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Sommario/riassunto	Women drinking during pregnancy can result in Fetal Alcohol Spectrum Disorder (FASD), which features neurodevelopmental deficit, facial dysmorphology, growth retardation, and learning disability. Research suggests the human brain is precisely shaped through an intrinsic, genetic-cellular expression that is orchestrated further upstream by an epigenetic program. This program can be influenced by environmental inputs such as alcohol. Current research suggests the genetic and epigenetics of FASD are becoming intertwined and inseparable. Now is the time for investigators to combine genetic, genomic and epigenetic alcohol research into an accessible, online platform discussion. Genetic analyses inform gene sets vulnerable to alcohol exposure during early neurulation. Prenatal alcohol exposure alters expression of gene subsets, including genes involved in neural specification, hematopoiesis, methylation, chromatin remodeling, histone variants, eye and heart development. Recently, quantitative map locusing (QTLs) that mediate alcohol-induced phenotype were identified between two mouse strains. Another question is -- besides amount, dose, and stage of alcohol exposure, why only 5% of women drinking have a newborn with FAS? Studies are also ongoing to answer this question by

characterizing genome-wide expression, allele-specific expression (ASE), gene polymorphisms (SNPs) and maternal genetic factors that influence alcohol vulnerability. Alcohol exposure during pregnancy, which can lead to FASD, has been used as a model to resolve the epigenetic pathway between environment and phenotype. Epigenetics modifies genetic outputs through alteration of 3D chromatin structure and accessibility of transcriptional machinery. Several laboratories have reported altered epigenetics, including DNA methylation and histone modification, in multiple models of FASD. During development DNA methylation is dynamic, yet orchestrated as methylation progresses in a precise spatiotemporal manner during neurulation and coincides with neural differentiation. Alcohol can directly influence epigenetics through alterations of the methionine pathway and subsequent DNA or histone methylation/acetylation. Alcohol also alters noncoding RNA including miRNA and transposable elements (TEs). Evidence suggests that miRNA expression may mediate ethanol teratology, and TEs may be affected by alcohol through altering DNA methylation at LTR. In this manner epigenetic and genetics of FASD are becoming mechanistically intertwined. Can alcohol-induced epigenomic alterations be passed through generations? Early epidemiological studies revealed infants with FASD-like features in the absence of maternal alcohol, where the fathers were alcoholics. Novel mechanisms for alcohol-induced phenotypes include altered sperm DNA methylation, hypomethylated paternal allele and heritable epimutation. These studies predict heritability of alcohol-induced epigenetic abnormalities and gene functionality across generations.