Child: care, health and development
Original Article
doi:10.1111/cch.12017

Diagnostic nomenclature for foetal alcohol spectrum disorders: the continuing challenge of causality

A. R. Miller*†‡

*Division of Developmental Pediatrics, Department of Pediatrics, University of British Columbia, Vancouver, BC, Canada
†Developmental Neurosciences & Child Health, Child & Family Research Institute, Vancouver, BC, Canada, and
‡Sunny Hill Health Centre for Children, Vancouver, BC, Canada

Accepted for publication 16 September 2012

Keywords
alcohol-related neurodevelopmental disorder, causality, foetal alcohol spectrum disorder(s), prenatal alcohol exposure, teratogenesis

Correspondence:
Anton R. Miller,
Department of Pediatrics,
University of British Columbia, F515-4480 Oak Street, Vancouver, BC,
Canada V6H 3V4
E-mail: amiller@cw.bc.ca

Abstract

Prenatal alcohol exposure is a risk factor for neurologically based cognitive and adaptive disability. Diagnostic nomenclature for prenatally exposed children with cognitive and adaptive disability who lack features for foetal alcohol syndrome (FAS) or partial FAS includes the terms alcohol-related neurodevelopmental disorder (ARND) and foetal alcohol spectrum disorder(s) (FASD). Although these terms are now widely used, this paper argues that both are problematic. ARND is flawed by unjustifiably turning a risk factor into a causal factor and shrouding the result in terminological ambiguity, while FASD is not appropriate as a clinical label, and its use as a proxy for ARND deflects critical attention from the causal inferencing that is integral to diagnosing children with an alcohol-related teratogenic condition. Existing nomenclature is at odds with logical and evidence-based diagnosing and also has implications for interpretation of epidemiological data. Diagnostic nomenclature that is not tightly linked to causal inference is preferable at the present stage of this field's development.

Introduction

The way we name things shapes how we think about them and count them. This paper analyses problems in the diagnostic nomenclature for children with cognitive–adaptive disabilities who were exposed to alcohol prenatally but lack physical stigmata for an ICD-10 diagnosis of dysmorphic foetal alcohol syndrome (FAS). Diagnostic nomenclature for this clinical scenario has evolved over time. Terms such as (possible) foetal alcohol effects (Aase *et al.* 1995) have been replaced by the diagnosis alcohol-related neurodevelopmental disorder (ARND), defined as 'a complex pattern of behavioral or cognitive impairment . . . in combination with a history of significant prenatal alcohol exposure' (Stratton *et al.* 1996). Children with ARND may also be described as having foetal alcohol spectrum disorder (FASD), an umbrella term to describe 'the range of effects, including physical, mental, behavioral and/or learning disabilities, that can occur in an individual whose mother drank alcohol during pregnancy' (Bertrand *et al.* 2004). Despite their pragmatic usefulness, ARND and FASD confront us with significant semantic, epistemic and scope-of-usage problems.

Alcohol-related neurodevelopmental disorder: semantic and epistemic issues

One limitation in ARND is that the term is inherently ambiguous. Literally, it refers to a neurodevelopmental disorder that is related, in some non-specific way, to prenatal alcohol exposure (PAE). However, the underlying intent is to indicate a causal relationship, as evident from the way the term is currently used, and from a general appreciation that non-causal associations

are of limited relevance in medicine. This raises a more fundamental limitation, which is epistemic. ARND is a diagnostic hybrid of phenotypic–descriptive and aetiological elements, within which PAE assumes the status of a causal factor. The problem is that, on the basis of current knowledge and logical principles, we are able only to designate PAE as a risk factor, and not as a causal factor.

Attempting to diagnose ARND fails to take account of the crucial difference between potential and retrodictive causal propositions in medicine, represented respectively by the questions '*Can* PAE cause neurodisability?' and '*Did* PAE cause neurodisability in this child?' (Kramer & Lane 1992). The former requires epidemiological evidence that PAE is associated with neurological harm, a proposition substantiated by a diverse and convincing body of research. The latter, by contrast, requires us to consider whether the outcome that occurred in a particular patient would have occurred if there had been no exposure to alcohol prenatally. A retrodictive causal proposition in the case of a prenatally exposed child presenting with impairment in cognitive, adaptive and behavioural functioning, but no somatic features of FAS, would be convincing if a high degree of specificity existed in the exposure–outcome relationship. The evidence for such specificity is weak, however. The clinical presentation of prenatally exposed children ranges from significant disability to no detectable abnormalities (Astley 2010), related in part to the multitude of factors known to modulate the phenotypic expression of alcohol's teratogenic potential (Abel 2006; Gemma *et al.* 2007; Shankar *et al.* 2007). And despite general agreement that an array of cognitive and executive function deficits, language impairments, social difficulties and poor adaptive skills are common among children putatively affected by alcohol as a teratogen, identification of a specific phenotype remains elusive (Mattson *et al.* 2011; Pei *et al.* 2011). Conversely, although a complex profile of impairment is considered a hallmark of neurodisability due to PAE, complex profiles are part of the fabric of childhood neurodevelopmental disorders and disabilities in general (Frith 2001; Bishop & Rutter 2009). Finally, other risk factors such as genetic susceptibility (Autti-Ramo *et al.* 2006), prenatal exposure to other substances and to stress (Guerrini *et al.* 2007), and the effects of early-life adversities, including neglectful and chaotic parenting styles (Lupien *et al.* 2009), are encountered fairly frequently among exposed children presenting with cognitive–adaptive and behavioural disability. The originators of the term ARND themselves acknowledged uncertainty as to whether the cognitive and behavioural features in ARND are caused by PAE or other possible factors (Stratton *et al.* 1996). A diagnosis of ARND posits a causal role for PAE, but the difficulties inherent in formulating a convincing retrodictive causal proposition around PAE undermine the credibility of ARND as a valid medical diagnosis.

A number of adverse consequences may result from use of a diagnosis that purports to be specific as to aetiology but which is more accurately formulated as a causal hypothesis. Such a diagnosis may be potentially misleading for diagnosed individuals, and may contribute to population level prevalence data that are ambiguous and difficult to interpret. In addition, ARND is a child health outcome that is mediated by, and attributable to, maternal behaviour. Careful consideration needs to be given to the ethical and even legal implications of making such a diagnosis in the face of doubts about the causal inference on which the diagnosis is based. Diagnosis of children's developmental disorders remains largely at the level of phenotypic descriptions (for example, infantile autism) and sometimes at the level of pathophysiological explanation (e.g. fragile X syndrome). A diagnosis that both describes and explains a child's developmental presentation is highly desirable, but the quest to attain this cannot overreach the bounds of logical inferencing and available evidence. An aetiology remains undetermined for many children with cognitive and adaptive disabilities (Shevell 2008), although newer genetic technologies are reducing the size of this group (Gropman & Batshaw 2010). Given this context, a history of exposure to alcohol prenatally cannot be taken to constitute an explanation for cognitive and adaptive disability in a child just because no alternative diagnosis is readily apparent. This is particularly true with low levels of alcohol exposure, given emerging evidence that, at least among younger children, adverse neurocognitive effects may not be demonstrable (Falgreen Eriksen *et al.* 2012; Skogerbø *et al.* 2012).

Foetal alcohol spectrum disorder: epistemic aspects and inappropriate usage

The expert group responsible for the consensus definition of FASD noted that the term was not intended to be used as a clinical diagnosis, and that consensus had not yet been reached regarding evidence-based diagnostic criteria for any prenatal alcohol-related condition other than FAS (Bertrand *et al.* 2004). Between 2005 and 2011, use of the term FASD increased more than threefold based on articles cited in PubMed, along with a tendency to indiscriminate and inappropriate use. Reports on the overall 'prevalence of FASD' (up to 5% among younger school children in the USA and some Western European countries) (May *et al.* 2009) and the now

widespread practice of referring to 'children with FASD' in research studies, policy documents and advocacy materials insidiously create the impression that FASD is actually a health condition that can be diagnosed and counted, as for children with cerebral palsy or autism. But whereas diagnostic criteria exist for cerebral palsy and autism, there are none for FASD. This is fitting, since FASD technically refers to a range of effects, was never intended as a clinical term to be applied to people and lacks the precise boundaries required of a clinical or diagnostic identifier. FASD has nevertheless become increasingly used as a convenient term to cover all varieties of clinical morbidity following PAE, including ARND which accounts for a large segment of the foetal alcohol 'spectrum' (Chudley 2008; Clarren & Lutke 2008; Astley 2010). This shift in usage has tended to camouflage the shortcomings and limitations of ARND as a diagnostic term, without resolving the underlying issue of causality.

A case study of the nuanced but intractable nature of this dilemma is the treatment of causality in the 4-Digit Diagnostic Code for FASD (4-Digit Code) (Astley 2004). The 4-Digit Code purports to diagnose the full spectrum of outcomes observed in individuals with PAE (the definition of FASD), while at the same time avoiding the problem of diagnoses that attribute a causal role to alcohol (Astley 2004). Diagnostic terms in the 4-Digit Code are constructed through combining ordinally coded information about a patient's alcohol exposure status with clinical data from three key phenotypic domains of FAS (growth deficiency, facial features and neurological or neuropsychological dysfunction). While it is true that terms such as 'neurodevelopmental disorder, alcohol exposed' do not assert a causal role for alcohol exposure, a causal link is strongly suggested through the intimate linkage of a descriptive–phenotypic element with one particular risk factor. More critically, existing definitions of FASD typically refer to 'a range of effects or outcomes . . . that occur in persons prenatally exposed to alcohol'. Since the words 'effects' and 'outcomes' are semantically linked to the notion of causality, it is debatable whether FASD can conceivably be 'diagnosed' without implicating a causal role for alcohol exposure. At a conceptual level, therefore, a tool purporting to diagnose persons with a range of effects and outcomes following PAE cannot evade the thorny problem of causal inferencing.

The situation is further complicated at the practical or operational level. The 4-Digit Code protocols point out that the characteristics of persons who were exposed to alcohol prenatally are not specific to, or necessarily caused by, that exposure, and clinicians are directed to seek and document 'all other adverse prenatal and postnatal exposures and events . . . that must be taken into consideration when deriving a diagnosis and intervention plan' (Astley 2004, pp. 3–4). These directives are totally appropriate in themselves, but three kinds of problems arise in the downstream translation and deployment of 4-Digit Code data. The first is that although additional factors might have been identified for patients within clinical and research populations, with a few exceptions (Coggins *et al*. 2007), this information remains unreported and unanalysed in research studies (Franklin *et al*. 2008; Rasmussen & Bisanz 2009); it is also unlikely to be sought by agencies responsible for public health statistics. The effect of this is to highlight PAE as the risk factor above all others, insidiously reinforcing the PAE's role as causal, rather than risk, factor, and possibly even as *the* causal factor. Second, in clinics and studies that use 4-Digit Coding, children with 'neurodevelopmental disorder, alcohol exposed', a putatively non-casual term and one which can be used with any level of documented alcohol exposure, emerge for purposes of analysis and discussion as 'children with FASD', and hence subject to the issues of semantics and causality discussed above. Finally, recommendations have been made and methods proposed to 'map' 4-Digit Codes to the Institute of Medicine schema for alcohol-related medical diagnoses, including ARND (Chudley *et al*. 2005). Wider acceptance and implementation of this practice can only serve to further blur the distinction between the non-causal 4-Digit codes, on one hand, and medical diagnoses that imply causality, on the other.

Nomenclature for alcohol-related teratogenesis: where to from here?

In this clash of semantic and epistemic considerations with clinical pragmatism, various arguments may be advanced as to why it is important, perhaps necessary, to retain ARND as a diagnosis. These arguments include the desirability and utility of a diagnosis that is both phenotypically and aetiologically informative, the need for a 'medical' diagnosis to ensure access to rehabilitative and supportive services, and its role in case-finding towards ensuring prevention of alcohol-related teratogenesis in subsequent pregnancies. Are there alternatives to ARND, and to FASD when this is used as a clinical proxy term for ARND, that are capable of addressing these concerns, but also the concern about using diagnostic terms founded on an unsupportable retrodictive causal proposition?

There is no easy solution, but an approach that decouples the phenotypic–descriptive and aetiological elements in diagnosing these children has much to recommend it. The phenotypic–

descriptive component could be covered by a term such as complex neurodevelopmental disorder, or neurodevelopmental spectrum disorder (Baca *et al*. 2011). The former is used only informally, the latter is newly minted and both lack clear definitions. However, with proper attention to definition development and consensus building around operational criteria, these terms could become an indispensible part of the diagnostic nomenclature for child developmental–behavioural disorders alongside established diagnoses such as autism or intellectual disability. Their usefulness would extend to the full range of developmentally complex children referred to specialized facilities for assessment and diagnosis, whether prenatally exposed or not, given the overlap in neuropsychological characteristics between these populations. Furthermore, because the proposed terms clearly indicate an underlying neurological basis for a child's difficulties, they can be instrumental when eligibility for services and supports requires some kind of 'medical' diagnosis.

The 'aetiological' component would be covered by a parallel process involving a more comprehensive process of identification and coding of risk factors for developmental dysfunction among children. A model for this approach is the coding of exposure 'Z-codes' in ICD-10-CA, an enhanced version of the International Classification of Diseases – 10th Edition developed for use in Canada, and which allows for coding of risk factors to health 'such as occupational and environmental factors, lifestyle and psycho-social circumstances' (Canadian Institute for Health Information 2012). To generate a list comprising the most salient risk factors for use within children's developmental and behavioural health would require an investment of effort, but would have enormous downstream usefulness as an epidemiological tool if widely implemented.

In the clinical setting, a child's final diagnostic formulation would consist of the phenotypic–descriptive term along with a statement of relevant risk factors. In this way, PAE is flagged for clinical purposes (such as follow-up aimed at recurrence prevention) as well as for administrative data collection. When a child is concluded to have complex neurodevelopmental disorder in the descriptive–phenotypic 'column' and PAE in the risk factors 'column', the probability of causality could be addressed on a case-by-case basis by multidisciplinary teams with specialized expertise in assessment of such children (Chudley *et al*. 2005; Bertrand & Dang 2012).

The approach outlined here reflects knowable clinical details while avoiding the pitfalls of a hybrid diagnosis based on the *post hoc ergo propter hoc* fallacy (following this therefore because of this). At the same time, it avoids the downstream accumulation of ambiguous and suspect epidemiological data linked to the terms ARND and FASD, and avoids terms that directly connote maternal culpability. It also obviates concerns that abandoning the current clinical nomenclature would hinder ongoing efforts aimed at FASD prevention in the population as a whole. It is relevant here to consider the success enjoyed by public health campaigns to reduce the incidence of lung cancer and melanoma without the need to diagnose individual patients with smoking-related or sun exposure-related cancers.

What are the implications of this approach for the term FASD? FASD has emerged as a potent concept that is highly relevant to pubic health, and should be continued to be deployed in that context. If it were to continue to be used as a clinical term, however ('children with FASD'), then a wide-ranging and public redefinition of FASD is needed, which explicitly acknowledges that FASD comprises clinical impairments *following*, but not necessarily *caused by*, PAE, and would entail replacement of words such as 'outcomes' and 'effects'.

This alternative nomenclature may only be required for as long as it takes to strengthen the evidence base in relation to causal relationships in behavioural phenotypes and specificity in the exposure–outcome relationship for PAE. Research strategies that may be helpful in this quest include wider exploration and use of the statistical parameter, aetiological fraction among the exposed, which estimates the relative risk of the outcome in exposed vs. non-exposed individuals, enabling us to quantify the likelihood that a particular behavioural or cognitive characteristic is attributable to exposure to a risk factor (Kramer 1988). In addition, studies that compare characteristics of exposed and non-exposed children, all of whom present with some form of cognitive–adaptive or neurological disability, can contribute to better specification of an alcohol exposure phenotype. Other research designs may also help to address unresolved questions about the extent of neurodisability in relationship to alcohol exposure, and the effect that different patterns of exposure may have (Gray *et al*. 2009).

Suggestions to change how we name things are likely to encounter resistance. However, acknowledging the limitations inherent in the current terminology and committing to build an improved evidence base offer the best hope that a more satisfactory, and ultimately a more sustainable, alternative diagnostic nomenclature can be devised for an area of established and increasing clinical, epidemiological and policy relevance.

Key messages

- Causal inferences and assumptions undermine our ability to diagnose prenatally exposed children with an alcohol-related teratogenic condition other than foetal alcohol syndrome or partial FAS.
- The available terminology for these children, specifically alcohol-related neurodevelopmental disorder and foetal alcohol spectrum disorder(s), creates dilemmas for clinicians because they involve a causal inference about the role of prenatal alcohol exposure which may not be justifiable in the case of individual patients, either logically or in light of current evidence. Epidemiological data using these terms may be problematic to interpret due to this issue, and there are ethical and perhaps legal issues in a diagnosis that implicates maternal behaviour in a child's condition, when the aetiology is not conclusive.
- Additional research evidence may assist with evaluating causal relationships at the individual patient level. Until that is available, it seems preferable to decouple the phenotypic–descriptive and aetiological elements in diagnosing these children. This would involve combining a new and more generic, but accurately descriptive term, such as complex neurodevelopmental disorder, with separate identification and coding of risk factors for neurodevelopmental morbidity among children, of which prenatal alcohol exposure is one.

Acknowledgements

Dr Miller gratefully acknowledges partial support for academic activities from the Sunny Hill Foundation for Children, the thoughtful comments of a number of respected colleagues on earlier versions of this paper and Jane Shen for her assistance with preparation of the manuscript as it has evolved.

References

Aase, J. M., Jones, K. L. & Clarren, S. K. (1995) Do we need the term 'FAE'. *Pediatrics*, **95**, 428–430.

Abel, E. L. (2006) Fetal alcohol syndrome: a cautionary note. *Current Pharmaceutical Design*, **12**, 1521–1529.

Astley, S. J. (2004) *Diagnostic Guide for Fetal Alcohol Spectrum Disorders: The 4-Digit Diagnostic Code*, 3rd edn. University of Washington Publication Services, Seattle, WA, USA.

Astley, S. J. (2010) Profile of the first 1400 patients receiving diagnostic evaluations for fetal alcohol spectrum disorder at the Washington state fetal alcohol syndrome diagnostic & prevention network. *Canadian Journal of Clinical Pharmacology/Journal Canadien De Pharmacologie Clinique*, **17**, e132–e164.

Autti-Ramo, I., Fagerlund, A., Ervalahti, N., Loimu, L., Korkman, M. & Hoyme, H. E. (2006) Fetal alcohol spectrum disorders in Finland: clinical delineation of 77 older children and adolescents. *American Journal of Medical Genetics. Part A*, **140**, 137–143.

Baca, C. B., Vickrey, B. G., Caplan, R., Vassar, S. D. & Berg, A. T. (2011) Psychiatric and medical comorbidity and quality of life outcomes in childhood-onset epilepsy. *Pediatrics*, **128**, e1532–e1543.

Bertrand, J. & Dang, E. (2012) Fetal alcohol spectrum disorders: review of teratogenicity, diagnosis and treatment issues. In: *Handbook of Children with Special Health Care Needs* (ed. D. Hollar), pp. 231–258. Springer, New York, NY, USA.

Bertrand, J., Floyd, R., Weber, M., O'Connor, M., Riley, E., Johnson, K., Cohen, D. & National Task Force on FAS/FAE (2004) *Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis*. Centers for Disease Control and Prevention, Atlanta, GA, USA.

Bishop, D. & Rutter, M. (2009) Neurodevelopmental disorders: conceptual issues. In: *Rutter's Child and Adolescent Psychiatry*, 5th edn (eds M. Rutter, D. V. M. Bishop, D. S. Pine, S. Scott, J. Stevenson, E. Taylor & A. Thapar), pp. 32–41. Blackwell Publishing, Oxford, UK.

Canadian Institute for Health Information (2012) ICD-10-CA. Available at: http://www.cihi.ca/CIHI-ext-portal/internet/en/document/standards+and+data+submission/standards/classification+and+coding/codingclass_icd10 (last accessed 19 November 2012).

Chudley, A. E. (2008) Fetal alcohol spectrum disorder: counting the invisible – mission impossible? *Archives of Disease in Childhood*, **93**, 721–722.

Chudley, A. E., Conry, J., Cook, J. L., Loock, C., Rosales, T., LeBlanc, N. & Public Health Agency of Canada's National Advisory Committee on Fetal Alcohol Spectrum Disorder (2005) Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *CMAJ: Canadian Medical Association Journal*, **172** (5 Suppl.), S1–S21.

Clarren, S. K. & Lutke, J. (2008) Building clinical capacity for fetal alcohol spectrum disorder diagnoses in western and northern Canada. *Canadian Journal of Clinical Pharmacology/Journal Canadien de Pharmacologie Clinique*, **15**, e223–e237.

Coggins, T. E., Timler, G. R. & Olswang, L. B. (2007) A state of double jeopardy: impact of prenatal alcohol exposure and adverse environments on the social communicative abilities of school-age children with fetal alcohol spectrum disorder. *Language, Speech, and Hearing Services in Schools*, **38**, 117–127.

Falgreen Eriksen, H. L., Mortensen, E., Kilburn, T., Underbjerg, M., Bertrand, J., Støvring, H., Wimberley, T., Grove, J. & Kesmodel, U. (2012) The effects of low to moderate prenatal alcohol exposure in early pregnancy on IQ in 5-year-old children. *BJOG: An International Journal of Obstetrics and Gynaecology*, **119**, 1191–1200.

Franklin, L., Deitz, J., Jirikowic, T. & Astley, S. (2008) Children with fetal alcohol spectrum disorders: problem behaviors and sensory

processing. *The American Journal of Occupational Therapy: Official Publication of the American Occupational Therapy Association*, **62**, 265–273.

Frith, U. (2001) What framework should we use for understanding developmental disorders? *Developmental Neuropsychology*, **20**, 555–563.

Gemma, S., Vichi, S. & Testai, E. (2007) Metabolic and genetic factors contributing to alcohol induced effects and fetal alcohol syndrome. *Neuroscience and Biobehavioral Reviews*, **31**, 221–229.

Gray, R., Mukherjee, R. A. S. & Rutter, M. (2009) Alcohol consumption during pregnancy and its effects on neurodevelopment: what is known and what remains uncertain. *Addiction*, **104**, 1270–1273.

Gropman, A. L. & Batshaw, M. L. (2010) Epigenetics, copy number variation, and other molecular mechanisms underlying neurodevelopmental disabilities: new insights and diagnostic approaches. *Journal of Developmental and Behavioral Pediatrics: JDBP*, **31**, 582–591.

Guerrini, I., Thomson, A. D. & Gurling, H. D. (2007) The importance of alcohol misuse, malnutrition and genetic susceptibility on brain growth and plasticity. *Neuroscience & Biobehavioral Reviews*, **31**, 212–220.

Kramer, M. S. (1988) *Clinical Epidemiology and Biostatistics: A Primer for Clinical Investigators and Decision-Makers*. Springer-Verlag, New York, NY, USA, pp. 265–268.

Kramer, M. S. & Lane, D. A. (1992) Causal propositions in clinical research and practice. *Journal of Clinical Epidemiology*, **45**, 639–649.

Lupien, S. J., McEwen, B. S., Gunnar, M. R. & Heim, C. (2009) Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews. Neuroscience*, **10**, 434–445.

Mattson, S. N., Crocker, N. & Nguyen, T. T. (2011) Fetal alcohol spectrum disorders: neuropsychological and behavioral features. *Neuropsychology Review*, **21**, 81–101.

May, P. A., Gossage, J. P., Kalberg, W. O., Robinson, L. K., Buckley, D., Manning, M. & Hoyme, H. E. (2009) Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Developmental Disabilities Research Reviews*, **15**, 176–192.

Pei, J., Job, J., Kully-Martens, K. & Rasmussen, C. (2011) Executive function and memory in children with fetal alcohol spectrum disorder. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, **17**, 290–309.

Rasmussen, C. & Bisanz, J. (2009) Executive functioning in children with fetal alcohol spectrum disorders: profiles and age-related differences. *Child Neuropsychology: A Journal on Normal and Abnormal Development in Childhood and Adolescence*, **15**, 201–215.

Shankar, K., Ronis, M. J. & Badger, T. M. (2007) Effects of pregnancy and nutritional status on alcohol metabolism. *Alcohol Research & Health: The Journal of the National Institute on Alcohol Abuse and Alcoholism*, **30**, 55–59.

Shevell, M. (2008) Global developmental delay and mental retardation or intellectual disability: conceptualization, evaluation, and etiology. *Pediatric Clinics of North America*, **55**, 1071–1084.

Skogerbø, A., Kesmodel, U., Wimberley, T., Støvring, H., Bertrand, J., Landrø, N. & Mortensen, E. (2012) The effects of low to moderate alcohol consumption and binge drinking in early pregnancy on executive function in 5-year-old children. *BJOG: An International Journal of Obstetrics and Gynaecology*, **119**, 1201–1210.

Stratton, K. R., Howe, C. J. & Battaglia, F. C. (eds) (1996) *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment*. National Academy Press, Washington, DC, USA.