National Organisation for Fetal Alcohol Spectrum Disorders

INQUIRY INTO EFFECTIVE APPROACHES TO PREVENTION AND DIAGNOSIS OF FETAL ALCOHOL SPECTRUM DISORDER

RESPONSE TO QUESTIONS WITH NOTICE FROM NOFASD AUSTRALIA

Submitted by:

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with support from Sophie and Cheryl

20th July 2020

Introduction:

NOFASD Australia would like to thank Senator Griff and the members of the Inquiry for undertaking the complex task of reviewing Australia's response to FASD.

In reference to NOFASD responses to Questions with Notice, these points are made:

1. There is a stigma surrounding FASD which hampers prevention, diagnosis and intervention;

2. Failure to recognise FASD leads to further harm for affected individuals, families and communities;

3. An Australian prevalence study would go some way towards addressing the issues related to prevention, diagnosis and intervention.

Question One:

Thank you for undertaking a survey of carers for the purposes of this inquiry (attachment 2). The survey highlights the many barriers to diagnosis including ignorance about FASD among doctors. There was also one comment, where a parent noted: "The paediatrician sees no value in a diagnosis, as he believes it attracts no extra help". As far as you are aware, is that attitude an isolated/rare occurrence?

NOFASD Response:

This attitude is a common occurrence. The NOFASD Helpline receives approximately 1000 individual contacts annually. It is reported to the Helpline that health and allied professionals deny the opportunity to screen for FASD for many reasons including that "they don't believe in FASD", "it is far more likely to be autism", "there are more services for autism".

Parent 1 Testimony:

My personal experience has been the lack of knowledge of prevalence, the impact of PAE at low levels and prior to pregnancy confirmation, and the high frequency of co-occurring diagnosis. My personal experience of 3 x GPs, 3 x paediatricians, 3 x clinical psychologists, 1 x speech pathologist and 1 x Occupational Therapist is a lack of knowledge. I believe because I am a professional, educated white woman, assumptions were made that I did not consume alcohol during my pregnancy and the question was never asked. If the question of PAE was asked and there was more knowledge amongst health professionals, my son could have been diagnosed as early as the age of 5, which is 9 years earlier than when the process of assessment commenced. This is a serious loss of time for early intervention.

Parent 2 Testimony:

We felt like we were in a whirlpool searching for "real" answers, after a diagnosis of Autism, but without an improvement in behaviours and eventually severe regression. We went from therapist to specialist searching for what felt like an elusive answer to the increasingly aggressive behaviours. Alcohol consumption was not questioned, and it was not a part of any discussion until I eventually stumbled onto information about FASD and produced this information at our next appointment. But although the psychologist was aware of FASD, he did not believe this was the case for our son because upon initial sight investigation, he did not appear to have the facial features. I researched more and found that facial features were not a requirement for a diagnosis of FASD. From there our journey to diagnosis began for our son. It is worth noting that upon a full diagnostic assessment, our son *does* have the 3 sentinel facial features despite the psychologist assuring us that they were not present – when in fact the Psychologist could not recognise the features.

Question Two:

You mention (p7 of submission) that "as many as 80% of people diagnosed with FASD were previously diagnosed incorrectly with another disability/disorder". What are the top misdiagnosed conditions for people with FASD?

NOFASD Response:

- The most common mis-diagnosis identified in the Canadian study by Chasnoff et al. 2015 was ADHD.

Reference - Misdiagnosis and Missed Diagnoses in Foster and Adopted Children With Prenatal Alcohol Exposure Ira J. Chasnoff, MD, Anne M. Wells, PhD, Lauren King, MA. Published in the Journal Pediatrics Volume 135, No. 2, February, 2015.

- A 2013 publication in Volume 1 of Recent Advances in Autism Spectrum Disorders (ASD), Chapter 20 by Kieran D. O'Malley and Susan D. Rich titled Clinical Implications of a Link between FASD and Autism Spectrum Disorder (page 457) noted the following:
  a) That in many countries there is ambivalence to accept the true prevalence of FASD and that this leads school systems and physicians to 'hide' many FASD patients under an ASD diagnosis (Page 458).

  b) Medications which work for Autism may cause problems for patients who, in reality have FASD (page 458).

  c) The link between ADHD and FASD is finding more universal acceptance and the link between Autism and FASD will not be far behind (page 465).

d) Collaborative work was needed in the two academic fields of ASD and FASD.

- In addition, in a 2019 study published in the Journal titled *Alcohol* 76 - 23-28 by Raja A.S. Mukherjee et al. noted that 74% of those with FASD also had an ADHD diagnosis and 68% had an Autism or social and communication disorder. In addition, the study further identified that damage due to alcohol was often attributed incorrectly to parental neglect. In cases of a co-morbid diagnosis it is likely that the FASD is often missed and the proposed treatments and pharmacology will be less successful without recognition of the FASD.

Parent 1 Testimony:

As an anecdotal example, I have never been asked, in any of the appointments I have attended with my son, whether I consumed alcohol during my pregnancy. An assumption was made, based on my appearance, communication skills and personal medical history. I visited numerous allied health professionals and developmental paediatricians asking about potential causes, concerns and assessments to assist my son with his challenges and challenging behaviours. It required assertive advocacy on behalf of my son, even after telling two GPs about the PAE, to gain a referral for my son to a paediatrician at the age of 13. Both GPs on two separate occasions suggested it was more likely my son had Autism Spectrum Disorder than FASD. I provided both GPs with a range of information to educate them. Despite extensive information, my disclosure of the PAE and the clinical psychologist and speech pathologist assessments, the referral to the developmental paediatrician was rejected by the paediatrician, with no reason given. Further assertive advocacy was required to determine the reasons and the referral was reinstated.

The lack of knowledge amongst many health professionals is critical, many less informed or less supported parents and carers would have walked away from the assessment process, which I did over a number of years, before I realised the possibility that FASD was present.

Question Three

SENATOR GRIFF*: In the interest of time, I'll read out a couple of questions that I'd like to have put on notice. The 2012 House of Representatives report titled FASD: the hidden harm was obviously very important to you, particularly you, Ms Gray. Most of the 19 recommendations have not yet been enacted. Can you on notice advise the committee which of the remaining 15 recommendations, in your view, are the most urgent, and what's been happening in that space?*

NOFASD response:

I have worked with the methodical table prepared by FARE in their submission to the Inquiry. FARE as an organisation have been proactive and effective in regard to the

FASD agenda. I have updated this chart and added in knowledge or information based on NOFASD's experience. I have attached this chart as ***Appendix One***.

One of the biggest problems since the Hidden Harm was released is that only the Health Department, in the Drug and Alcohol strategy branch, has been really proactive in regard to FASD. I have a vivid memory of a round-table 3 years ago on FASD held in Canberra and there were 20 seats at the table and the only vacant one was the NDIS representative who was supposed to attend the event. The name plate sat there vacant through the whole day. There has been great reluctance on the part of the probably 50 – 100 (at the minimum) government departments across Australia which need to address FASD because it is intimately connected to their mission and goals. The NDIA has recently done a little more in regard to FASD but not enough.

The most appalling oversights are the state-based Child Protection Services. The number of children in care is a daily news headline and we are not aware of any jurisdiction in Australia which is screening for FASD. As recently as July 1, 2020 we were advocating to support a social worker who was attempting to get a child screened for FASD in a large Australian state. The senior paediatrician in the government service denied referral, based on the belief that the child did not have facial features. In this case the expert was wrong twice. Firstly, because less than 15% of those affected have the facial features and secondly this child *did* have the features but because they are subtle the health professional did not know what he/she was actually looking for. Events like this are a very common occurrence on the Helpline.

We have incidents reported to us in some states that child protection departments point blank refuse to screen for FASD – even when the indicators are compelling. Yet these children are no doubt moved from foster home to foster home because placements break down and the disability is not recognised. They are frequently reunited with parents and these placements also break down because of continued substance use and the likelihood that there is multi-generational undiagnosed FASD present. The overlap between children-in-care and young offenders is well known and even with the robust research into young offenders in WA, NOFASD is not aware of any Australian systems routinely screening for FASD, despite all the indicators being present.

A US State Government in June 2020 introduced mandatory screening for all children entering state care and when this was promoted on the NOFASD Facebook page the positive reaction from many parents expressed hope that this would happen in Australia.

We were aware of employees in Australian state government departments who were prevented by their supervisors from lodging submissions to this Inquiry.

It would be an excellent outcome of this inquiry if State Government child protection departments routinely screened for FASD and no longer required foster parents to

have to invest hours in fruitless advocacy to get better outcomes for children in their care.

Question Four

CHAIR: *Cheryl, in particular, I want to direct this as a question on notice to you. In outlining your experiences, you commented on the need for more support at home. When you're answering the questions around the NDIS, could you expand on that and provide more detail around what forms of support would support you in caring for your son and supporting your son now, and also into the future? Because not all those helps maybe available from the NDIS. I just want to get a full picture of what sort of supports are necessary, in your opinion. Is that clear?*

NOFASD Response:

As CEO of NOFASD I was an observer and interested bystander through Cheryl and [redacted] long NDIS journey for their son. I was incredibly humbled and admired their tenacity, their commitment, and their capacity to continue to research and advocate for their son. At times, the treatment they received disturbed me greatly. They were humiliated, made to feel like they were expecting too much, intentionally given a “run around”, spoken to in ways which patronised them. Underlining all of this was a total lack of understanding about FASD right through the entire system.

NOFASD started some intensive engagement with the NDIS and after a number of compelling letters to the Minister we were given an opportunity to outline some of our concerns to a Ministerial Advisor and soon after our grant application to the NDIS was successful. This came after five unsuccessful submissions. This has enabled NOFASD to commit some resource to a National Information Program and we are within a month or so of being able to release the first online FASD training which is recognised and accredited by CanFASD (Canada) and NOFASD Australia. It will be offered free and is a series of modules with a certificate issued upon successful completion. This will create a situation where parents and carers can now expect a minimum level of competency when they interact with health professionals and service providers.

Parent 1 Testimony:

My experience to date:

A number of families report a ‘road-block getting through the NDIS access stage due to lack of understanding and knowledge from Local Area Coordinators (LACs) in relation to FASD.

The cost of a FASD diagnosis and the extensive waitlists within both the private and public systems to access FASD informed assessments, result in long waiting periods for families to access the NDIS whilst they continue to live with the daily challenges for the young person they are supporting. Waitlists are 2 years in most places. The

current diagnostic system is likely to be only the “tip of the iceberg” in regards to FASD prevalence because to obtain a FASD diagnosis the patient first has to be seen by a health professional who understands FASD and considers the possibility of this referral.

In addition, there is lack of:

- assessment services for adult diagnosis, long waitlists and pricing often double that of a child diagnosis;
- recognition from the NDIA of FASD as a listed disability/disorder to raise awareness of prevalence with planners and service providers;
- engagement from the NDIA to access information and training on FASD to inform policy makers and a top down approach to awareness raising;
- whole of organisation approach to upskilling NDIS planners and key staff to ensure they are FASD informed.

Parent 2 Testimony (Cheryl):

I thank the committee for providing me this opportunity to outline the difficulties I have encountered with the NDIS and what I propose would be required to address the shortfalls relating to FASD.

In order to paint a picture of the difficult journey which I experienced whilst advocating for our son, I would like to refer you to my personal submission (Submission Number 24) which I made to this Senate Inquiry.

- A very important step to help all those living with FASD and those who care for them to access and benefit from the NDIS, would be for FASD to be clearly and officially recognised as a disability. This would alleviate so many hurdles for carers.
- NDIS Planners should be specifically trained to understand FASD, not just a basic awareness of it.
    - It was apparent, in our case the initial Planners had not ensured they were educated about FASD and this was confirmed when I questioned this during our interview.
    - The framework for the questions asked, appeared to be based around providing support for those with physical disabilities. It was as if your individual situation didn’t fit the accepted profile and target of the questions. There wasn’t scope for stepping outside the designated disability fields.

    - They should also ensure they thoroughly research each individual case of FASD, as there is such a wide spectrum.

    - Planners should thoroughly understand the strengths and deficits of each individual living with FASD, so that they have a good understanding of the types of therapies and support that will be of most benefit.

- Respect the experience and knowledge of parents and carers. The day-to-day experience of parents and carers who live in this world, is the most important information the planner will be able to gain in order to aid in developing an appropriate plan.

- Caring for someone living with FASD is extremely stressful and exhausting, it is a 24 hour a day, 7 day a week role, with little to no respite. It is vital that any plan includes support for carers and parents. If the carer is not supported, they are then unable to support the person living with FASD, nor any other family members. Burnout is a common theme amongst carers, and this then jeopardises their health and ultimately the whole family structure.

  Services such as:

    - Household cleaning

    - Cooking

    - Gardening

    - Running errands

    - Attending to personal appointments

- It is extremely difficult for a parent/carer to complete the tasks outlined above, whilst attending to the high-level needs of the person they care for. By providing carers with these supports, not only does it take away a huge workload burden from the carer, it also supports relationship building between the carer and the person they care for with FASD. It is difficult to factor in time to just "be" and spend quality time with the family member living with FASD due to the high level of care required. I referred to the concept of "poolside supervision" being required for people with FASD. This is not an exaggeration and when an adult is providing genuine poolside supervision, they cannot be responsible for other tasks at the same time.

- It is very important for planners to understand that traditional therapies often don't work for those living with FASD. In fact, they often trigger aggressive behaviours. Being open to therapies that are often more effective, such as

animal assisted therapy, play therapy etc., will obtain a much more positive outcome.

- It is important for the NDIS to ensure that there is funding available for service providers and any employees who will work with the person living with FASD, to be FASD trained. This ensures that the service provider can:
    - Suggest which employee(s) would be the best choice. The service provider would also be able to advise if they have no staff suitable at that time.
    - Any employees deemed suitable would then undertake FASD training themselves.
    - Training helps staff to understand how best to work with their client and also how to avoid triggers.
    - This is essential to reduce the chance of problem behaviours following any time spent with a support worker.
- Many students with FASD find it difficult to attend mainstream school either full-time or at all. Whilst the education department is responsible for the education of the young person living with FASD, the simple truth is that it would be a rare situation for any school to have the resources or funding required. Simple support in an NDIS plan to assist with this are:
    - Support for a carer to work with the student one-on-one either at the school or at home.
    - This support would not be to force the student with FASD to complete the tasks as other students are expected to, but instead to work with strengths and incorporate these into an aligned outcome.
    - For some students with FASD, even tasks designed to work with strengths may be too overwhelming. Instead, a support worker could work with the student to develop social thinking skills in a non-invasive manner.
    - Acknowledge and support Carers who have no other options but to home educate a person with FASD.
- FASD is not Autism, therefore the strategies and supports for people with Autism very often don't work for those with FASD, this is why education about FASD, and the differing supports required is vitally important. Autism strategies often serve to exacerbate FASD behaviours.
- One size does not fit all, FASD is a spectrum, which affects and impact each person differently, this means a support package and process must be flexible

to meet those needs of the individual and the circumstances required by the individual, parent or carer. These circumstances will be broad and varied, and this must be accommodated for.

- People affected by FASD are in no way responsible for what has happened to them and in many cases the accepted norms or practices of the day are responsible. This is the hand they have been dealt, we can either support them to be the best they possibly can and be positive contributors to our society, or we can do nothing and deal with the consequences.

Thank you for the opportunity to provide this information and please don't hesitate to contact me should you require clarification or further information.

Question Five

Education seems to be another major issue where many carers report they struggle to get extra assistance and the burden falls to parents to educate teachers. What do you think is the barrier to schools recognising FASD as an issue they need to address and accommodate? (Do you consider it is tied to funding? Teacher education? Lack of top-down direction from education depts?)

NOFASD Response

Parent 1 Testimony:

All of the above! Teachers do not receive adequate training on neurodevelopmental impairments, impacts, challenges, and strengths during teacher training. An understanding of the neurodevelopmental disorders and impacts need to be embedded into teacher training. Specific disabilities, disorders, and the impact on a child's learning, along with the strategies to help the children learn differently, must be part of teacher training.

The education system and curriculum in Australia (and many other developed western countries) requires rigid criteria to be taught and met for each year group. Learning environments are often static, with traditional classroom settings. Embedding learning into teacher training to create meaning and relevance, such as drawing upon an individual's interests and experience for project-based learning, learning by doing, multi-sensory learning, practical demonstrations, and meaningful hands on involvement in tasks. Providing a variety of learning environments and accommodating the different learning styles. Utilising movement, practical tasks, regular breaks, audio, and visual content and suitably paced lessons to enable students to be a part of a transformational learning environment, rather than the current transactional learning which takes place.

There is little direction from some education departments nationally in relation to accessing FASD training for teachers. FASD is not seen as an issue for a large number of students and therefore to invest time in specialist training which appears to be for a minority of students, is not a cost-effective endeavour. The lack of awareness of community prevalence and therefore prevalence amongst students within the education system is a key issue to raising awareness and providing training.

Parent 2 Testimony:

The education system seems to focus purely on educational statistics for students and schools. A student with FASD cannot cope well, let alone thrive in that sort of environment. FASD students need programs based on their interests and strengths in order to learn effectively. For many students with FASD, this may indeed mean that it will not align with the school curriculum and this must be accommodated. Being forced to fit into a system based on statistics will not provide them with the education they need to live the best life possible. This will come from an Individual Learning Plan that will most likely be very different to special needs learning plans that schools currently provide.

Question Six

Regarding the other survey you conducted (attachment 1) which showed 44% of the women who responded said no health professional had spoken to them about alcohol and pregnancy, and only 10% received information prior to conception: Given the damage alcohol can cause in the first few weeks of pregnancy, is not providing advice about drinking until after conception leaving it too late to avoid harm?

NOFASD Response:

If a woman is sexually active and not using reliable birth control, then counselling around alcohol harm from conception to the end of breast-feeding should be discussed routinely. In general, Australians are quite shocked when they hear this because for many couples falling pregnant can take some time. It comes as a huge surprise that the entire period of pre-conception planning requires abstinence from alcohol.

In addition, approximately 50% of pregnancies are unplanned which is why the messages about contraception practices or abstinence are required if alcohol is being consumed.

These hard facts are unpopular in a country where the alcohol industry strongly resisted warning labels and harmful use of alcohol occurs nationally.

Parent 1 Testimony:

YES, advice needs to be given to individuals at the earliest opportunity to prevent an alcohol exposed pregnancy.

At what point do you consider health professionals should (routinely) engage with women on the risks of alcohol in pregnancy?

When patients of child-bearing age attend a medical appointment, information about PAE should be an item that is routinely discussed. Any women attending an appointment related to sexual health and birth control, should be routinely provided with this information.

This is not just a task for medical professionals, there are many teenagers and emerging adults who will be at a higher risk because they do not engage with medical practitioners to enquire about sexual health/birth control methods. Information and awareness should be embedded in the school health curriculum – as many other health issues are.

Question Seven

The submission (attachment 1) mentioned some women receiving advice that low-level drinking, e.g. a glass once or twice a week, was acceptable in pregnancy. Noting Sophie's compelling story, can you advise whether NOFASD is aware of many FASD cases that have resulted from low level drinking – that is, where the mother only drank according to the guidelines at the time?

NOFASD response:

NOFASD has anecdotal information on this issue. NOFASD is aware of compelling and relevant research and has detailed this below.

Anecdotal information

NOFASD receives 1000 Helpline enquires and 153,000 website views annually. Many of the Helpline enquiries are anonymous and the contact seeks and obtains information about FASD but does not identify themselves. Additionally, website visitors often go straight to these pages and spend time before reviewing other information pages about FASD. We believe these user journeys are indicators that women recognise that their children are displaying signs of FASD but are reluctant to pursue formal diagnosis. As a result of these user journeys NOFASD is in the process of expanding a "Frequently Asked Questions" series of pages to address these questions. We are also proposing a Birth Mothers section which will specifically address women who suspect that FASD may have impacted their child.

Emerging research

Compelling research using cranio-facial imaging was undertaken as part of the AQUA study in Melbourne. This emerging research points to the harms of low risk drinking. The most critical research is attached as **Appendix 2** and relates to evidence of FASD like changes being visible in the cranio-facial shape of the developing fetus with low risk exposure. The lead researcher was Evi Muggli.

Animal model research on the impact of alcohol on developing young is extensive and all outcomes point to alcohol having a negative impact a pregnancy, even at low levels. Animal model research has been conducted using rigorous methodology and the strength of the outcomes should not be over-looked.

Parent 1 Testimony:

I don't have any information or know of information on this. Susan Astley's video on the Twin Study, identifying the impact of only 1 glass of wine per day could assist with supporting information from the USA.

Parent 2 Testimony:

Having a standard questionnaire that women are asked to complete within the first few weeks after child-birth, would greatly help with information in the future.

Question Eight

Can you also please elaborate on the NDIS services and supports available for people with FASD and their families, and what you consider are areas of unmet need (for example, lack of carer respite was identified in the hearing?)

NOFASD Response:

It is critical that services and supports are **FASD Informed** and it is very important to stress this. Clinicians such as OTs, Speech Therapists, Clinical Psychologists, play therapists, art therapists, equine therapy need to understand the disability. Respite can also be provided but the word 'respite' is no longer used and the requirement needs to be written into the NDIS Planning goals, e.g. assistance to access community, social and sporting activities, in-home support with independent living skills, improved access to education, holiday programs, active overnight assistance, assistance to access work. 'Overnight respite' is now called 'Short-term Accommodation Assistance'.

Question Nine

Senator Griff's concluding question about an independent life was briefly answered during the hearing. However, both parents subsequently felt that this is an important

area and required a more thoughtful response. Therefore, additional comments have been included here.

NOFASD Response:

Children with FASD and young people with FASD grow quickly into adults with FASD. Unsupported, as arguably most adults with FASD in Australia are, they may face a life of poor education outcomes, unsuccessful employment, increased risk of substance use disorders, increased involvement in risk-taking behaviours, challenges forming relationships, risk of homelessness, risk of being a victim of violent behaviour or perpetrating violence and contacts with justice systems. The highest risk is premature death, evidenced by Canadian research which documented that 34 years of age is the average life-span for people with a FASD diagnosis.

Parent 1 Testimony:

Re an independent life:

My son's neurodevelopmental impairments are in the domains of executive functioning, attention, language, and cognition, he has recently undergone further testing and we are awaiting the outcomes of these assessments. His impairments are classified as severe, however, he lives what appears to be, to the outside world… a very 'normal' life.

- His most significant challenges are currently within mainstream education as he is unable to concentrate, learn in the same way as his peers, retain information unless it is taught using different modes of teaching and repeated a number of times. He is not understood by his teachers and as his advocate I am in communication with his school several times a week. This is exhausting and at times stressful, however these efforts improve my son's school experience.

- He is extremely vulnerable and trusting of strangers, particularly with older teenagers. Left unsupervised, he would be taken advantage of and exploited, he is easily led. In complete contrast to this, he is a good judge of character with some people, reading faces and body language exceptionally well….his impairments mean he makes different decisions depending on the situation and how believable, in his mind, somebody is. His responses are very unpredictable.

- He has issues with food which could result in him not eating for 12 hours at a time because there is nothing in the house that he would 'like to eat'. The foods he likes to eat change on a regular basis and when he becomes hungry, he seeks calorific and sweet food, foods that are not often kept in the house to maintain a healthy diet. Many people affected by FASD have sensory issues related to food because their sense of smell and taste is

impaired. Food which a neurotypical person does not find problematic can be nauseating for a person with FASD.

- He finds certain tasks hard to perform due to an aversion to touching some textures, for example, putting items into a bin due to germs, touching some wet foods. His phobias in this regard are extreme when compared to a neurotypical child and are not able to be resolved by parenting strategies.

- He has anxiety, particularly related to learning environments and environments where new skills are needed to be learnt and then demonstrated. He is quite high functioning and manages to mask his anxiety well. His anxiety plays out in his behaviour towards his parents and his younger sibling with verbal anger, name calling and negative statements.

- He has a fascination with fast cars and motorbikes. Because he is high functioning, he will manage, with support, to achieve a driving licence and pass a driving test. The ability to drive a vehicle does not equate to being able to be in charge of a vehicle or drive the vehicle in a suitable manner. This area of his life and his safety is of the highest concern to us as his parents. I know of other parents of people with FASD who fear this situation and in one case I know an adult with FASD who lost their license and their parent is hopeful she/he will will not be able to regain it.

- He is likely to always need ongoing support with budgeting and money management, particularly in relation to the perseverance he has when he decides he wants a particular item, his spending becomes a 'need' and is impulsive.

- Support with training and employment – this is an area that will be essential for my son. As he progresses towards the end of Year 9, maintaining engagement at school is becoming increasingly difficult. He will move into a blended program in Year 10 and it is hoped the additional support at the age of 15 will assist him to learn sufficient communication and behavioural skills to enter a disability employment scheme after the age of 16.

- My son would not be able to work in an unsupported work environment due to the complexities of his impairments and the understanding that is needed to focus on his strengths to maintain ongoing employment.

Parent 2 Testimony:

- Due to our son's impulsive behaviours he will always need guidance and supervision from an appropriate adult. This will always be a priority for our son's safety and wellbeing.

- To make decisions about his own health and wellbeing, he will need support, as his obsessions will prevent good choices.

- When he has an obsession, it overtakes all his thoughts.
- Being easily mislead is, and always will be, a huge risk. Significant issues are drugs, alcohol, risky behaviours, and dangerous activities. Therefore, constant support and guidance are essential. This doesn't mean just talking things through, this means constantly ensuring a responsible adult is always with him. I see this as being one of the most significant protective factors in the future to ensure he has no contact with the justice system. If he were given the freedoms that a neurotypical teenager has it is likely he would already have had this contact with justice. I was once asked if he had an offending record by an NDIS planner who advised me that this would make it easier for me to access NDIS support. We were devastated to be asked this question because we have devoted ourselves to prevent this – and it seemed shocking that such a record would be an advantage to obtaining much needed services.
- Family is extremely important to our son and it has been essential that we ensure that regardless of mine or my husband's future, that he will always be loved and cared for.
    - A difficult thing that needed to be done, was to set up our wills so that all information was available for a family member who would act as a guardian entrusted to make the best decisions possible, in the event that we weren't able to.
    - Whilst legally this is until our son turns 18, we know that he will always need this arrangement.
    - Our son's adult life is a great worry for us.
- Our current world and its environment will be a significant challenge for our son as an adult.
- We have slowly built our future around our son.
    - Ensuring there will always be a place with us for him.
    - Our caravan has been purchased specifically to ensure a full-size adult bunk, so he can continue to travel with us as an adult.
    - Re-setting our minds regarding our future as retirees and hopeful travellers. It has been very important to do this, so as to reduce any sense of loss.
- Planning for supports for us as older people to manage our son.
- Our son yearns to have the independence that others his age have. It is likely our challenges will increase as he ages and we work to support him, while

ensuring that he has as much choice and control as he can safely have to reach his potential.

Enclosed:

Appendix 1: NOFASD review of progress of the recommendations from the previous Inquiry into FASD – The Hidden Harm;

Appendix 2: "Association Between Prenatal Alcohol Exposure and Craniofacial Shape of Children at 12 Months of Age" Research Article (Muggli et al 2017);

Appendix 3. Letter to Ministers regarding Clear Alcohol Labelling. This was included because of the additional insight into FASD provided by the description.

Questions with Notice – Appendix One

NOFASD, AUSTRALIA

Note: NOFASD acknowledges the extensive work which has been undertaken by FARE throughout the last decade on FASD. NOFASD is grateful for their support and commitment. The follow summary is an adapted and updated document from FARE's November 2019 submission to the Inquiry.

PROGRESS OF RECOMMENDATIONS FROM PREVIOUS INQUIRY

FARE's submission in Nov 2019 said of the 19 recommendations made by the House of Representatives inquiry into FASD, The Hidden Harm in 2012.

Since November 2019 a number of initiatives have been undertaken.

MET	PARTIALLY MET	NOT MET
3	**4**	**12**
Recommendations: 1, 2 and 15	Recommendations: 4, 9, 10 & 16	Recommendations: 3, 5, 6,7, 8, 11, 12, 13, 14, 17, 18 & 19

Since Nov 2019 more work has been undertaken – THESE ARE HIGHLIGHTED IN BRIGHT GREEN

MET	#	WHAT THE RECOMMENDATION SAID	PROGRESS
Yes	1	National Plan of Action	The National Action Plan 2013-14 to 2016-17 (with $9.2 million in funding allocated). 2nd National Strategic Action Plan released 2018 ($7.2 million funding)
Yes	2	Establish an ongoing Fetal Alcohol Spectrum Disorders (FASD) Reference Group.	The FASD Technical Network was established in 2014 to oversee the implementation of the National FASD Action Plan. The Network provided advice to the Australian Government on how Australia could achieve a strategic coordinated approach to FASD. The Australian Government disbanded the network in 2017. **A revised group has been established but has only met once.**
Yes	15	Diagnostic Tool	Australian FASD Diagnostic Instrument was released in 2016 and online training on its use has been developed. Review of use and

MET	#	WHAT THE RECOMMENDATION SAID	PROGRESS
			update has been commissioned.
Partially	4	1. That NHMRC and health professional bodies are: • fully aware of the Alcohol Guidelines • Training on alcohol and pregnancy and skilled in asking That by 2014 all health professionals promote consistent messages.	Women Want to Know has done some of this. Launched on 1 July 2014 and provides resources including free online training to health professionals. Will be re-established as part of the National Awareness Campaign. **Pockets of work have been undertaken but this remains an area which needs further commitment. It is a chronic problem on the Helpline. Health professionals promote very inconsistent messages and have inconsistent knowledge of the disability.**
Partially	9	State and Territory governments to identify and implement effective strategies for pregnant women with alcohol dependence or misuse.	NDARC published "Supporting pregnant women who use alcohol or other drugs: a review of the evidence" but no information on if it is being used by AOD services. **NOFASD notes that there is a critical lack of awareness amongst AOD services about FASD.** **Initiatives by State and Territory Governments have been minimal.**
Yes	10	Health warning labels for alcoholic beverages and appropriate format and design of the labels by 1 March 2013.	**Ministers due to make final decision 17 July after a 2-year consultation period by FSANZ.** **This decision was passed at the meeting. Disappointing that the Federal Government did not vote in favour of the recommended label and concerning given the focus on FASD.**
Partially	16	Develop and implement a National Fetal Alcohol Spectrum Disorders (FASD) diagnostic and management services strategy.	**No national strategy but a FASD Clinical Network was established by clinicians in 2015, the network communicates by email.** **FASD Hub established in 2017 and has continued funding, is a central repository of documents and resources.** **NOFASD provides resource and referral services but the information is collected and gathered and is not part of a national strategy.**
No	17	Education material and training for:	**Grant Opportunities for some of this work were released in**

MET	#	WHAT THE RECOMMENDATION SAID	PROGRESS
		• special education teacher aides and class teachers; • parents, foster carers and foster care agencies; • police and court officials; • youth workers and drug and alcohol officers; and officers in correctional facilities and juvenile detention centres	**December 2019. Contracts awarded and commencing in May-June 2020.** **The grant amounts are relatively small to achieve what is hoped for.** **WA has training materials for those working in juvenile detention through the Understanding FASD: A Guide for Justice Professionals project.** **NOFASD provides education sessions on request for justice professionals.** **This recommendation requires effective engagement and liaison with over 40 state managed systems. Even with the grant funding it will be difficult to effectively impact them all.** **For example, no jurisdiction screens routinely for FASD when children enter care yet the rates of FASD amongst children in care, who have been removed because of AOD misuse, are likely to be well over 30%. It is a moral and ethical failure by children's services that this screening is not undertaken.** **No jurisdiction is screening for FASD amongst offenders yet the presumed rate of FASD as recorded in the landmark Banksia Hill study in WA was 50% the confirmed rate was 37%.**
No	3	Govt establish (within 12 months) a national diagnostic and management services strategy.	**A national FASD diagnostic and management services strategy has not been undertaken.**
No	5	Mechanism to record women's alcohol consumption during pregnancy, or at the time of birth for women and ensure information is recorded.	**Hunter New England NHMRC research collects information on women's alcohol consumption during pregnancy. It includes reporting consumption data, leadership support and local clinical guidelines. Also, electronic prompts and reminders for staff. This is needed nationally.** **AIHW has added 2 alcohol questions, voluntary uptake to Perinatal**

MET	#	WHAT THE RECOMMENDATION SAID	PROGRESS
			National Minimal Data Set (NMDS). Reports available 2021.
Yes	6	General awareness campaign.	**Grant funding awarded to FARE shifts this to yes.**
No	7	Health advisory label on pregnancy and ovulation testing kits. In place by 1 October 2013.	**No work undertaken on this. This would be an excellent placement and education opportunity for alcohol and pregnancy warning labels.**
No	8	States and Territories assist Indigenous communities to introduce community led initiatives to reduce high-risk consumption impact.	**There are small localised groups responding to FASD in some states and territories but states and territories have been slow to recognise, lead or support FASD initiatives.**
No	11	General health warning labels for alcoholic beverages • Consisting of text and a symbol on products, advertising and packaging by 1 Jan 2014 • The minimum size, position and content of health warnings be regulated • accompanied by a comprehensive public awareness campaign.	**FARE has recently been awarded funding for a general public awareness campaign however as yet NOFASD is not aware how this will translate when labelling is initiated.**
No	12	Commission an independent study into the impacts of the pricing and availability of alcohol and changing patterns of consumption by age and gender by 1 October 2013.	**No work undertaken but on 1 October 2018 the Northern Territory Government introduced a minimum unit price (MUP) on alcohol.**
No	13	Commission an independent study into the impacts and appropriateness of current alcohol marketing strategies directed to young people. Including sport and social media by 2013.	**NOFASD is not aware of work undertaken and this is an area critical to raising awareness of the harms of alcohol in pregnancy. The recent explosion of targeted marketing during COVID has been indicative of the gross over-reach of the industry and its capacity to infiltrate and deluge young people with pro-alcohol messages which normalise and glamorise excessive and regular consumption.**
No	14	Develop a National Alcohol Sales Reform Plan	**NOFASD has no knowledge of work undertaken.**
No	18	Include FASD in the List of Recognised Disabilities and the Better Start for Children with a Disability Initiative.	**The situation remains unchanged since 2012.** **NDIS only recognises Fetal Alcohol Syndrome under List B: Congenital conditions - cases where malformations cannot be corrected by surgery or other treatment and result in permanent impairment but with variable severity. Despite numerous attempts this terminology has not been updated.** **NOFASD was awarded funding of $700,000 over 3 years in November 2019 by the NDIS to implement a National Information**

MET	#	WHAT THE RECOMMENDATION SAID	PROGRESS
			Program about FASD. **Systemic structures which list recognised disabilities generally exclude FASD.** **The NDIS ILC Program discriminates against FASD in the grant application process because the disability has to be listed in a category named "Other" when applying because FASD is not listed. Many other disabilities are listed. The funded outcomes reflect the listed and named disabilities. NOFASD has raised this issue with the NDIS on a number of occasions, thus far without change.**
No	19	Recognise that people with FASD have a cognitive impairment and amend the eligibility criteria to enable access to support services and diversionary laws.	**No known work undertaken other than one small justice and employment services focussed grant recently awarded to a private provider in WA.** **On a regular basis issues emerge demonstrating the significant discrimination experienced by people with, or suspected of having, FASD.** **Gilbert and Tobin, a national law firm have dedicated some pro bono services to these issues and often provide advice when they can.** **There is widespread lack of understanding of support services which will be effective for people with FASD.**

JAMA Pediatrics | Original Investigation

Association Between Prenatal Alcohol Exposure and Craniofacial Shape of Children at 12 Months of Age

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IMPORTANCE Children who receive a diagnosis of fetal alcohol spectrum disorder may have a characteristic facial appearance in addition to neurodevelopmental impairment. It is not well understood whether there is a gradient of facial characteristics of children who did not receive a diagnosis of fetal alcohol spectrum disorder but who were exposed to a range of common drinking patterns during pregnancy.

OBJECTIVE To examine the association between dose, frequency, and timing of prenatal alcohol exposure and craniofacial phenotype in 12-month-old children.

DESIGN, SETTING, AND PARTICIPANTS A prospective cohort study was performed from January 1, 2011, to December 30, 2014, among mothers recruited in the first trimester of pregnancy from low-risk, public maternity clinics in metropolitan Melbourne, Australia. A total of 415 white children were included in this analysis of 3-dimensional craniofacial images taken at 12 months of age. Analysis was performed with objective, holistic craniofacial phenotyping using dense surface models of the face and head. Partial least square regression models included covariates known to affect craniofacial shape.

EXPOSURES Low, moderate to high, or binge-level alcohol exposure in the first trimester or throughout pregnancy.

MAIN OUTCOMES AND MEASURES Anatomical differences in global and regional craniofacial shape between children of women who abstained from alcohol during pregnancy and children with varying levels of prenatal alcohol exposure.

RESULTS Of the 415 children in the study (195 girls and 220 boys; mean [SD] age, 363.0 [8.3] days), a consistent association between craniofacial shape and prenatal alcohol exposure was observed at almost any level regardless of whether exposure occurred only in the first trimester or throughout pregnancy. Regions of difference were concentrated around the midface, nose, lips, and eyes. Directional visualization showed that these differences corresponded to general recession of the midface and superior displacement of the nose, especially the tip of the nose, indicating shortening of the nose and upturning of the nose tip. Differences were most pronounced between groups with no exposure and groups with low exposure in the first trimester (forehead), moderate to high exposure in the first trimester (eyes, midface, chin, and parietal region), and binge-level exposure in the first trimester (chin).

CONCLUSIONS AND RELEVANCE Prenatal alcohol exposure, even at low levels, can influence craniofacial development. Although the clinical significance of these findings is yet to be determined, they support the conclusion that for women who are or may become pregnant, avoiding alcohol is the safest option.

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Supplemental content

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Prenatal alcohol exposure (PAE) is a major preventable cause of health and developmental problems in children. It may cause irreversible damage to the brain, resulting in fetal alcohol spectrum disorder (FASD), which is characterized by learning difficulties, executive dysfunction, impaired speech, motor problems, and behavior problems.[1] Fetal alcohol spectrum disorder may affect 3% to 5% of mainstream school-aged children, with many remaining undiagnosed.[2] Fetal alcohol syndrome (FAS) is diagnosed when cognitive impairment occurs together with abnormalities of growth and a characteristic facial phenotype. Diagnostic criteria of FAS include short palpebral fissures, flat philtrum, and thin upper lip,[3] but the condition has also been associated with midfacial and auricular anomalies.[4] Detailed morphologic studies of the face have found a reduction in ear length and midfacial hypoplasia, evidenced by reduced midfacial depth and flattening of the nasal bridge and malar regions and a reduction in the size of the neurocranium and face (reduced upper facial width, head circumference, and total facial height), as well as retrognathia and micrognathia (reduced bigonial breadth), in individuals who received a diagnosis of partial FAS and in individuals with heavy PAE who do not meet the diagnostic criteria for FASD.[5-7] Furthermore, comparisons of the physical characteristics of children with FASD and the physical characteristics of children without FASD have shown that maternal drinking measures significantly correlated with facial dysmorphology, with higher levels of drinking predicting higher dysmorphology scores.[8-10] Together these findings suggest a possible dose-related association between PAE and craniofacial shape.

Most of the studies cited have used facial measurements, which capture limited information and are prone to measurement error, or used clinical examination, which may be subjective. In 2013, Suttie and colleagues[5] used objective, spatially dense morphometric techniques to analyze 3-dimensional (3-D) photographs in a social setting in which high alcohol intake is common. These techniques enable the analysis of the whole facial surface and do not require subjective assessment. In their study, FAS and partial FAS phenotypes were characterized by the difference between the average facial shape of control individuals and individuals with FAS or partial FAS. They also performed a clustering analysis of individual faces and found that some children with heavy PAE displayed anomalies consistent with FAS or partial FAS. Here, we extend this work to examine the phenotype of children who did not receive a diagnosis of FASD but who had lower levels of PAE, by using a shape regression–based approach, which allows us to control for covariates while comparing average faces statistically.[11]

Key Points

Question Is there an association between different levels of prenatal alcohol exposure and child craniofacial shape at 12 months?

Findings This cohort study conducted an objective and sensitive craniofacial phenotype analysis of 415 children, which showed an association between prenatal alcohol exposure and craniofacial shape at almost every level of exposure examined. Differences in the midface and nose resemble midface anomalies associated with fetal alcohol spectrum disorder.

Meaning Any alcohol consumption has consequences on craniofacial development, supporting advice that complete abstinence from alcohol while pregnant is the safest option; it remains unclear whether the facial differences are associated neurocognitive outcomes of prenatal alcohol exposure.

Methods

Study Population

Asking Questions About Alcohol in Pregnancy (AQUA) is a population-based longitudinal study of the neurodevelopmental outcomes in children with PAE, with a focus on low to moderate alcohol consumption.[12] Participants are 1570 women and their offspring, recruited in early pregnancy during a 12-month period from July 25, 2011, to July 30, 2012, from low-risk public maternity clinics in Melbourne, Australia. Three-dimensional craniofacial images were captured at 12 months from a subset of 517 participants. Included in this analysis are the images of 415 white children whose mothers were not lifetime abstainers and for whom we have complete information on all covariates. The AQUA study was approved by the Human Research Ethics Committees of Eastern Health (E54/1011), Mercy Health (R11/14), Monash Health (11071B), the Royal Women's Hospital (11/20), and the Royal Children's Hospital (31055A) in Melbourne, Australia. Mothers provided written consent prior to image capture.

Measurement of Alcohol Exposure

Questionnaires were collected that had detailed information on the quantity and frequency of alcohol consumption for the 3 months before pregnancy and for each trimester, including the time prior to recognition of pregnancy. Frequency of drinking and typical amount and type of alcoholic drink were combined to provide a single-exposure measure for each stage of pregnancy, expressed in grams of absolute alcohol (AA) and using algorithms previously described.[13-15]

Exposure levels were low (≤20 g of AA per occasion and ≤70 g of AA per week), moderate (21-49 g of AA per occasion and ≤70 g of AA per week), high (>70 g of AA per week), and binge (≥50 g of AA per occasion).[12] Mothers who were abstinent throughout pregnancy comprised the control group for all analyses.

Analysis used a 3-tiered approach in which PAE tier 1 consisted of children of women who drank any alcohol while pregnant. Tier 2 subdivided the exposure group into those with PAE in the first trimester only and those with PAE throughout pregnancy. Tier 3 further subdivided the exposure group into low, moderate to high, or binge-level drinking before becoming aware of pregnancy and whether exposure occurred in the first trimester only or throughout pregnancy.

Image Acquisition and Preprocessing

Medical photographers not involved in the analysis collected 3-D craniofacial images between January 8, 2013, and Febru-

ary 11, 2014, inclusive, within 2 weeks of the child's first birthday, at the Royal Children's Hospital in Melbourne, Australia. Image acquisition was via the 3dMD 7-pod system (3dMD Corp), which captures a 360° image of the head, including the face and cranium. To ensure that images were unobscured by hair and to capture the shape of the neurocranium, a tight-fitting stocking was placed over the cranial vault. A neutral facial expression was not ensured at all times, and some images were taken with the mouth open and others with the mouth closed. Mouth position in the images was subsequently standardized individually using a previously published method[16] to ensure that each image had a neutral expression.

Craniofacial Measurement

Craniofacial measurement was undertaken by a researcher (H.M.) blinded to the participants' PAE group. To represent the entire surface of the face, a spatially dense array of 69 587 points on a template 1-year-old face (derived with bootstrapping[17]) was automatically placed onto each target image by a 3-D surface registration algorithm. This process gradually warps the shape of the template into the shape of the target face,[18,19] thus sampling each face at corresponding locations across the entire surface. Each point configuration was made symmetrical.[17] The location, orientation, and size of all point configurations were standardized using generalized Procrustes analysis.[20] Fitting of the template was performed in Mevislab, a platform for medical image visualization and developing image processing algorithms (http://www.mevislab.de), using custom-built modules developed at Katholieke Universiteit Leuven, Leuven, Belgium.[19]

Covariates

Regression covariates included risk factors known to be, or that could plausibly be, associated with craniofacial shape: child's sex, which is known to be associated with early craniofacial dimorphism[16]; maternal age, a risk factor for malformations[21]; and maternal smoking in pregnancy, a risk factor for malformations of the head and face.[22] The child's birth weight and maternal prepregnancy body mass index were included as factors potentially resulting in greater fat deposition around the cheeks.

To gauge individual variation in alcohol metabolism, mothers were asked how quickly they felt the effects of alcohol (very slowly or slowly, normally, or very quickly or quickly). This variable was considered as a potential modifier of any association between PAE and craniofacial shape. Although the measure is subjective, self-perceived alcohol effects may serve as a proxy variable, coding for unknown, complex, polygenic determinants of alcohol metabolism.

Statistical Analysis

The distribution of PAE groups and covariates was described using frequency counts and percentages if categorical, and mean (SD) values if continuous. To test for a difference in craniofacial shape between the control and each PAE group in each tier, partial least squares regression models were fitted. Partial least squares regression was chosen because the point configurations are highly collinear and because there are more point coordinates than observations (faces). All models included PAE group (coded as control = 0 and PAE = 1) to model the association with PAE. The predictors listed were controlled for by including them in each partial least squares regression model. A second analysis computed the same regression models to examine any association between craniofacial differences and PAE, stratified according to self-perceived alcohol effects.

At the global and regional level, the statistical association that controlled for covariates (partial R^2) was computed. Statistical significance was computed at the global level using a permutation test with 1000 permutations.[23(p278)] $P < .05$ was considered significant. Given that analysis of almost 70 000 points results in mean differences that vary across the face, craniofacial anatomical differences associated with PAE at each point were plotted using false colormaps, which show a generic face with each point indexed in color to the amount of difference and highlighting which regions were more changed or less changed.[11,24] Partial R^2, total anatomical difference, and difference in lateral and vertical directions and depth were plotted using separate colormaps.

For partial least squares regression and permutation testing, we used a custom-written code (by H.M.) in the Python programming language. The figures were generated using the Mayavi 3D visualization library (http://code.enthought.com/projects/mayavi/).

Results

The characteristics of the children included in the analysis are summarized in **Table 1**. The cohort included 220 boys and 195 girls with a mean (SD) age of 363.0 (8.3) days at imaging. Of the 326 children with PAE, 133 (40.8%) were exposed in the first trimester only and 193 (59.2%) throughout the pregnancy.

Global and Regional Association Between PAE and Craniofacial Shape

There was no significant association between PAE and craniofacial shape at the global level (**Table 2**). There were, however, regional mean differences in craniofacial shape of children exposed to any alcohol (tier 1), regardless of whether PAE occurred in the first trimester only or throughout pregnancy (tier 2) (**Figure 1**). Regions of difference were concentrated around the midface, nose, lips, and eyes. Directional visualization showed that these differences corresponded to a general recession of the midface (Figure 1; blue areas in the depth section) and a superior displacement of points of the nose, especially the tip of the nose (Figure 1; red areas in the vertical section), indicating a shortening of the nose and upturning of the nose tip. This craniofacial phenotype was evident in both tiers 1 and 2.

In tier 3, changes were most marked with moderate to high PAE in the first trimester around eyes, midface, chin, and parietal region and with binge-level PAE in the first trimester around the lower lip. More important, a consistent phenotype comprising a recessed midface and upturned

Table 1. Characteristics of Children With Craniofacial Images Included in Analysis

Characteristic	All Children (N = 415)	PAE Tier 1 Categorization[a]		P Value[b]
		Abstinent (Control) (n = 89)	Any Alcohol (n = 326)	
Maternal, mean (SD)				
Age, y	32.7 (4.0)	32.7 (4.0)	32.7 (4.0)	.55
Prepregnancy BMI	24.7 (5.5)	25.8 (6.0)	24.4 (5.3)	.02
Maternal smoking in pregnancy, No. (%)				
No	361 (87.0)	81 (91.0)	280 (85.9)	.20
Yes	54 (13.0)	8 (9.0)	46 (14.1)	
Child birth weight, mean (SD), g	3518.6 (458.9)	3532.5 (448.5)	3514.8 (462.3)	.37
Child's sex, No. (%)				
Male	220 (53.0)	46 (51.7)	174 (53.4)	.78
Female	195 (47.0)	43 (48.3)	152 (46.6)	
Child age, mean (SD), d	363 (8.3)	365 (8.4)	363 (8.3)	.02
Maternal self-perceived alcohol effect, No. (%)				
Very slowly or slowly	28 (6.7)	5 (5.6)	23 (7.1)	.09
Normally	233 (56.1)	42 (47.2)	191 (58.6)	
Very quickly or quickly	154 (37.1)	42 (47.2)	112 (34.4)	
Alcohol exposure, No. (%)				
PAE tier 2				
Drank alcohol in T1 only[c]	NA	NA	133 (40.8)	NA
Drank alcohol throughout pregnancy	NA	NA	193 (59.2)	NA
PAE tier 3				
Low in T1, abstinent in T2 and T3	NA	NA	49 (15.0)	NA
Moderate to high, in T1, abstinent in T2 and T3[d]	NA	NA	46 (14.1)	NA
Binge in period before awareness of pregnancy, abstinent in T2 and T3[e]	NA	NA	38 (11.7)	NA
Low in T1, low to moderate in T2 and/or T3	NA	NA	39 (12.0)	NA
Moderate to high in T1, any level in T2 and/or T3[f]	NA	NA	84 (25.8)	NA
Binge in period before awareness of pregnancy, low to moderate in T2 and/or T3[g]	NA	NA	70 (21.5)	NA

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); PAE, prenatal alcohol exposure; T1, first trimester; T2, second trimester; T3, third trimester.

[a] The definition of the tiers is given in the Measurement of Alcohol Exposure subsection of the Methods.

[b] Two-sample *t* test if continuous and Pearson χ^2 if categorical.

[c] A total of 115 mothers (86.5%) drank only before awareness of pregnancy.

[d] Includes 2 mothers (4.3%) who drank at high levels.

[e] Includes 26 mothers (68.4%) with infrequent or single binge episodes totaling 70 g of absolute alcohol per week or less and 7 mothers (18.4%) with weekly binges totaling more than 70 g of absolute alcohol per week.

[f] Includes 24 mothers (28.6%) who drank at high levels.

[g] Includes 34 mothers (48.6%) with infrequent or single binge episodes totaling 70 g of absolute alcohol per week or less and 20 mothers (28.6%) with weekly binges totaling more than 70 g of absolute alcohol per week.

Table 2. Global Association Between Prenatal Alcohol Exposure and Craniofacial Shape

Prenatal Alcohol Exposure[a]	No.	Partial R^2, %[b]	P Value
Tier 1			
Drank alcohol while pregnant	326	0.3	.26
Tier 2			
Drank alcohol in T1 only	133	0.5	.24
Drank alcohol throughout pregnancy	193	0.3	.47
Tier 3			
Low in T1, abstinent in T2 and T3	49	0.8	.31
Moderate to high in T1, abstinent in T2 and T3	46	1.4	.06
Binge in period before awareness of pregnancy, abstinent in T2 and T3	38	0.5	.72
Low in T1, low to moderate in T2 and/or T3	29	0.8	.41
Moderate to high in T1, any level in T2 and/or T3	84	0.6	.37
Binge in period before awareness of pregnancy, low to moderate in T2 and/or T3	70	0.4	.70

Abbreviations: T1, first trimester; T2, second trimester; T3, third trimester.

[a] The definition of the tiers is given in the Measurement of Alcohol Exposure subsection of the Methods.

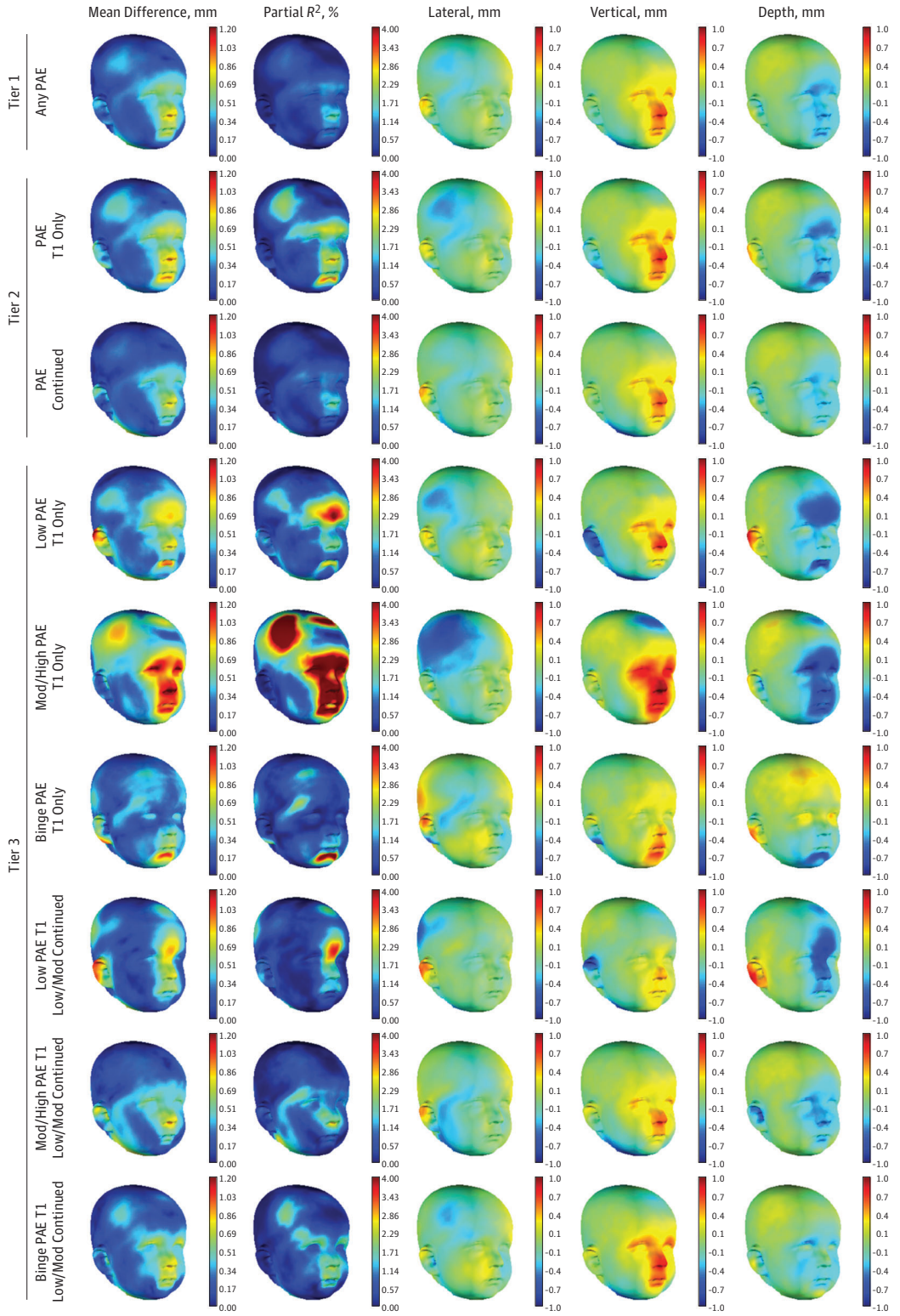
[b] Compared with controls (ie, abstinent throughout pregnancy). Adjusted for maternal age, prepregnancy body mass index, smoking during pregnancy, child's sex, and birth weight.

nose was evident in all exposure categories except in those with binge-level exposure in the first trimester only, where the recession was confined to the chin (retrognathia). Overall, point differences reached a maximum of 1.2 mm between groups.

Stratified Regional Association Between PAE and Craniofacial Shape

The partial least squares regression analysis was repeated, stratified by maternal report of feeling the effects of alcohol normally and very quickly or quickly (omitting those who

Figure 1. Regional Associations Between Prenatal Alcohol Exposure (PAE) and Craniofacial Shape



Mean difference highlights anatomical regions that are most different between the groups; Partial R^2 is plotted per vertex; Lateral, Vertical, and Depth show the magnitude of the displacements in each direction. In the lateral portion, red indicates right displacement and blue indicates left displacement. In the vertical portion, red indicates superior displacement and blue indicates inferior displacement. In the depth portion, red indicates anterior displacement and blue indicates posterior displacement. All analyses are adjusted for maternal age, maternal prepregnancy body mass index, maternal smoking during pregnancy, and child's birth weight and sex. The definition of the tiers is given in the Measurement of Alcohol Exposure subsection of the Methods. Low/Mod indicates low to moderate; Mod/High, moderate to high; and T1, first trimester.

Table 3. Global Association Between Prenatal Alcohol Exposure and Craniofacial Shape, Stratified by Self-perceived Alcohol Effect

Prenatal Alcohol Exposure[a]	Self-perceived Alcohol Effect[b]					
	Normally			Quickly or Very Quickly		
	No.	Partial R^2, %[c]	*P* Value	No.	Partial R^2, %[c]	*P* Value
Tier 1						
Drank alcohol while pregnant	191	0.3	.52	112	0.1	.12
Tier 2						
Drank alcohol in T1 only	72	0.9	.37	55	0.7	.74
Drank alcohol throughout pregnancy	119	0.6	.45	57	2.9	.005
Tier 3						
Low in T1, abstinent in T2 and T3	22	2.4	.14	26	1.3	.47
Moderate to high in T1, abstinent in T2 and T3	24	1.5	.38	19	2.5	.11
Binge in period before awareness of pregnancy, abstinent in T2 and T3	26	1.7	.26	10	2.6	.18
Low in T1, low to moderate in T2 and/or T3	18	1.5	.47	20	3.4	.04
Moderate to high in T1, any level in T2 and/or T3	58	1.3	.22	20	4.2	.006
Binge in period before awareness of pregnancy, low to moderate in T2 and/or T3	43	0.7	.80	17	2.6	.12

Abbreviations: T1, first trimester; T2, second trimester; T3, third trimester.

[a] The definition of the tiers is given in the Measurement of Alcohol Exposure subsection of the Methods.

[b] Excluded from analysis are 28 images where mothers reported feeling the effects of alcohol slowly or very slowly.

[c] Compared with controls (ie, abstinent throughout pregnancy). Adjusted for maternal age, prepregnancy body mass index, smoking during pregnancy, and child's sex and birth weight.

reported feeling the effects of alcohol slowly or very slowly owing to small group numbers). For children of those who felt the effects of alcohol normally, there was no significant global association in any exposure category (**Table 3**). Overall, the craniofacial phenotype was similar to that in the unstratified analysis but was more pronounced in the stratum of mothers who reported feeling the effect of alcohol very quickly or quickly. There was also a significant association at the global level in those who drank throughout in the tier 2 and tier 3 analyses (with the exception of the binge-level category). Regional associations with PAE (**Figure 2**) reached a difference of 1.6 mm in the forehead and midface in the stratum of mothers who reported feeling the effect of alcohol very quickly or quickly.

Association of Covariates With Craniofacial Shape

There were significant global differences in craniofacial shape associated with maternal age, prepregnancy body mass index, children's birth weight, and child's sex. Maternal smoking during pregnancy was not associated with craniofacial shape at a global level. The tier 1 regression model showing these associations is in eTable in the Supplement (global associations) and eFigure in the Supplement (regional associations).

Discussion

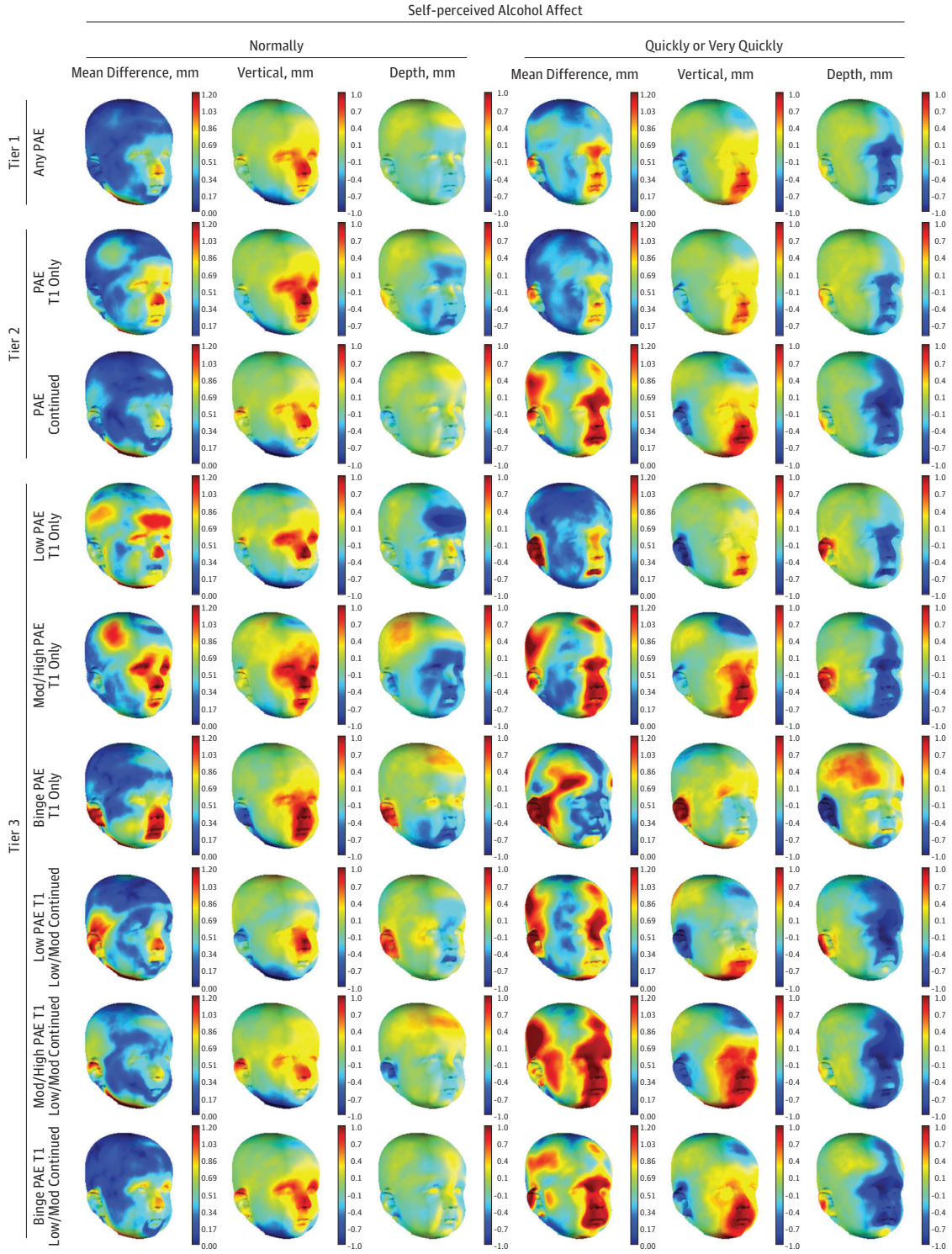
To our knowledge, this study is the first to examine the association between the face of the child and common patterns of PAE, using objective, holistic methods of craniofacial phenotyping. A consistent association with craniofacial shape was observed in almost all exposure groups, with differences concentrated on regions around the nose, eyes, upper lips, and lower lips. Results indicate a mild midfacial recession suggestive of subclinical hypoplasia and an upturning of the nasal tip in those exposed to alcohol prenatally. This phenotype was evident even when drinking was at a low level and mothers ceased alcohol consumption in the first trimester.

In the unstratified analysis, we observed the strongest association with facial shape in children of mothers who drank at moderate levels in the first trimester only. In the stratified analysis, craniofacial differences were strongest in children of mothers who said they felt the effects of alcohol quickly, particularly if they continued drinking throughout pregnancy and initially drank at moderate levels. The apparent discrepancy between the 2 analyses may be because fewer women who reported feeling the effects of alcohol quickly drank throughout pregnancy.[15] A higher proportion of mothers who felt the effects of alcohol normally in those exposure groups may have masked the association in the unstratified analysis.

The PAE group with binge-level alcohol consumption comprised 50% to 70% of mothers with infrequent or single-binge exposures, while in 20% to 30% of mothers, the binge exposure occurred weekly or more often. This heterogeneity may explain why we did not see a strong association between binge-level PAE and craniofacial shape.

Facial abnormalities have previously been described in children with PAE who do not meet diagnostic criteria for FAS. Suttie et al[5] analyzed 69 children whose mothers consumed up to 13 standard drinks (14 g of AA) per week or drank at binge levels and found that 28 children showed patterns of facial anomalies similar to those seen in FAS or partial FAS. These anomalies were also associated with cognitive impairments. In their group comparisons, those authors noted an elevation of the lower portion of the nose in those with FAS, as well as smoothing of the philtrum,

Figure 2. Regional Associations Between Prenatal Alcohol Exposure (PAE) and Craniofacial Shape, Stratified by Self-perceived Alcohol Effect



Mean difference highlights anatomical regions that are most different between the groups. Vertical and Depth show the magnitude of the displacements in each direction. In the vertical portion, red indicates superior displacement and blue indicates inferior displacement. In the depth portion, red indicates anterior displacement and blue indicates posterior displacement. All analyses are adjusted for maternal age, maternal prepregnancy body mass index, maternal smoking during pregnancy, and child's birth weight and sex. The definition of the tiers is given in the Measurement of Alcohol Exposure subsection of the Methods. Low/Mod indicates low to moderate; Mod/High, moderate to high; and T1, first trimester.

reduced palpebral fissure length, midfacial hypoplasia, and retrognathia. The only previous study to examine low levels of PAE found that 66% of 79 newborns in the exposure group had some facial abnormality.[25] However, observed abnormalities were not objectively defined and assessed by unblinded observers. Moreover, the weekly mean intake of alcohol per trimester was reported retrospectively, and it is not possible to know if there were spikes in consumption at particular time points, including the first trimester and the period before recognition of pregnancy. Our study has examined the association of low levels of PAE using a more rigorous assessment of both face shape and alcohol consumption. We show that certain aspects of the phenotype (upturning of the nasal tip and midfacial hypoplasia) can be detected even when the maximum number of standard drinks (10 g of AA) did not exceed 7 per week and 2 per occasion. We did not observe the classic diagnostic features for FAS of smooth philtrum, reduced palpebral fissure length, and thin vermillion of the upper lip; it is likely that these features emerge only at higher levels of exposure.

During embryogenesis, facial bone and cartilage are derived from the cranial neural crest. Sizing and positioning of facial elements begins 17 to 18 days after fertilization and before most pregnancies are recognized.[26] Evidence from mouse studies shows that exposure to ethanol affects all stages of neural crest development, resulting in variation in craniofacial appearance, depending on the gestational timing of exposure. For example, alcohol exposure at gestational day 7 (the 15th-17th day in human development[27]) leads to severe midfacial hypoplasia, an elongated upper lip, and a deficient philtrum, while exposure at day 8.5 causes mild midfacial hypoplasia, a shortened upper lip, and a preserved philtrum.[28] We observed a similar facial phenotype to that seen in animal models, particularly after first-trimester moderate PAE. Although it was not possible to localize the timing of exposure as precisely as in these animal studies, our findings confirm an association between moderate PAE and facial shape in the first trimester in humans, which is convergent with the animal evidence.

Even in children of mothers who drink heavily, facial abnormalities associated with PAE are highly variable,[5] which may reflect variation in the timing of exposure in the first trimester or unmeasured risk factors. For example, genetic variants in maternal and fetal alcohol metabolism have been shown to influence the level of alcohol and/or its toxic metabolites experienced by the fetus. Mendelian randomization studies using the Avon Longitudinal Study of Parents and Children predicted different patterns of PAE and variable outcomes in offspring depending on several variants in the alcohol dehydrogenase gene in the mother or child (eg, *ADH1B* [OMIM 103720], predictive of reduced alcohol consumption and associated with higher academic achievement in offspring).[29,30] Furthermore, regarding the direct role of genes involved in alcohol metabolism in modifying risk, evidence from laboratory studies using ethanol-sensitive and ethanol-resistant chickens, mice, and zebra fish provides insight into the multifactorial genetics of ethanol-mediated cell signaling disruption and neural crest apoptosis.[31]

We observed that children of mothers who reported feeling the effects of alcohol quickly or very quickly exhibited larger craniofacial differences in most exposure groups. We hypothesize that this rate of feeling the effects of alcohol reflects genetically determined variation in alcohol metabolism. The rate of feeling the effects of alcohol may ultimately be clinically useful to differentiate individuals with a greater susceptibility to the effects of alcohol, conferring increased fetal vulnerability, and may in part explain the heterogeneity of outcomes in alcohol-exposed pregnancies and in FASD.

Craniofacial development closely corresponds to brain induction and expansion, and, as such, characteristic facial differences have been linked to brain abnormalities and cognitive outcome in FASD.[31] Correlative face-brain phenotypes have been described in human and animal studies, suggesting that the type and severity of brain abnormality may be predicted in part by hypoplasia of the midface,[28,32] and that the classic facial features of FASD (short palpebral fissure, smooth philtrum, and thin upper lip) may assist in identifying children at risk of developing neurobehavioral deficits.[5] Given that cognitive outcomes for the children in our study have not yet been examined in this context, it is as yet unknown if the craniofacial differences found are of diagnostic or predictive value.

Strengths and Limitations

This study is a well-described cohort of mother-child dyads with detailed PAE data and classification not available in many other studies. The method of craniofacial phenotypic analysis used in this study is the most objective and sensitive available, to our knowledge. Although the magnitude of the association was variable, we observed very similar anatomical differences using almost every PAE categorization, which essentially constitutes several replications of the findings and demonstrates their robustness. A link between these facial changes and brain structure and functioning remains to be investigated; in the meantime, we provide additional evidence for an association between alcohol consumption and the developing fetus.

We did not observe the auricular anomalies previously documented with low-level PAE in other studies.[4] Owing to the geometry of the multiple cameras used for imaging and the anatomy of the ear, it was not possible to capture high-quality images of the ear, and the analysis may have been obscured by photographic noise in this region.

We postulate that the rate of feeling the effects of alcohol is a proxy for metabolic factors influencing PAE and its association with facial shape, but have no direct measure such as blood alcohol concentration or alcohol elimination rates to examine. Investigation of maternal and/or child allelic differences (genotypes) at specific genes associated with alcohol metabolism or alcohol use behavior is under way.

Conclusions

The results of this study suggest that even low levels of alcohol consumption can influence craniofacial development of

the fetus and confirm that the first trimester is a critical period. We observed aspects of a craniofacial phenotype with almost any level of PAE, something previously only documented following a high level of long-term alcohol exposure. Although the clinical significance of our findings is yet to be determined, these findings support the conclusion that, for women who are, or may become pregnant, avoiding alcohol is the safest option.

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Appendix 3: Letter to Ministers regarding Clear Alcohol Labelling

Please note that any identifying information within this email is not to be used for public viewing.

Dear Minister,

My name is [redacted]. I'm Chair of The Board of NOFASD Australia and the parent of a teenager living with FASD (Fetal Alcohol Spectrum Disorder). I'm am also a constituent of your electorate, as I live in Tatura. I am asking for your support **this Friday 17th July, 2020,** to endorse an evidence-based red, black and white warning label on alcoholic beverages, which has been recommended by Food Standards Australia New Zealand. The health warning is to pregnant women and the label clearly identifies that it is unsafe for the mother of an unborn baby to drink alcohol whilst pregnant. Anything less than this clear labelling is failing to protect as many unborn children from the debilitating lifetime affects this can bring.

My husband and I adopted our son 15 years ago. He was diagnosed with FASD at the age of 10. FASD is a direct result from exposure to a baby in-utero. Every age and every stage of our son's development has been challenging. Challenges that many would barely believe possible. Challenges that turn your values into thin ice, so fragile that you're scared to remember them. You probably have them too: respect, honesty, fairness, trust, forgiveness. Our style of parenting has become a continual adaption to our son's needs, including how we include our values in a non-confronting way for our son.

Our son has poor executive functioning, due to the brain damage caused by alcohol in utero. This means he is unable to process so much of the information around him and it turns his world into turmoil.

- His mind continually races because in everyday life there too many things going on for him to try and take in. This occurs because the front part of his brain has been damaged through in-utero alcohol exposure. He struggles to process just one thing at a time. His brain is overloaded and screaming, it's frightening….so he lashes out.
- When he tries to remember what he was just asked to do, he gets upset that he can't remember what it was….so he lashes out.
- Transferring information in writing from a book onto paper, for example, is a nightmare. His brain can see the word(s) but organising them to re-write is painful to watch. He gets frustrated, objects to doing this task, (which is often

a part of his schooling) and he destroys things. There often is no warning of his frustration.

There are always variances to the degree of severity with these behaviours, but they range from objecting and yelling to severe aggression, both physically and verbally. There is also property damage.

My husband and I employ strategies that have been significantly refined for our son, but even these can be no match for the brain damage he has suffered. It's important to note that this damage has occurred during development, not after, so the case of neuroplasticity repair does not apply. This is lifelong.

We have significantly modified our home, to reduce anxiety both for our son and us. Food, chemicals, cutlery…anything that could pose a risk, is under lock and key. His impulsive behaviours (that also occur due to damage during development in-utero), are too great to risk anything and everything being available to him. Add to this the additional factor of a high IQ, something many do not realise is possible for someone living with FASD. Our son can formulate a highly developed plan, which if able to carry out, would be dangerous. He does not have the filter that says, "I shouldn't do this, it's risky". Supervision is completely necessary at all times.

Whilst our son has a high IQ, his executive functioning deficits prevent him learning at the level his intelligence requires. Can you imagine the frustration this creates? Again, this is a trigger for out of control behaviour. There is no school that can cater to his needs, so I Home Educate him, using a strengths-based curriculum. This doesn't mean that our days are plain sailing, they're not. However, by developing his own learning plan, based upon his strengths, the outcomes we achieve are much higher than he could possibly achieve in a mainstream school. So, when I say our care is 24/7, it really is 24/7, 365 days a year.

The last thing I would like to share with you today is how our son feels about living with FASD.

- He hates having out of control behaviour and what he says and does during that time.
- His remorse is so great that depression and thoughts of self-harm occur.
- The physical and mental fatigue of just constantly working so hard to process information takes its toll.

So why do we as parents continue to do what we do? We love our son, and this alone gives us the strength to continue every day to create the best outcomes

possible for him, for us and our family. He is family and our bonds are greater than anything.

Finally, I ask you…. Would you wish this for any child, any family? This could be someone in the future that is close to you…Imagine it. You have a chance to prevent this for many children in the future, by voting for the clear red, black and white labelling or encouraging your colleagues who will be part of this vote on Friday.

There simply is no choice here, the evidence based red, black and white labelling, must become mandatory on all alcoholic beverages. It needs to be seen, not blended in with the remainder of the label. How many women will actually read the ingredients on an alcoholic drink container? Not many! So, if a watered-down warning is blended in with this information, it is rarely going to be seen. The warning **needs** to be seen, to prevent as many children of the next generation from being born with FASD, as possible.

Thank-you for taking the time to read my story. **I urge you to contact me to discuss our situation and FASD further**. My contact details are below. I now encourage you to watch the short video below of my husband [redacted] and I, speaking of our support for the clear labelling.

[redacted]

Warm Regards

[redacted]
NOFASD Australia