for much longer. All three of the panel members conveyed the misery of this diagnosis, but also the need to make it, in order that the support can be accessed, which can so improve the experience of patients and their families. It is timely that the topic for the Frowen Essay Prize for this year, advertised in this issue of *Alcoholis*, also concerns fetal alcohol exposure.

Progress on minimum unit pricing

The MCA is aligned with all the other authoritative medical voices in supporting the introduction of a minimum unit price (MUP) for alcohol, ideally of 50 pence per unit. The long awaited vote on MUP in Scotland took place in November and the measure was removed from the bill. This is disappointing but not unexpected. The feeling from colleagues in Scotland was that, despite this result, progress has been made in gaining wider acceptance of the importance of alcohol as a health problem. Meanwhile in Manchester, the introduction of a MUP by-law of 50 pence per unit is being considered. Despite the government's

From the editor *continued...*

resistance to this idea nationally, there seems to be some central support for this local initiative. Most recently, the coalition government has proposed tax changes and a ban on below-cost selling. The consensus among many alcohol organisations is that these changes are much too little. The tax increase only addresses super-strength beer, and would affect less than 0.5% of total alcohol sales. Importantly, cider is excluded. The suggested ban on below-cost selling is likewise anticipated to have almost no effect, being equivalent to an MUP of only 18–26 pence per unit. This would be equivalent to banning the sale of wine at less than £2 per bottle.

In the journals

Two recent articles relevant to the pattern of heavy drinking

in the UK may be of particular interest to our readers. In *Alcohol and Alcoholism*, Simon Moore writes about the possibility of substitution of alcohol with other substances, should alcohol become less accessible, for instance due to price or tax increases.¹ This piece is accompanied by an editorial and some commentary pieces, and makes for a fascinating debate. In the *BMJ*, a group from France suggest that a binge-drinking pattern may account for higher rates of ischaemic heart disease in Ireland, compared with France.²

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What do we know about fetal alcohol syndrome in Scotland?

Dr Maggie Watts, consultant in public health medicine, Alisa Hospital, Ayrshire

The adverse effects of alcohol have been known for centuries, and writers have also recorded comments about alcohol in pregnancy, according to the social and philosophical mores of the age. A specific association between alcohol and damage to the fetus, producing a characteristic set of abnormalities, was first described in a medical journal by a French paediatrician, Paul Lemoine, in 1968,¹ and subsequently in English journals by Jones *et al* in 1973.² These articles described babies and children damaged by alcohol *in utero*, such that it produced a characteristic set of anomalies encompassing growth retardation; characteristic faces with smooth

philtrum, thin upper lip vermilion, and narrowed palpebral fissures; and neurodevelopmental delay. This represents fetal alcohol syndrome (FAS), the most easily recognisable form of fetal alcohol damage. However, ingestion of alcohol during pregnancy can cause a spectrum of disorders (the fetal alcohol spectrum disorder (FASD) group of conditions), which are characterised by growth failure and neurological deficits.

The incidence and prevalence of FAS varies widely around the world, according to the type of study and the population examined. Those in larger, more diverse populations

in terms of alcohol consumption, ethnicity and demography, tend towards lower rates (0.1–0.67 in every 1,000), with surveillance studies using non-specialist medical staff giving rates of 0.37–0.67 in every 1,000.³ Alcohol in Scotland is ubiquitous, and the Scots people are notorious for their drinking cultures. In 2009, sales of alcohol reached 50.9 million litres of absolute alcohol – enough sold for every person over the age of 18 years to consume 1,227 units of pure alcohol, an average of 23.6 units per person per week.⁴ While 21% of Scottish women aged 16–34 report drinking no alcohol, 29% of women aged 16–34, and 24% of women aged 25–34, report drinking more than 14 units a week, with 28% having more than six units of alcohol on their heaviest drinking day.⁵ Drinking behaviour during pregnancy has not been studied extensively in Scotland. Self-reporting in population surveys indicate prevalence of alcohol use at 25–50%.⁶ With female drinking at such levels, it is perhaps surprising that more attention is not given to FASD in Scotland.

Following the publication of papers by Jones *et al* in 1973 and 1974, there was some interest in Scotland. In the west of the country, Beattie *et al* produced a paper identifying 40 children with the features of FAS, and stated that ‘these

Table 1 Recording of FAS in data systems: Scotland, 1998–2003

Data source	1998	1999	2000	2001	2002	2003
Mortality records, Registrar General For Scotland	0	0	0	0	0	–
SMR 1 – hospital discharges	18	8	10	12	14	12
SMR 11 – neonatal unit discharges	3	2	6	3	–	–
Preschool child health surveillance*	–	–	3	2	–	–
Continuous Morbidity Records (GP data system)†	–	–	0	0	3	–

– data unavailable; *10 of 15 health boards; † coverage 8% general population
SMR = Scottish Morbidity Records

findings provide clear evidence that in the west of Scotland maternal alcohol abuse during pregnancy is a significant cause of morbidity and mortality in children.⁷ In the east of Scotland, Plant was carrying out a prospective cohort study designed to establish whether or not birth abnormalities were associated with self-reported drinking rates.⁸ Whilst Plant did not identify any cases of FAS, the study did show a level of alcohol-related birth abnormalities of 1%.

Since that time, there has been very little published on FAS or FASD in Scotland. Using the rates from surveillance,⁹ it is estimated that 20–36 babies are born each year in Scotland with FAS, and up to 10 times more with FASD. This would indicate 400 or more children under 16 years of age with FAS, and considerably more living with FASD.

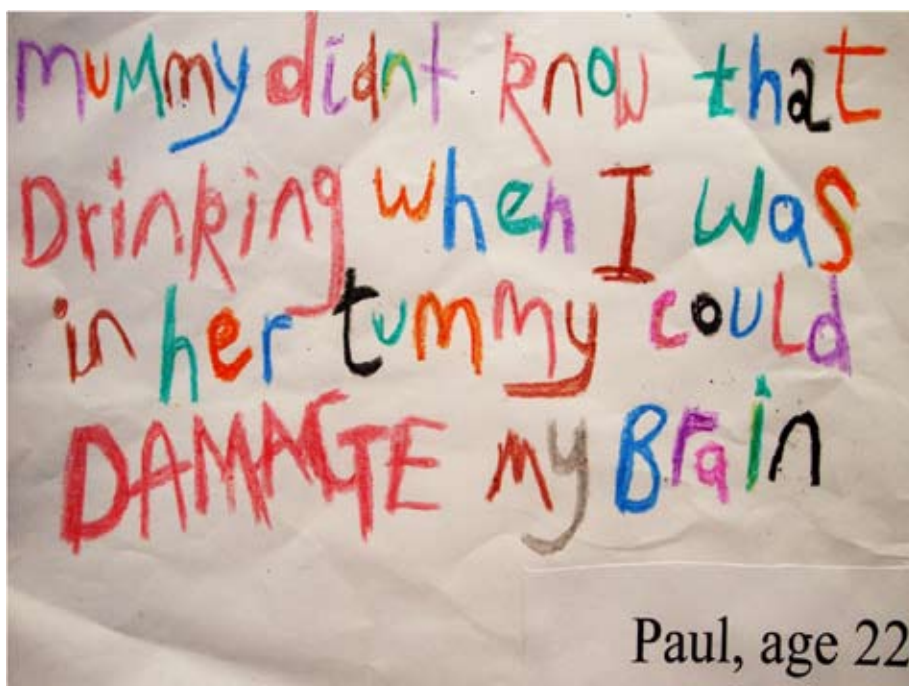
A study undertaken in 2004 identified the occurrence of FAS as recorded in the Scottish health system (Table 1).¹⁰ Since FAS is not a condition that requires children to be hospitalised, the national recording systems for hospital discharges (Scottish Morbidity Record (SMR) 01) and for maternity units (SMR 11) show very low numbers. No children died with a diagnosis of FAS, and the preschool surveillance and GP continuous morbidity recording systems recorded very low numbers.

This information, coupled with a local survey of professional staff in health, ►

Continuing with the theme of fetal alcohol syndrome, here we feature another two entries, from medical students, for the National Alcohol Awareness Day poster competition.

'Mummy didn't know' (top) is by Matthew Baldwin, Ryan Stevens, Sam Haines and Pete Sugden.

'Mummy I don't want your hangover' (bottom) is by Matt Angilley, Holly Pope, Caroline Bracchi, Sophie McGlade and Richard Gardland.



education and social work, indicated an awareness of the diagnosis and a recognition that underdiagnosis could be a problem, but little experience in either making it or working with children with FAS. However, professionals did consider that alcohol in pregnancy could be a major issue with harmful consequences for the fetus.

A postal survey study in 2007 investigated the experience, knowledge, attitudes and diagnosis of FAS amongst paediatricians across Scotland.¹¹ This indicated that two-thirds of respondents had diagnosed FAS, but less than a quarter reported confidence in making the diagnosis. Knowledge of the optimal age for diagnosis of FAS, length of experience, and quality of training received were significantly associated with having made a diagnosis of FAS in the past. Confidence in diagnosing FAS was significantly associated with the

frequency of enquiry of alcohol use, receipt of high-quality training, and beliefs in the benefits of early diagnosis. The results of these more recent studies indicate that further research is required into the prevention, detection, diagnosis and management of FASD in Scotland. High-quality training programmes may improve the diagnosis of FAS, but this needs to be coupled with improvements in case ascertainment, supported and coordinated surveillance, effective recording systems, and non-stigmatising approaches to families and affected children.

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Overview of diagnostic issues and behavioural difficulties in people affected by fetal alcohol spectrum disorder

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Introduction

Fetal alcohol spectrum disorder (FASD) is an umbrella term that denotes the range of outcomes seen in people exposed to alcohol whilst in the womb. This can vary greatly in terms of presentation and severity. While much of the early work was conducted in people who were most clearly affected, there has been an increasing recognition of people exposed to alcohol, but who do not show obvious initial characteristics. Those without clear neurological deficits or facial features can still have significant behavioural, functional and emotional conditions that require lifelong input and support. The term fetal alcohol syndrome was first coined by Smith and Jones in 1973; they presented a case report of eight children in *The Lancet*.¹ Despite this, the first actual reports in the scientific literature of alcohol consumption affecting children, was by Lemoine in

1967.² He reported, with his midwifery colleague, 127 cases of affected individuals. Unfortunately, as this was not in an English language journal, it did not come to recognition until later on.

Diagnosis

For almost 40 years now, there has been a great deal of work on the condition. In 1996, the Institute of Medicine (IOM) in America – broadly equivalent to our medical royal colleges – came together to define a set of criteria by which the condition should be diagnosed. Despite this, there remain confusion and disagreement over how to diagnose FASD, especially in the absence of facial features. Box 1 highlights the core facial features and the wider diagnostic criteria set out by the IOM in 1996.

What has become increasingly clear, and has also led to the most controversy, is the fact that the most recognisable

manifestation of this syndrome, full fetal alcohol syndrome (FAS), represents less than 10% of those affected. The majority of exposed individuals sustain damage to the brain, without facial features

Box 1 Facial features and diagnostic criteria defined by IOM

Core facial features

- 1 Short palpebral fissures
- 2 Flattened elongated philtrum
- 3 Thinner upper lip vermillion

Diagnostic labels

- 1 Fetal alcohol syndrome alcohol known
- 2 Fetal alcohol syndrome alcohol unknown
- 3 Partial fetal alcohol syndrome
- 4 Alcohol-related birth defect
- 5 Alcohol-related neurodevelopmental disorder (ARND)

necessarily being affected. The face is affected in a window of opportunity, at around weeks 6–8 of gestation. It also requires heavy alcohol exposure to cause significant issues. The literature would suggest that only around 40% of women who drink heavily during pregnancy will have children with recognisable facial features. This period of vulnerability is in contrast to that which affects the brain, which begins to develop from around day 18 in gestation, and develops throughout pregnancy. In animal models, different timing of alcohol exposure has been shown to cause different types of damage to the fetus. These relate specifically to apoptotic damage, influencing gene expression, cell signalling, cell adhesion and scaffolding, all of which are damaged by smaller amounts of alcohol than those required to cause the facial effects. The brain is thus vulnerable throughout pregnancy.

Because of the uncertainty in making the wider diagnosis, attempts to better classify the full range of conditions have been developed. These include tools such as the four-digit assessment tool. The differences and similarities between the diagnostic systems are shown in Table 1. What is interesting is that there are many similarities, but also differences. These relate to the desire to maximise sensitivity and specificity of diagnosis. The ongoing

disagreement about making an ARND diagnosis, and what should constitute this, has perpetuated the problem. These discrepancies continue to affect the ability of families to get appropriate help.

Epidemiology

The rates of FASD in the community vary by the population studied. Early experiences suggested that the condition was one confined to the Native American population. This was later found not to be the case. FASD cases have been found in all populations and all socioeconomic backgrounds. With increasing understanding, the rates of FASD have also been revised. Recent studies in different parts of the world have found differing levels. Two broad methods have been used to derive estimates of prevalence, namely passive and active surveillance models. The passive surveillance models offered some insights, but have been shown to underreport considerably the rates of FASD. Active surveillance research has increasingly been conducted in several countries. Whilst some issues exist about the design, they remain the best epidemiological studies in the field conducted to date.

Rates reported vary between 3 per 1,000 (population study in Lazio, Italy) and 85 per 1,000 (population study in Cape Town, South Africa,

including a high-risk alcohol group) for full FAS. This was further shown to represent only 10% of the whole group in these respective studies. While the figure in the UK remains unknown, the international rates quoted by the Centre for Disease Control (CDC) suggest that 1–2% of the US population may be affected by alcohol, and high-risk populations exponentially so.

Behavioural difficulties

Behavioural conditions in people with FASD are complicated, as they do not always present in consistent ways. That is to say, standard diagnostic profiles using the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* or the *International Classification of Diseases* are not always neatly met. The underlying behavioural profile has, however, been reasonably well defined. It is understanding the relationship between the phenomenological diagnostic criteria and the underlying function, that can help to guide both areas for assessment and later management.

Children and adults with FASD classically have a very mixed profile of cognitive functioning, with deficits in areas such as working memory, executive function, and processing speed. This has been shown in numerous studies of neurocognitive functioning in people with FASD. Importantly, the profiles have been shown not to be consistent with expected patterns compared to their IQ, or by their FASD diagnosis. Damage has been shown in the myelination of white matter of the brain, the connections between brain regions, and the volumetric size of brain regions compared with normal controls. These have been shown on function and volumetric MRI scans.

These deficits are further affected by time pressure and the need for accuracy. The implication is that you will see an individual who will act and not necessarily think of the consequences of their behaviours where rapid processing and linking of information is required. High-pressure situations make this worse. This description is consistent with the common complaints made by carers about individuals

Table 1 Different diagnostic classification systems currently in use today: similarities and differences

	Centre for Disease Control	Institute of Medicine	Canadian	Four-digit
Face	10th percentile PFL and rank 4/5 on lip philtrum	10th percentile PFL and rank 4/5 on lip philtrum	3rd percentile PFL and rank 4/5 on lip philtrum	3rd percentile PFL and rank 4/5 on lip philtrum
Growth	Pre- / post-natal growth below 10th percentile	Pre- / post-natal growth below 10th percentile	Pre- / post-natal growth below 10th percentile	Pre- / post-natal growth below 10th percentile
Neurological	1 out of several brain parameters including OFC <10%, CNS deficits	1 out of 1 brain parameters including OFC <10%, CNS deficits or abnormal structure	3+ soft hard neurological signs	1 out of several brain parameters including OFC <10%, CNS deficits
Alcohol	Cofirmed or unknown	Confirmed to be excessive or unknown	Confirmed or unknown	Confirmed or unknown

OFC = orbitofrontal circumference; PFL = palpebral fissure length; CNS = central nervous system

Table 2 Secondary disabilities found in a cohort of over 300 individuals in a 21-year cohort with FASD (Streissguth)³

Disability	%
Psychiatric problem	90
Disrupted school experience	60
Trouble with the law	60
Confinement	50
Inappropriate sexual behaviour	50
Alcohol/drug problems	30

with FASD. Namely, that they cannot stop themselves from acting and that they fail to learn from consequence.

The latter part is also affected by deficits in working memory. Animal literature has shown damage to the neural pathways that consolidate memory. CA1 and CA3 fibres have been shown to be damaged, leading to problems in working memory and the consolidation pathways. The behavioural presentation is of someone who cannot remember what has just been told to them. This is

not to say they cannot learn, only that it can take longer, and that other methods using multi-source approaches may be required. Often, the fact that information can be presented from the distant past (long-term memory) confuses the picture. The impression, then, is of deliberate non-compliance, especially to those unfamiliar with the condition.

Probably the single area of greatest difficulty lies in executive functioning. Deficits and damage to frontal lobe functioning, through its connections to other areas, cause the most problems faced by this group. This often presents as a struggle with inhibitory control and social understanding. At its worst this leads to the individual meeting diagnostic criteria for attention deficit hyperactivity disorder (ADHD) or autism, but to lesser degrees of difficulty, simply lead to problems in their daily lives and potential over-expectation by society.

Management approaches

The management of this group begins with prevention, but the public health strategies employed will only ever have a limited effect. For those individuals

affected, only by first assessing in detail both the phenotype and the measurable endophenotype, can a plan of management be formulated. This assessment will include careful picking out of the effects of early environmental experiences, as well as understanding their relationships to inherent biological vulnerability. Often, simply by replacing or supporting the areas of deficit and providing consistency, the individual will improve. The earlier an individual accepts their diagnosis, the more likely a good outcome is. For those who do not get support, or who fail to accept their diagnosis, the outcomes have been shown to be poor. Over 90% will have secondary mental health problems, and many struggle with wide areas of function.

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Michael Frowen Memorial Essay Prize 2011

Awarded for a paper not exceeding 3,000 words (excluding references and title)

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