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To: The Parliament of the Commonwealth of Australia, Standing Committee on Social Policy and Legal Affairs

RE: **Inquiry into Foetal Alcohol Spectrum Disorder, 2012**

I wish to make the following comments to highlight the complexities associated with the causes of Foetal Alcohol Spectrum Disorder (FASD). I also wish to reinforce the need to avoid inflicting individualised blame for FASD through future interventions and policy approaches. My comments are based on findings from my PhD research. The research explored how practice and policy may influence how the origins of child health problems are understood and who or what is held accountable.

Foetal Alcohol Spectrum Disorder

FASD refers to several different, but related, conditions including Foetal Alcohol Syndrome (FAS), Alcohol-Related Neurodevelopmental Disorder (ARND) and Alcohol-Related Birth Defects (ARBD) (Manning & Hoyme, 2007; Peadon, Fremantle, Bower & Elliot, 2008). It is important to emphasise that exposure to alcohol during foetal development does not automatically cause FASD. Instead, the development of FASD involves complex processes where alcohol exposure interacts with many other risk factors.

Risk factors

Throughout the existing medical literature numerous risk factors for FASD are identified. The majority of those discussed are *maternal* risk factors, that is, characteristics of the pregnant or mothering woman. Such factors include low education levels, genetics, untreated or poorly treated mental health problems, social isolation, reduced access to prenatal and postnatal care services and a history of victimhood and abuse (Chudley *et al.*, 2005). Other risk factors are related to the characteristics of the father or the child itself. For example, paternal alcohol consumption and paternal drug use at the time of conception are identified as increasing the risk to the foetus (Chudley *et al.*, 2005). Paternal genetics are also suspected to have some influence over whether a foetus will develop FASD (Green & Stoler, 2007). However, the reasons that these paternal characteristics may increase the risk of FASD are not stated explicitly and far less attention is directed to them than is directed to examination of risks that emerge from women's bodies and behaviours. Various characteristics of the foetus are also identified as risk factors. These include genetics, prenatal exposure to cocaine and smoking, including passive smoke, inadequate nutrition and living in a poor developmental environment, which may exist as a result of the abuse or stress of a pregnant woman (Green & Stoler, 2007).

Despite the multitude of factors that are thought to influence the development of FASD, most discussions about causation and prevention are ultimately dominated by a focus on maternal alcohol consumption (the topic of most other submissions reinforces this focus). I will provide further evidence of how this focus develops through a brief analysis of an article published in 2007 that reviewed existing evidence on the causes of FASD.

Reductionism and its effect in creating blame

The review paper by Green & Stoler (2007) explains that many factors influence the development of FASD and, in particular, foetal genetics have an important role. Such genetic factors may explain why women of a similar age can consume comparable amounts of alcohol during their pregnancies and some will give birth to a child with FASD while others will not (Green & Stoler, 2007). Furthermore, links between ethnicity and genetic susceptibility to FASD were proposed in the review. More specifically, the authors referred to research that has shown that African American people appear to be more genetically susceptible to FASD than people from other ethnic groups in the US (Green & Stoler, 2007). The authors also highlight that some forms of genetic predisposition to FASD are present only within the *male* genotype (Green & Stoler, 2007). This means that men *may* have a greater likelihood than women of having a genetic susceptibility to the effects of FASD. This also means that, theoretically, men may be more likely than women to pass on such a predisposition to their offspring.

Despite the identification of these various factors, however, the tone that permeates the review article constructs women as entirely responsible for preventing and causing FASD. This is made clear since all of the preventative strategies that are identified relate to the behaviours and characteristics of women. Women's responsibility for FASD is constructed on the basis that genetic susceptibility, regardless of its origin, can only contribute to the development of FASD if a woman consumes alcohol during pregnancy. Therefore, on the basis of such a simplified cause and effect relationship, which ignores the acknowledged relational, environmental and biological pathways to FASD, women are rendered exclusively responsible.

Maintaining and perpetuating such a simplistic focus is problematic. We have considerable evidence to suggest that it is not *only* women's consumption of alcohol that determines whether, and how severely, a child will experience FASD. While it may be easier to concentrate our focus on the end point of causation, that is women's consumption of alcohol during pregnancy, it is not adequate if we are serious about preventing FASD.

Moving forward

In the future we must avoid perpetuating such a reductive and damaging approach by broadening the scope of our thinking, practice, research and policies. It is vital that all future practice and policy acknowledges and examines the complexities that are associated with FASD. In particular, we need to question how policy can address the social, cultural and economic problems that may make it difficult for men and women to optimise their health, and minimise their alcohol consumption, around the time of conception.

As part of broadening our focus it will be necessary to translate these ideas into mainstream health advice. In doing so it will be beneficial to emphasise more clearly as part of public health guidelines that while women and men can behave in ways that are conducive to good reproductive health, adjusting individual behaviour will not ensure reproductive health in *all* cases. The current guideline that "There is **no safe time** to drink alcohol during pregnancy, there is **no safe amount** of alcohol." (Government of South Australia, 2007) [Emphasis in original] does not do this. Instead it perpetuates a reductive focus that is not fully supported by research evidence. It also creates the potential for misplaced blame on women by supporting the belief that they are solely responsible for FASD, regardless of how much alcohol they consumed, when they consumed it, why they consumed it or what other factors contributed to their child's development.

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References:

Chudley, A, Conry, J, Cook, J, Loock, C, Rosales, T & LeBlanc, N (2005) 'Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis', Canadian Medical Association Journal, vol. 172 (5 suppl), pp. S1-S21.

Government of South Australia (2007) *Pregnancy and alcohol don't mix: Information card*, Women's and Children's Hospital, Adelaide.

Green, R & Stoler, J (2007) 'Alcohol dehydrogenase 1B genotype and fetal alcohol syndrome: A HuGE minireview', *American Journal of Obstetrics & Gynecology*, vol. 197, no. 1, pp. 12-25.

Manning, M & Hoyme, H (2007) 'Fetal alcohol spectrum disorders: A practical clinical approach to diagnosis', *Neuroscience and Behavioral Reviews*, vol. 31, pp. 230-238.

Peadon, E, Fremantle, E, Bower, C & Elliot, E (2008) 'International survey of diagnostic services for children with Fetal Alcohol Spectrum Disorders', BMC Pediatrics, vol. 8, no. 12, pp. 1-8.