

study and that the diagnostic test used was not sensitive enough to detect subtle alcohol-induced damage in the children. Dr. Casiro stated that "I believe it is wrong and inappropriate to take that study based on those findings, to say that it is safe for a pregnant woman to have seven or eight drinks a week during pregnancy."<sup>26</sup>

The question of heredity and/or race as factors in higher or lower susceptibility to FAS or FAE was raised a number of times during our hearings. No witness was able to provide information that either heredity or race was a factor in the incidence of either condition. In his book on FAS, Abel notes that ethanol, the intoxicating component in alcoholic beverages, is primarily metabolized in the body to acetaldehyde through the action of the enzyme, alcohol dehydrogenase. The acetaldehyde itself is then broken down in a series of reactions.

It is known that alcohol dehydrogenase exists in a number of genetically different variants, resulting in different levels of acetaldehyde production after consumption of alcohol. Also, acetaldehyde is considerably more toxic to humans than is ethanol. Abel states that it is possible that "the genetic variations in acetaldehyde production or metabolism may be a critical maternal risk factor for foetal alcohol effects."<sup>27</sup> It is not known, however, if this is a significant factor in the incidence of FAS or FAE in different social or racial groups.

In the absence of definitive evidence on the question of dose-response relationships in foetal alcohol syndrome and foetal alcohol effects, the Sub-Committee believes that a pregnant woman should abstain from alcohol throughout her pregnancy. We also believe that the father can assist the mother by also abstaining from alcohol during the pregnancy. We believe this position is particularly appropriate in the matter of foetal alcohol effects. It appears that FAE may be caused by smaller amounts of alcohol than is full-blown FAS.

The Sub-Committee received very pertinent testimony on this point from one of the witnesses who appeared before us:

" . . . I would like to tell you briefly of my own experience with FAE. . . .in 1988 I began to come to the conclusion that my first born child was foetal alcohol affected.

"I worked throughout my pregnancy, ending my work day with a commute to my middle class home in the suburbs. During the meal preparation I often would have one drink. I was vigilant about not taking any medication, and had quit smoking prior to my pregnancy.

"During my pregnancy I asked my doctor about alcohol. In 1968-69 his knowledge was that a couple of drinks were fine. My son Jeff was born on February 11, 1969, a healthy baby. He appeared bright, and developmentally he kept up with his peers. His attention span was very short, however, and he was a very busy child. I rejected the term hyperactive.

"By the time he was three, he had had stitches three times. He was fearless, and did not seem to learn from his falls. In grade 2 he was identified as learning disabled, and he repeated grade 2. The painful years of testing, learning assistance, and special education classes began. During this time I worked very hard to be an advocate for him in the school system, and I also tried to help him maintain his self-esteem.

"He told me when he was 16 that, after watching a public broadcasting system program on learning disabilities, he sat and cried. With much support he completed high school at age 19. He began working in construction. His well-developed social skills, his charm and wit made hiring easy for him, plus he had a resumé at the ready that masked his grade 4 reading level.

<sup>26</sup> Proceedings, Issue 8, p. 21.

<sup>27</sup> Abel (1990), pp. 39-41.