PRIME For Life Documentation

Biological Research

1. Adoption Research
2. Twin Research
3. Positive Family History: Alcohol and Drug Use and Problems
4. Animal Studies
5. Genes, Markers, and Biochemistry
6. Neurobiology of Drug Response and Addiction
7. Tolerance (Low Response) as a Risk Factor
8. EEG—Brain Waves: A Possible Risk Indicator
9. Other Alcohol Responses that Might Mediate Risk
10. Flushing: A (Primarily) Protective Factor
11. Theorized Role of TIQS (Thiq)—apparently not an explanation for addiction

Biological Issues in Impairment

12. Alcohol Absorption & Distribution
13. Alcohol Metabolism & Elimination
14. Tolerance
15. Individual Differences-Altitude
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Psychological Influences and Outcomes

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Biological Research

1. **Adoption Research**


2. *Twin Research*


3. **Positive Family History: Alcohol and Drug Use and Problems**


4. Animal Studies


5. **Genes, Markers, and Biochemistry**


6. **Neurobiology of Drug Response and Addiction**


7. **Tolerance (Low Response) as a Risk Factor**


8. **EEG—Brain Waves: A Possible Risk Indicator**


9. **Other Alcohol Responses that Might Mediate Risk**


VanDusen, & S. Mednick (Eds.), *Longitudinal research in alcoholism* (pp. 107-124), Boston: Kluwer-Nihoff.


10. Flushing: A (Primarily) Protective Factor


11. Theorized Role of TIQS (Thiq)—apparently not an explanation for addiction


Biological Issues in Impairment

12. *Alcohol Absorption & Distribution*¹¹


13. **Alcohol Metabolism & Elimination**


14. **Tolerance**


15. Individual Differences—Altitude


16. **Individual Differences-Age**


17. **Individual Differences-Gender**


**Psychological Influences and Outcomes**

18. *Neuropsychology*¹²


19. **Perception of Risk**


20. **Attitudes and Expectancy**


21. **Personality: Sensation Seeking, Rebelliousness, etc.**


22. **State Dependent Learning**


23. **Stress, Stressors, & PTSD**


24. Depression


25. **Antisocial Personality Disorder**


26. **Other Psychiatric Diagnosis**


27. **Drinking Driver Traits**


**Social Influences**

28. **Availability**


29. **Gender Issues (also see 17)**


30. **Family Influences**


31. Religious Influences


32. **Perception of Norms/Peer Influences**


33. **Cultural Differences and Influences**


**Alcohol and Drug Use Across the Life Span**

34. **Prenatal Exposure as a Predictor of Later Problems**


35. **Age of First Use as a Predictor of Later Problems**


36. **Adolescent Alcohol and Drug Use and Problems**


37. **College Alcohol and Drug Choices**


38.  *Adult Alcohol and Drug Choices*


39. **Longitudinal Research on Drinking or Alcoholism**


40. *Longitudinal Research on Illegal Drug Use*


**Quantity/Frequency Outcomes**

41. **Overall Morbidity and Mortality**


problems in multiple studies: A research synthesis from the collaborative alcohol-related longitudinal project. *Addiction, 89*, 1143-1156.


41.34. Göransson, M., & Hanson, B. S. (1994). How much can data on days with heavy drinking decrease the underestimation of true alcohol consumption. *Journal of Studies on Alcohol, 55*, 695-700.


42. **Cardio Vascular Disease**


42.111. Sher, L. (2003). Effects of heavy alcohol consumption on the cardiovascular system may be mediated in part by the influence of alcohol-induced depression on the immune system. *Medical Hypotheses, 60*, 702-706.


43. **Liver and Pancreas Conditions**


44. **Brain, Cognitive Functioning & Nervous System**


44.18. Dlugos, C. A., & Pentney, R. A. (1997). Morphometric evidence that the total number of synapses on purkinje neurons of old 344 rats is reduced after long-term ethanol treatment and restored to control levels after recovery. *Alcohol & Alcoholism, 32*, 161-172.


45. **Adolescent Brain**


46. *Cancer*


47. **Birth Defects: FAS, ARND, & ARBD**


48. **Dependence & General Problem Status**


49. **Impairment**


50. *Traumatic Injury and Death*


51. *Alcohol Impaired Driving (also see 27 and 52)*


Drugs

52. Drugs and Driving


of the 13th International Conference on Alcohol, Drugs and Traffic Safety. Adelaide, Australia.


53. **Marijuana: Dependence/Addiction**


54. **Marijuana: Other Risks**


55. **Stimulants**


56. **General**


Phases

57. Alcoholism Definition and Diagnosis


58. *Dependence: Definition and Diagnosis*


59. **General Issues Relevant to Phases**


PRIME for Life Program and Model

60. **Scientific Publications & Reports Specific to PRIME for Life**


61. **Persuasion and Change Theory**


62. Learning and Teaching


1 Family incidence studies established the need for research that could separate heredity and environment (nature and nurture). Adoption research filled that need and, in turn, lessened the need for further studies on family incidence. Of the four major adoption studies that have been done on alcoholism, only the first (Roe, 1944) failed to show a genetic impact. That study failed to rigorously define alcoholism. The later, better designed studies all concur that alcoholism runs in families due mostly to heredity rather than the effect of being raised with a parent who has alcoholism. Each study also indicates that problem drinking that does not meet strict standards for defining alcoholism might not be genetically influenced.

2 Twin research has confirmed that risk for alcoholism can be inherited. It has also confirmed that alcoholism itself is not inherited. That is, risk for alcoholism is genetically established, but development of alcoholism is not under complete genetic control. People are not born with alcoholism, they are born with a level of risk for developing alcoholism. Other factors determine whether or not alcoholism actually develops. Quantity and frequency of drinking plays that role. Researchers have now turned their attention from “Does alcoholism run in families?” to “What is inherited that explains why alcoholism runs in families?” The primary means of answering that question is research comparing children who have birth parents with alcoholism and children who do not have alcoholism in their birth families. However, twin research is a continuing means of determining the extent of genetic influence on a variety of alcohol responses and outcomes. Thus far, this research has demonstrated that risk for a variety of alcohol-related health problems, rates of metabolism, initial tolerance level, and quantity and frequency of drinking are all influenced, each in varying degrees, by heredity. (DOC. 2)

3 Early studies on family incidence were important in establishing that alcoholism does run in families. However, by themselves, family incidence research cannot tell whether alcoholism runs in families due to heredity or environment. Once adoption and twin studies established a genetic link in addiction, family incidence studies began examining differences in families with a history of alcoholism and those without. In spite of high tolerance being common among children whose parents have alcoholism, a number of studies indicate that, as a group, children whose parents have alcoholism do not drink more than children whose parents do not have alcoholism through adolescence and young adulthood. There is some indication that they do begin drinking at an earlier age and that they experience more problems on the same amount of drinking.

4 Animal research is an important means of studying the effects of heredity and identifying the way in which alcohol and drugs work in the brain and nervous system. Selective breeding has produced mouse and rat strains that are either addiction prone or addiction resistant, have low tolerance or high tolerance, and are alcohol preferring or non-alcohol preferring. (DOC. 4)

5 There is no single gene for alcoholism or addiction. Researchers have found multiple “candidate” genes that seem to be related to increased risk for alcoholism. (DOC. 5.35, 5.37, 5.39)

Genetic markers are things that accompany increased risk, but do not explain the increase. They could be thought of a “genetic baggage” that come along with the same gene(s) that might increase risk for alcoholism. Their benefit is that they are visible without doing genetic testing. At this point, much research on the biochemistry of addiction primarily focuses on neurotransmitters and related chemicals. (DOC. 5)
Research to determine what is inherited that increases risk for alcoholism has led to a number of important findings since 1977. Thus far, research has indicated that high tolerance, or low response to alcohol, as a young adult is a very powerful biological predictor of risk for future alcoholism. By itself it appears to be a better predictor than family history of alcoholism. However, children of alcoholics are more likely to have a higher tolerance, and in combination with family history, high tolerance is an extremely powerful predictor. (DOC. 7)

Research has demonstrated a variety of EEG differences both in people who have alcoholism, and in their children. The meaning of the differences is not completely clear. Research on alpha and beta waves may indicate that alcohol causes greater relaxation or a more positive subjective response in COAs. Research on the P300 may be a measure of disinhibition. (DOC. 8)

Research has demonstrated that children whose birth parents have alcoholism do not metabolize alcohol at a different rate than other people. They do, however, demonstrate a variety of other differences in tension response, body sway, heart rate, and stress hormones. (DOC. 9)

Some people experience a facial flush when drinking small amounts of alcohol. This can be mild, moderate or severe. flushing is especially common among some groups of people of Asian descent. Those with a severe flush have very low rates of alcoholism thus, it could be thought of a biological protection. There is some evidence that a mild flush may increase risk for alcoholism, at least among Asians. The flush is caused by a buildup of acetaldehyde, which has led to the study of acetaldehyde levels in people with alcoholism and their children. (DOC. 7.15, 44.21)

TIQs (or THIQs) are widely discussed in the alcoholism field and you can find extensive references in the bibliography. However, the role they play in alcoholism, if any, has been a matter of debate among scientists. At this point, there is general consensus that the role is minimal, if at all. It seems that our field has been guilty of much over-statement about the role of TIQs (THIQ) in explaining alcoholism. (DOC. 11)

Rates of absorption, distribution and metabolism (and a small amount of excretion) determine the number of drinks it takes to reach any blood alcohol level. Tolerance is a measure of the degree of impairment at any blood alcohol level. Together, these processes explain the great variety in individual response to alcohol. (DOC. 12)

Additional explanation of why children whose parents have alcoholism may experience more problems with alcohol may come from research on neuropsychology. This research demonstrates that some subtle, but important, differences occur in brain functioning both for people with alcoholism and their children. Some of the problems that have been attributed to either heavy drinking or to the effects of living with a parent with alcoholism may actually be “genetic baggage” that accompanies increased biological risk for alcoholism. (DOC. 18)