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Developmental models of drinking in youth: Where to they begin and where should they end?

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Developmental models of (problem) drinking in youth: Where do they begin and where should they end?

30 years ago

The Dominant Theory of Adolescent Drinking Origins

- **Begins in early adolescence or late middle childhood**
- **A social behavior, brought on by the status changes of adolescence**
- **Triggered by increased availability, peer use, and peer pressure to use**
- **Enhanced by involvement in social relationships and contexts where use is high**

The Dominant Theory of the Development of Alcoholism

- **Core genetic diathesis (diatheses); runs heavily in families**
- **Triggered by first drinking experiences**
- **Pharmacokinetics differ. Absorption, distribution, and metabolism of ethanol differ**

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- **Core genetic diathesis (diatheses); runs heavily in families**
- **Triggered by first drinking experiences**
- **Pharmacokinetics differ. Absorption, distribution, and metabolism of ethanol differs**
- **Pharmacodynamics differ. Subjective and objective response to ethanol is different – lower sensitivity of response, more reward value**

The core characteristics of the phenotype are alcohol related mechanisms

….. and the relevant environmental contexts are those which titrate use (availability, modeling of use by others, lack of a social control system which provides penalties for use)

The More Modern Version of Adolescent Drinking Origins

- **Involvement mediated by precursive cognitions about drinking (expectancies)**
- **Ergo, drinking is action upon an object, and cognitions about the object are precursive elements in the action sequence**

The More Modern Version of Adolescent Drinking Origins

- **Involvement mediated by precursive cognitions about drinking (expectancies)**
- **Ergo, drinking is action upon an object, and cognitions about the object are precursive elements in the action sequence**
- **The beginnings of a developmental model…**
- **But still an alcohol specific model**

My goals

- **To parse the core problem alcohol phenotype**

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- **To parse the core problem alcohol phenotype**

  To parse: To resolve into component parts, and describe them.

  WEBSTER'S Ninth New Collegiate Dictionary (1991)

- **Using these components, to present a developmental aggregation model from early childhood through adolescence into early adulthood**
- **To present early findings on the prediction of alcohol use disorder utilizing this framework**

Characteristics of the core phenotype

a) **multidimensional**, involving alcohol specific and nonalcohol-specific but predisposing risk elements

b) **cumulative**, involving the aggregation of risk for an alcohol problem end point when multiple component risk factors are present

c) Development always occurs in context; therefore, **contextual** factors should also contribute to phenotypic emergence

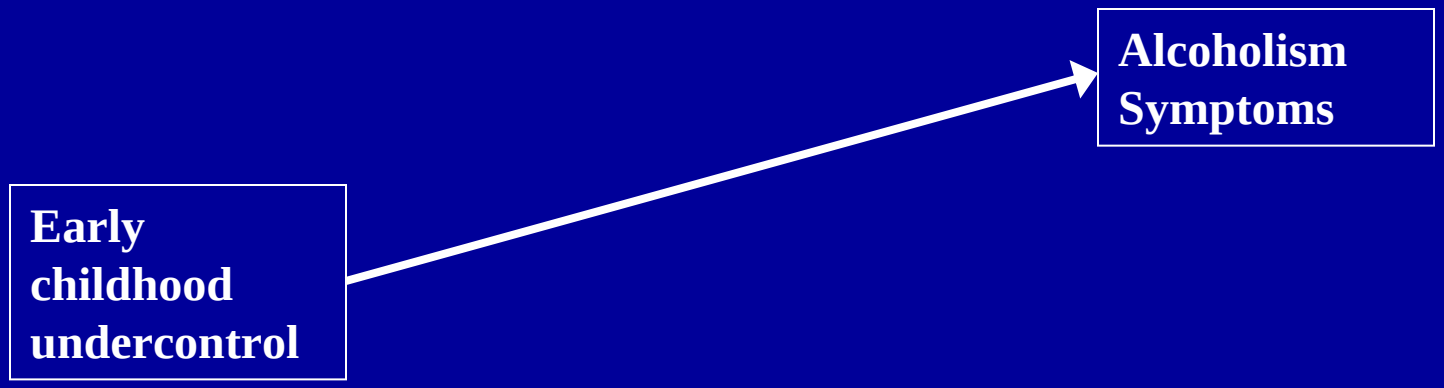
d) since aggregation occurs across time, the phenotype also has the developmental attribute of **epigenesis**…involving increasing complexity of structure over the course of development

Content Characteristics of the model: A: Nonalcohol specific predisposing risk elements: I

- **Externalizing behaviors involving… undercontrol, impulsivity, high activity, aggressiveness, rule breaking**

UNDERCONTROL: the tendency to express rather than contain one's impulses and behaviors.

Predisposing non-alcohol specific risk pathway I

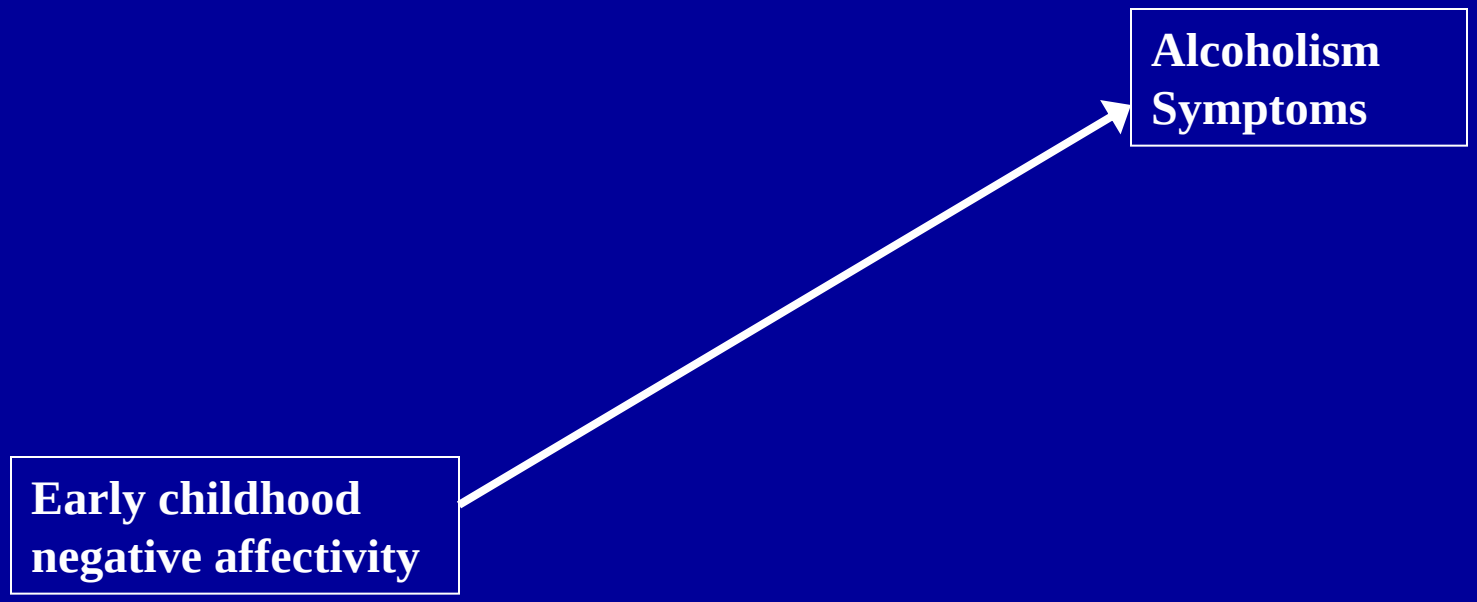


Content Characteristics of the model: A: Nonalcohol specific predisposing risk elements: II

- **Negative affectivity involving… internalizing behaviors… sadness, anxiety, depression, social inhibition**

NEGATIVE AFFECTIVITY: Responsivity to negative emotional stimulation, greater negative emotional response, a propensity to label events as negative or to focus on the negative aspects of events.

Predisposing non-alcohol specific risk pathway II

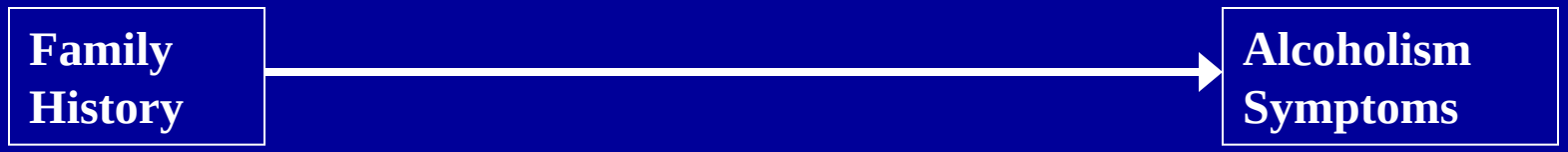


Content Characteristics of the model: B: Alcohol specific risk elements: I

- **Positive family history of alcoholism**

An index of familiality/heritability of AUD

Predisposing alcohol-specific risk pathway I



Content Characteristics of the model: B: Alcohol specific risk elements: II

- **Precocious alcohol involvement (early first drinking experience)**

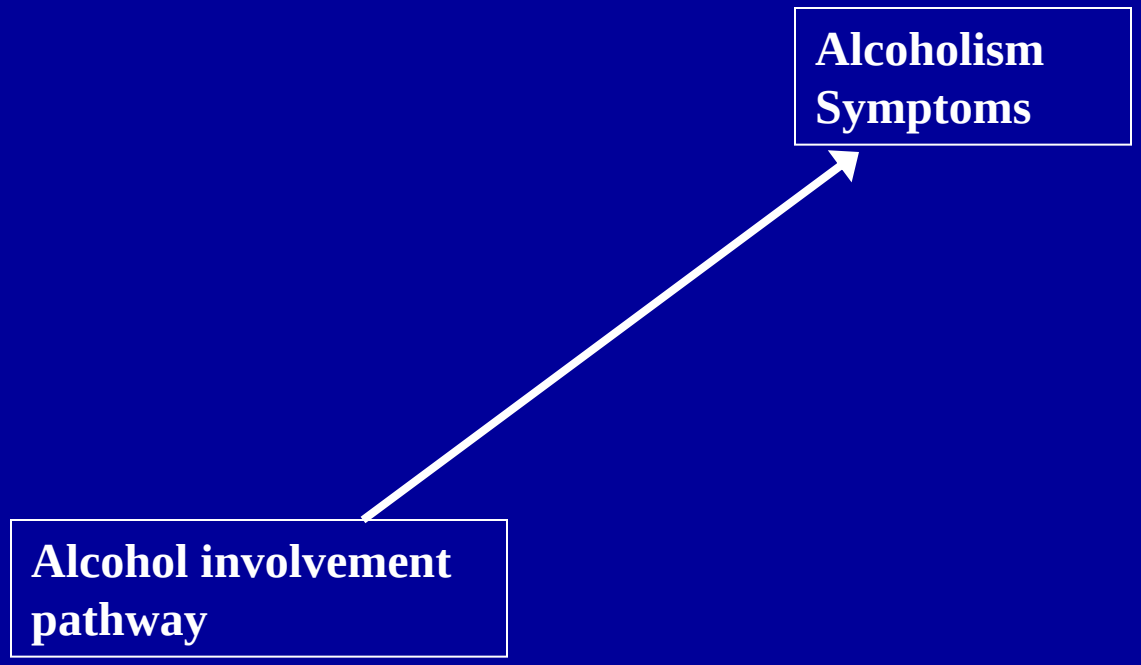
Early onset of drinking is a proxy for adolescent problem drinking: More injuries, violence, drunk driving during later adolescence (Gruber et al., 1996), lifetime risk of injury is greater (Hingson et al. 2000), and probability of AUD is 4X greater (Grant & Dawson, 1997).

Content Characteristics of the model: B: Alcohol specific risk elements: III

- **Early problem use (early drunkenness)**

  An early direct indicator of abusive drinking

Predisposing alcohol-specific risk pathway II

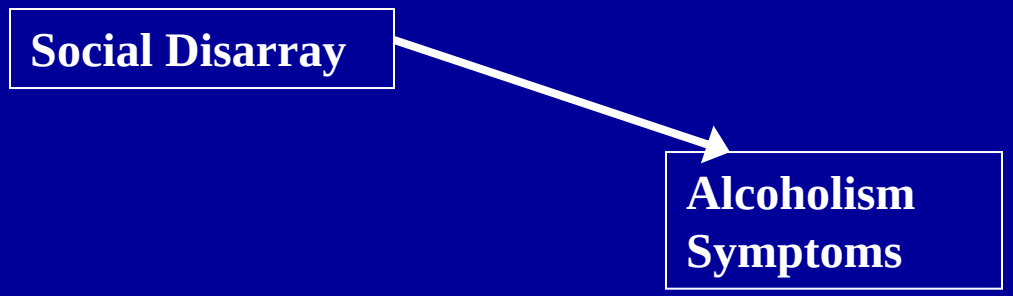


Content Characteristics of the model: C: Contextual factors that enhance or detract from alcohol involvement

Nonspecific enhancers:

- Stress
- Family conflict (may also be a content specific enhancer of undercontrol)

Contextual enhancement or dampening



Content Characteristics of the model: D: Epigenetic changes in the core phenotype over the course of development

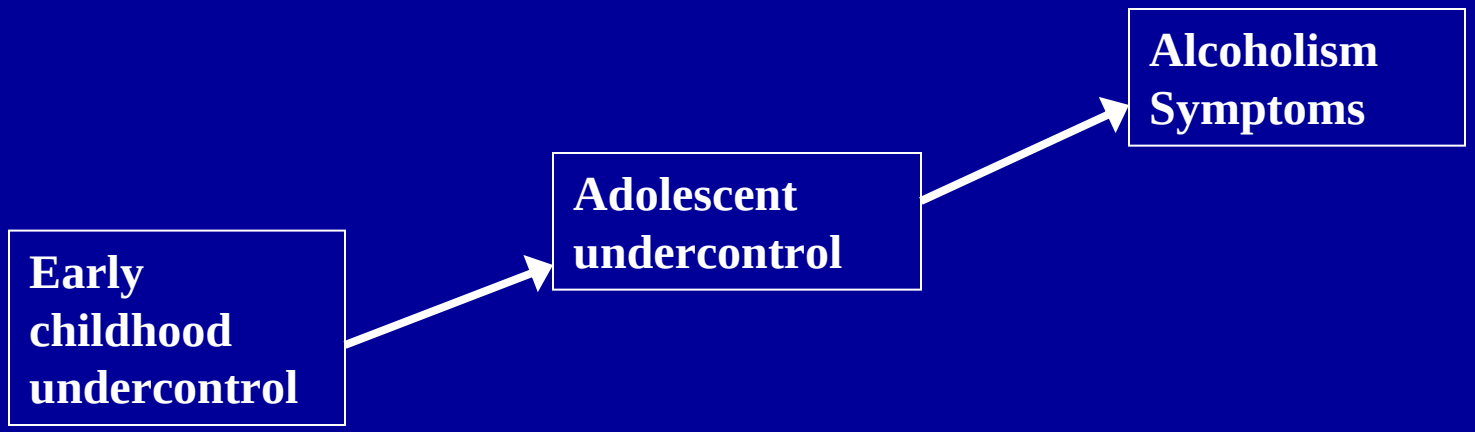
Specialization/differentiation of the undercontrol phenotype into antisocial symptomatology?

Specialization/differentiation of the negative affectivity phenotype into a) depressive symptomatology?

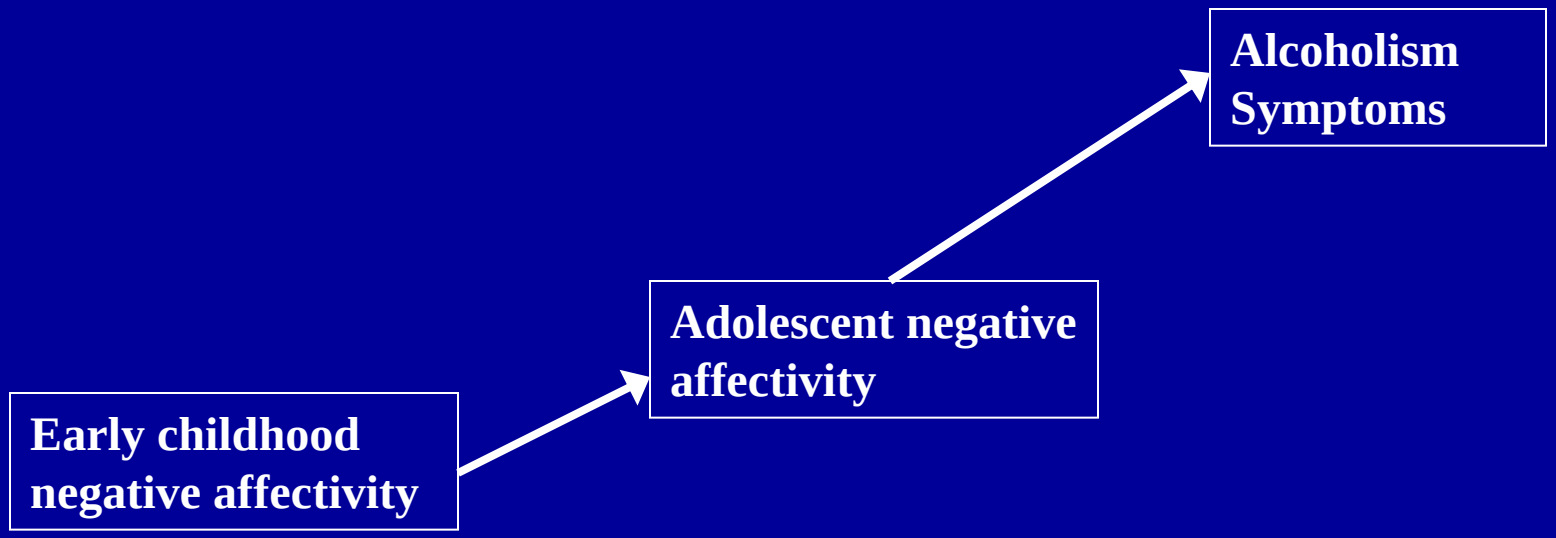
b) anxiety symptomatology?

c) social withdrawal?

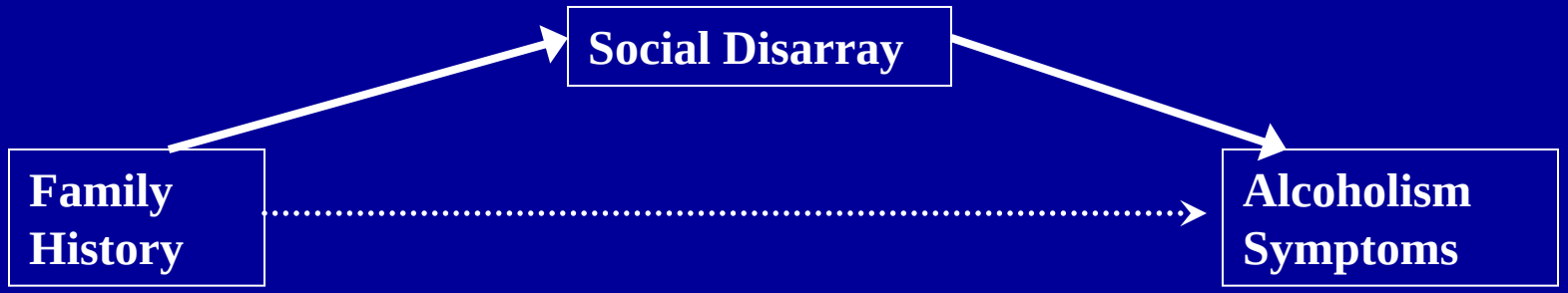
Epigenetic change I?



Epigenetic change II?



Contextual mediation of family risk



The composite phenotype over time....

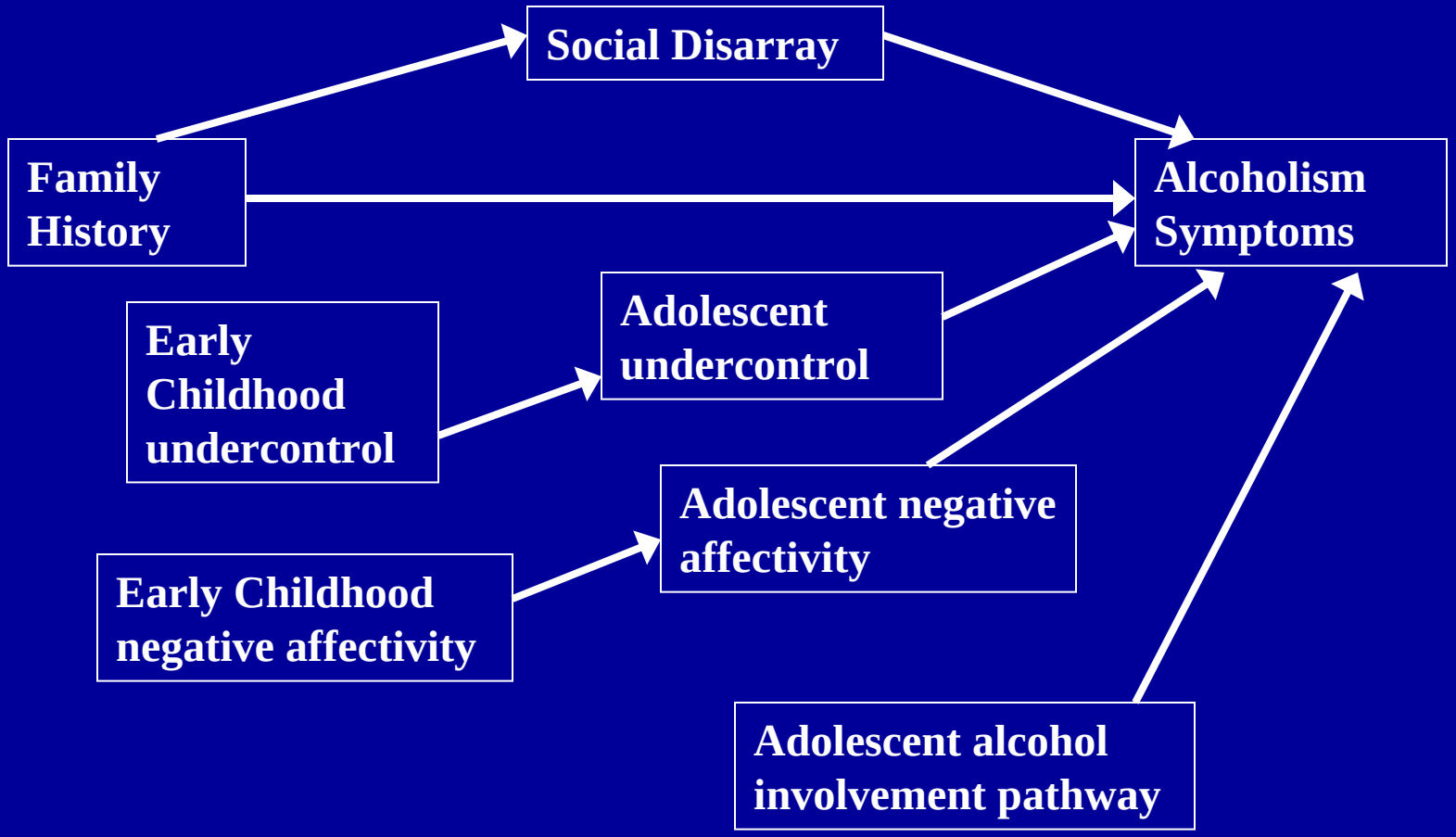
a) involving multiple pathways, both alcohol specific and nonalcohol-specific

b) epigenetic

c) operating in the presence of a facilitating or risk dampening environment

d) developmental aggregation of risk leading to the multicomponential phenotypic endpoint

Developmental aggregation of the endpoint phenotype



Venue: The Michigan Longitudinal Study*

•Alcoholic families recruited via court records and community canvassing; inclusion based on presence of father's alcoholism.

•Ecologically comparable control families resided in and were recruited from same neighborhoods.

•Mother's alcoholism and other psychiatric status free to vary in alcoholic families.

•Family participants: Biological father, mother and initially 3-5 year old son (initial target child (TC)) and all full siblings within +/- 8 years of TC, step-parents, and now third generation Ss ((N~2,100 individuals)

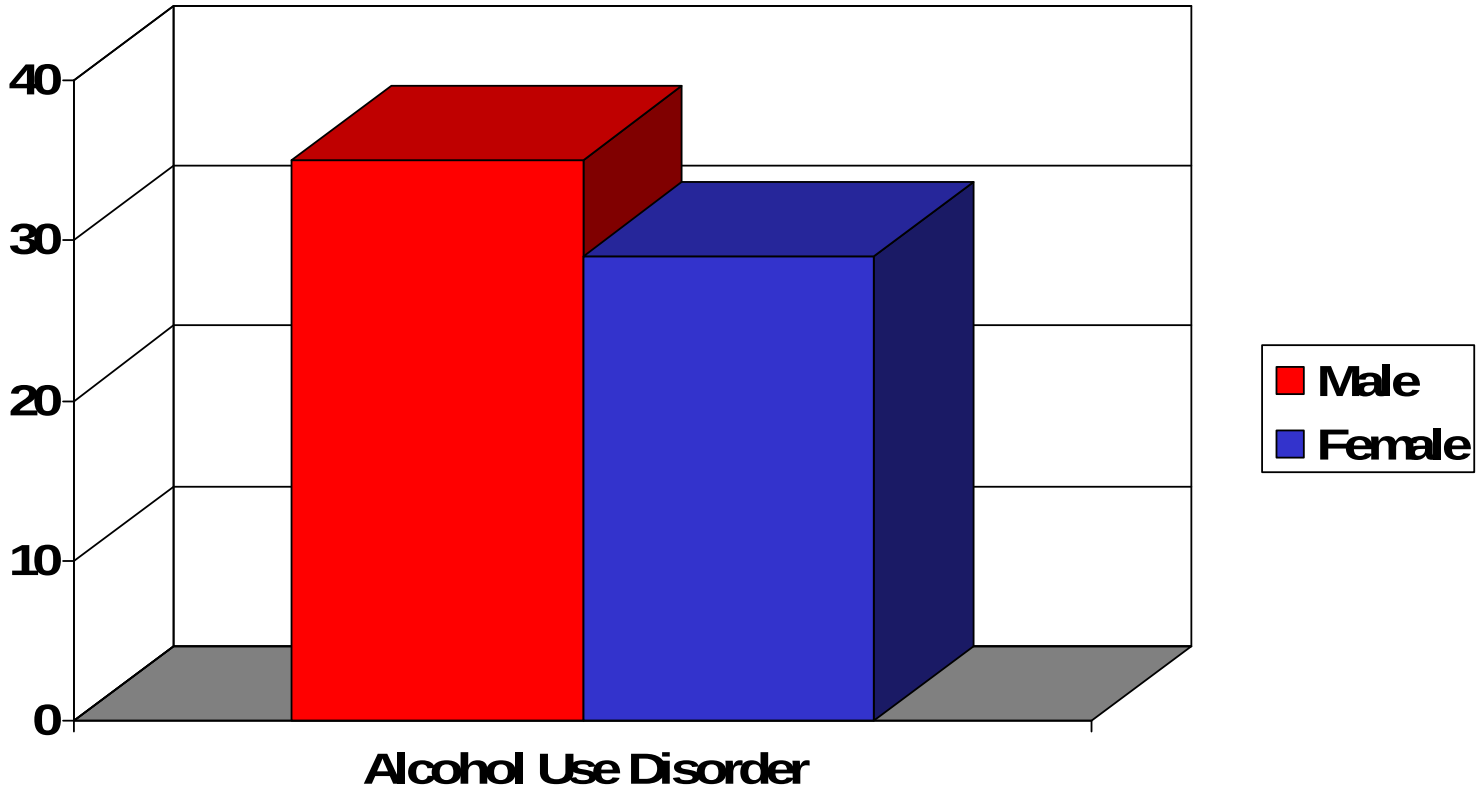
A joint venture of the University of Michigan and Michigan State University

Method

- Assessment at 3 year intervals; currently in the 15 and 18 year follow-ups [Wave 6 = ages 18-20; Wave 7 = ages 21-23 for core group of probands].
- Tracking markers of risk and psychosocial outcomes from early childhood into adulthood for children
- Tracking clinical course and correlates of symptomatology for adult participants
- Biological sample collection , early genotyping , and neuroimaging in progress.
- Current analyses based on 339 probands who entered the study at Wave 1 (age 3-5) and have passed through either Wave 6 (age 18-20) or Wave 7 (age 21-23) of the protocol.

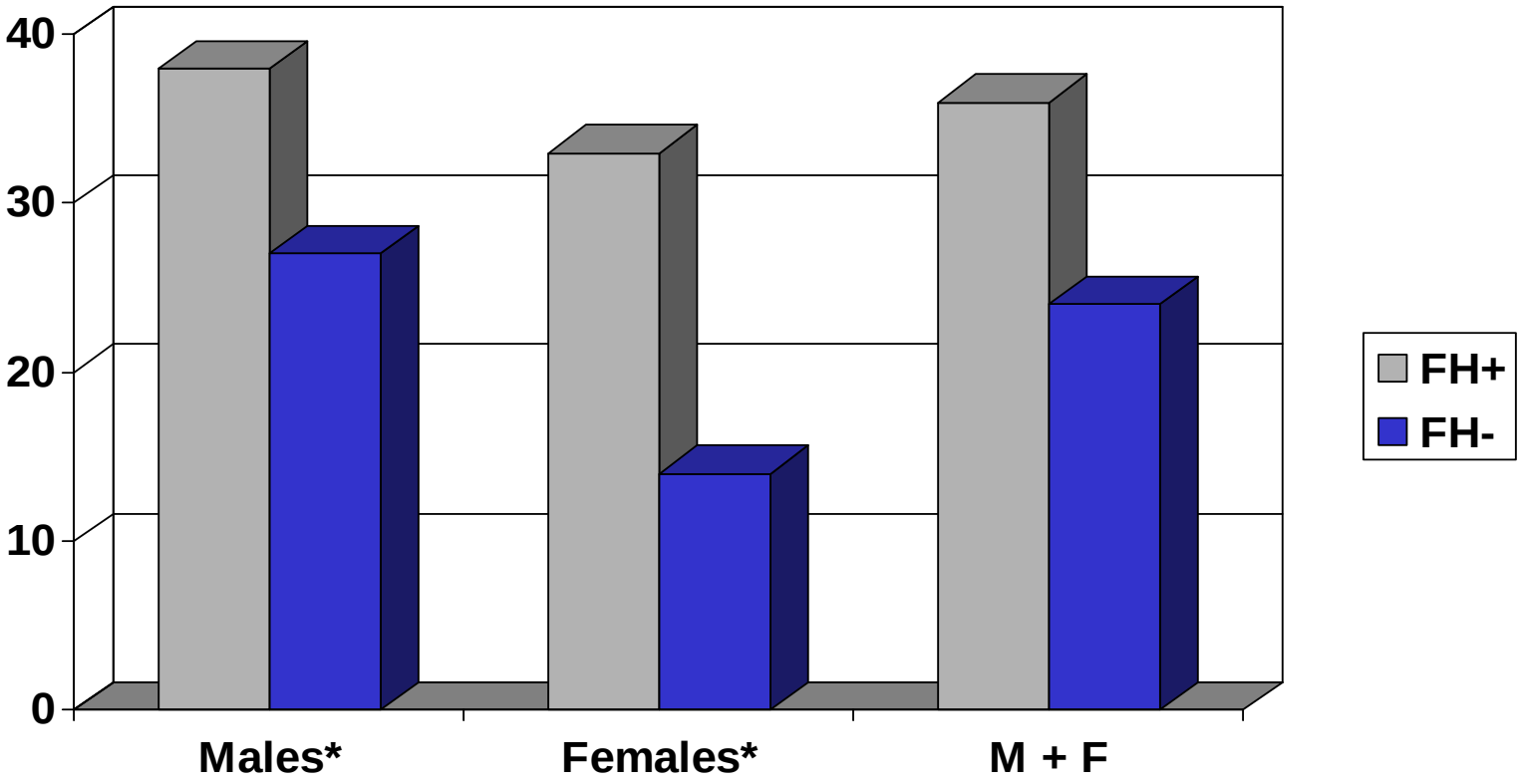
Diagnostic endpoint in early adulthood (ages 18-23)

Lifetime Alcohol Use Disorder (%) in MLS probands at age 18-23



Sex difference is ns.

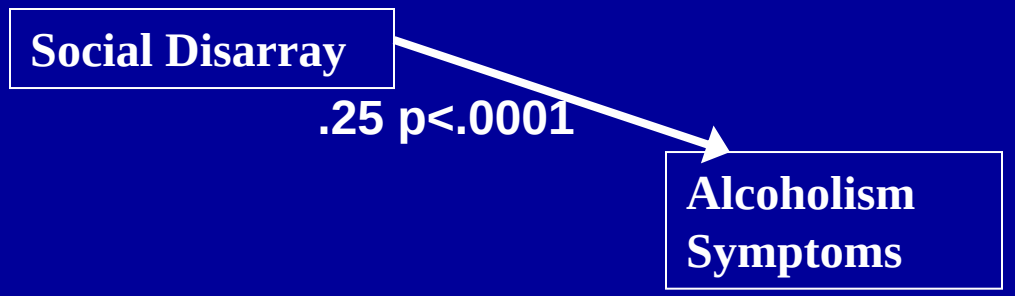
Lifetime Alcohol Use Disorder in MLS probands at Age 18-23 by Sex and Family History (%)



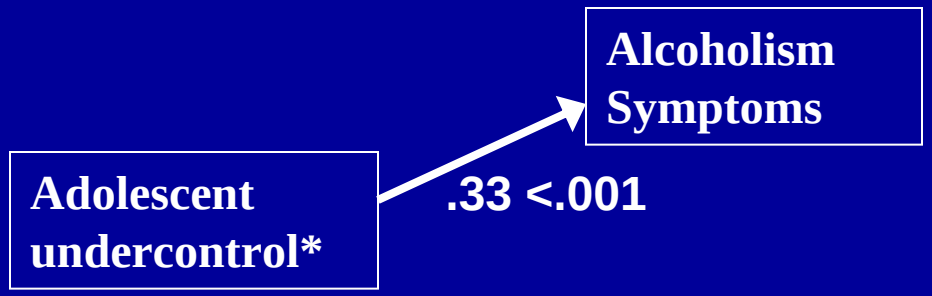
Within sex relationship of FH to dx is not significant;
for total sample, *p* = .05

Predisposing alcohol-specific risk pathway I

Social context effects on risk transmission

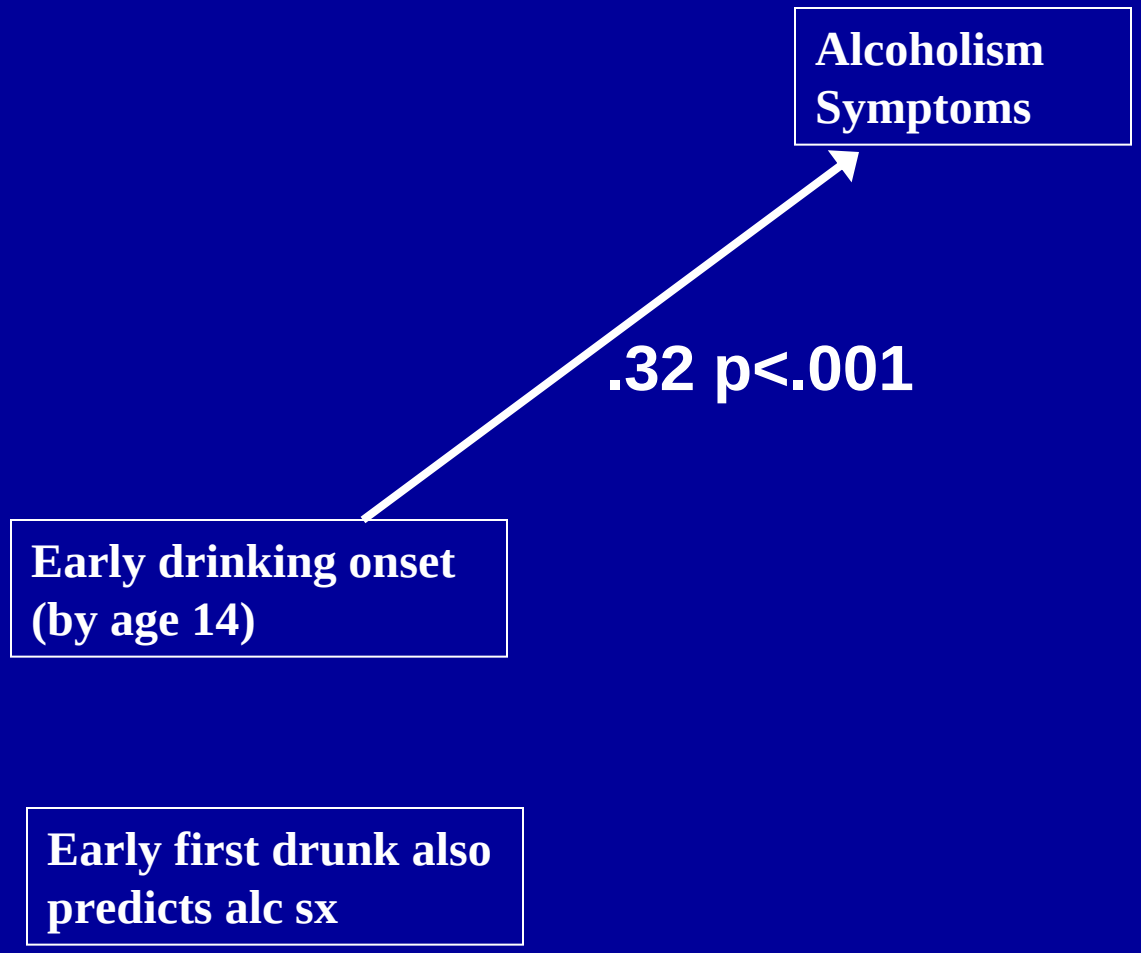


Adolescent undercontrol predicts the endpoint phenotype



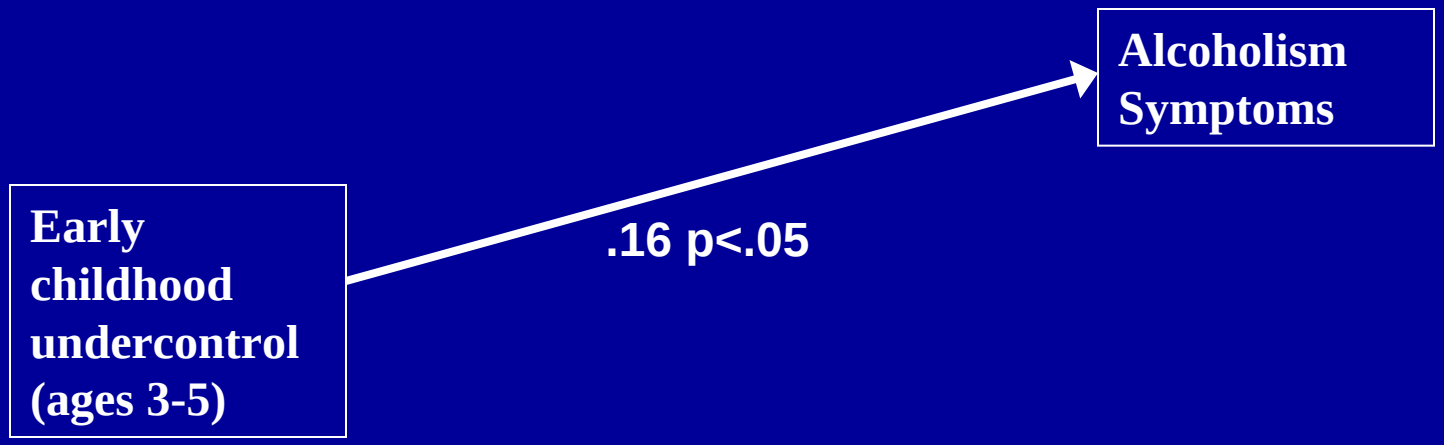
*** A parallel relationship holds for adolescent antisocial behavior, although slightly weaker**

Predisposing alcohol-specific risk pathway II

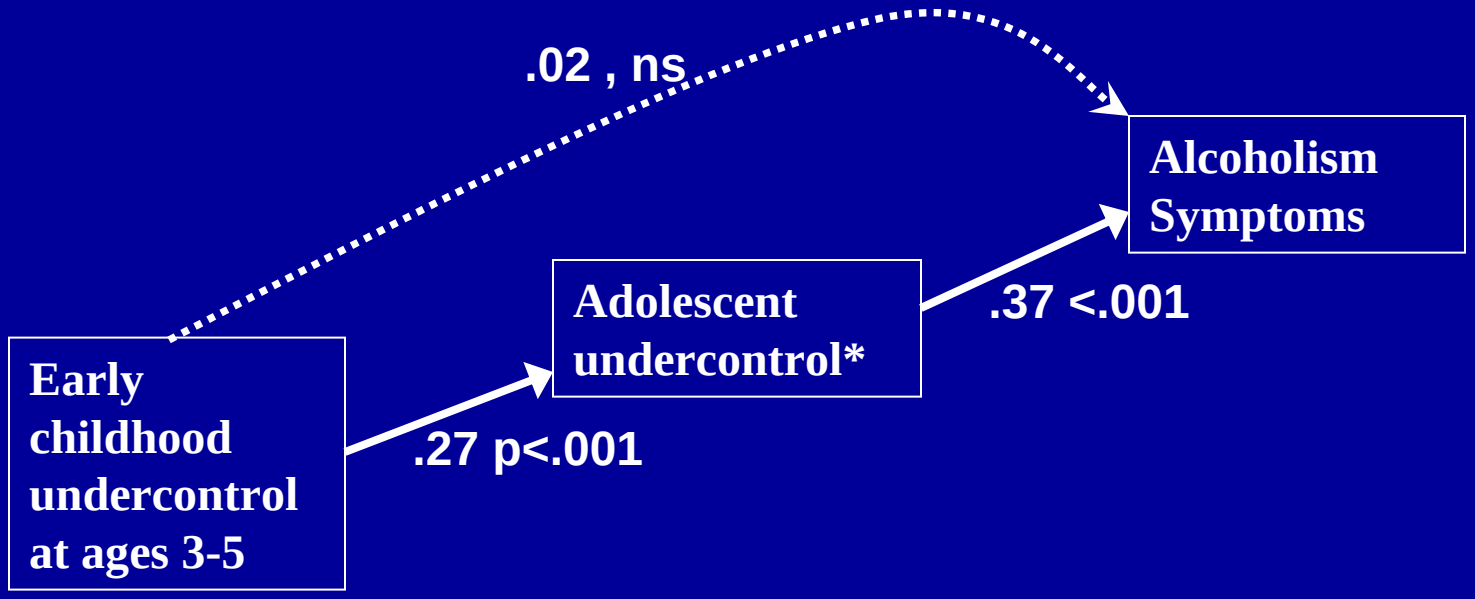


Looking upstream from adolescence…

Predisposing nonspecific risk pathways identifiable in early childhood I; Undercontrol



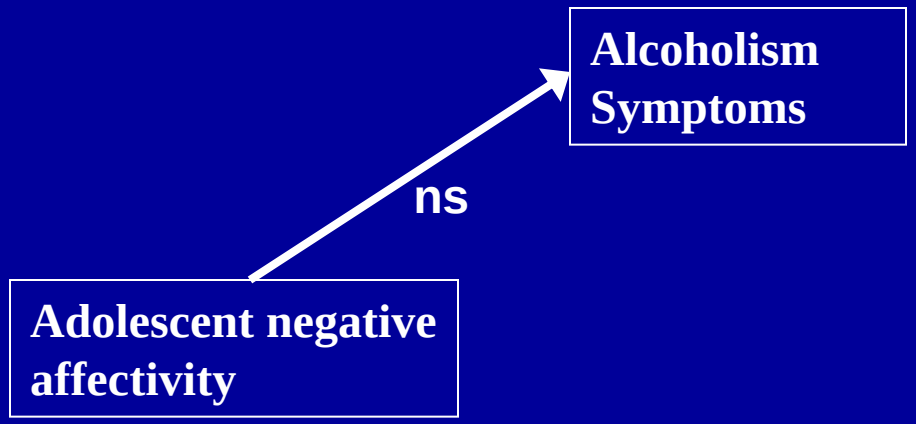
Epigenetic developmental continuity in undercontrol



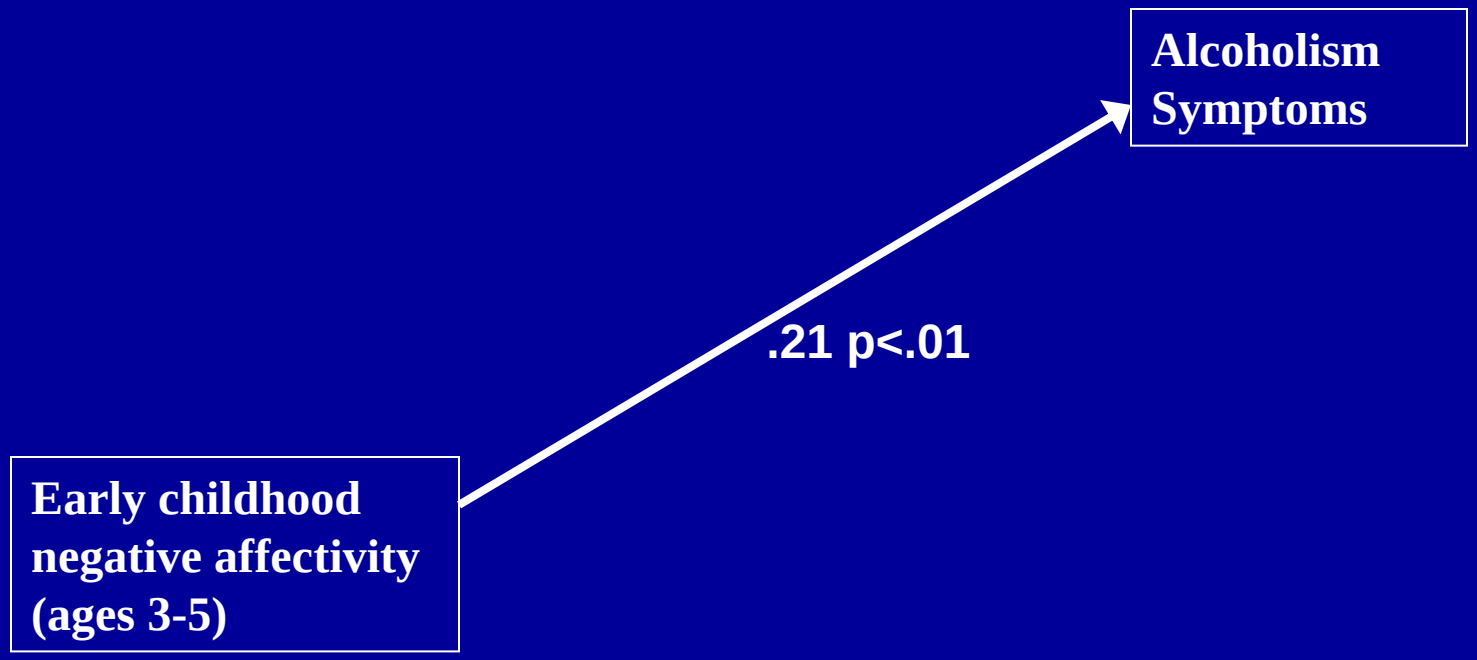
*** Mediated relationship also holds for adolescent antisocial behavior, although slightly weaker**

Mediated relationship is significant (Sobel test)

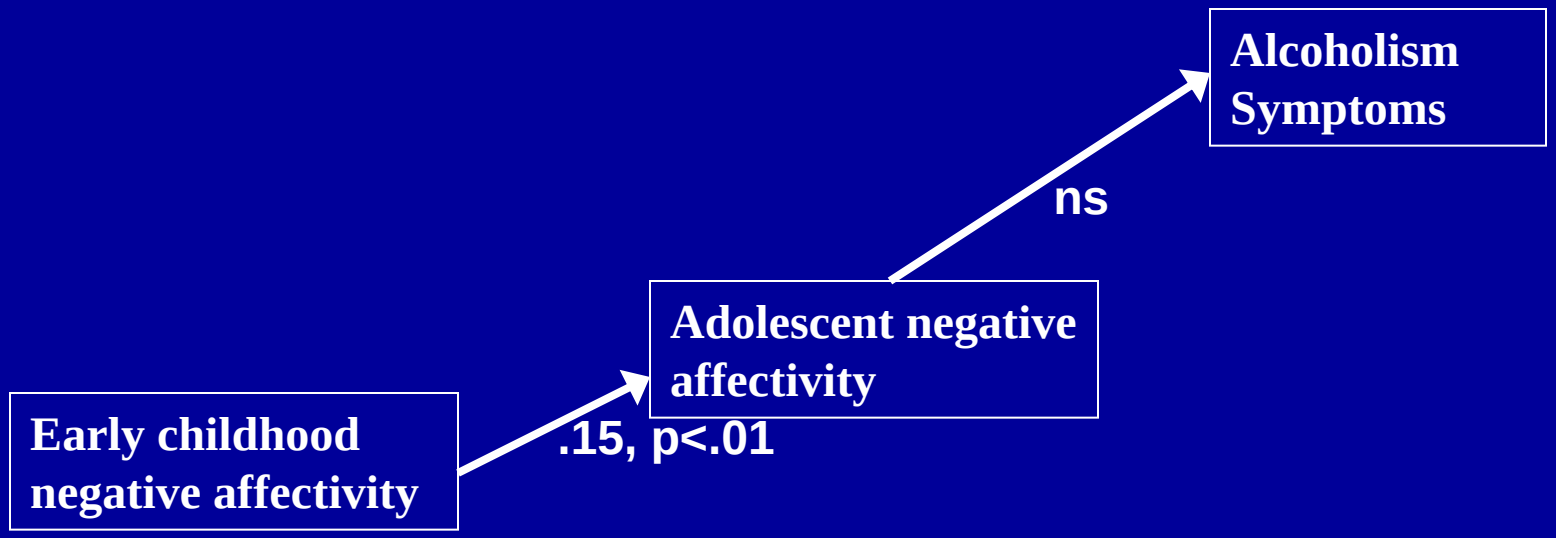
Adolescent negative affectivity does not predict the endpoint phenotype



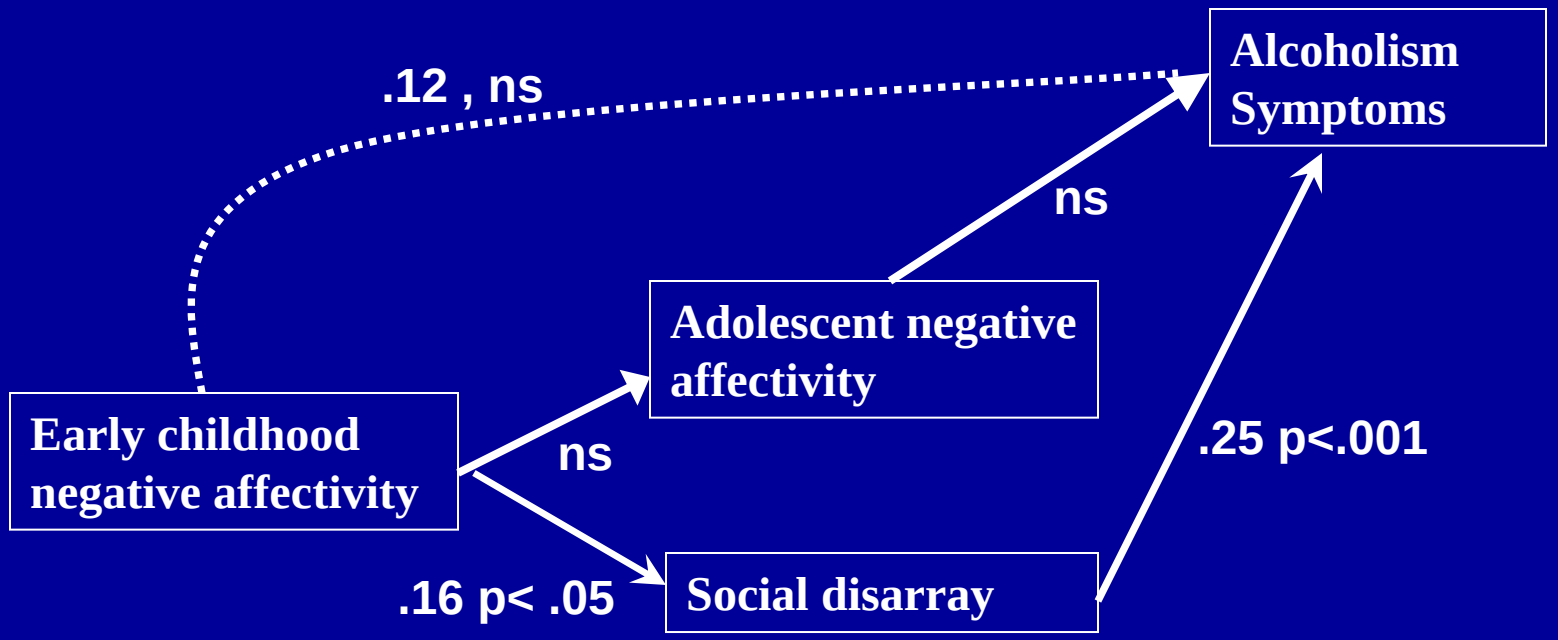
Predisposing nonspecific risk pathways identifiable in early childhood II: Negative affectivity



No epigenetic developmental continuity for negative affectivity

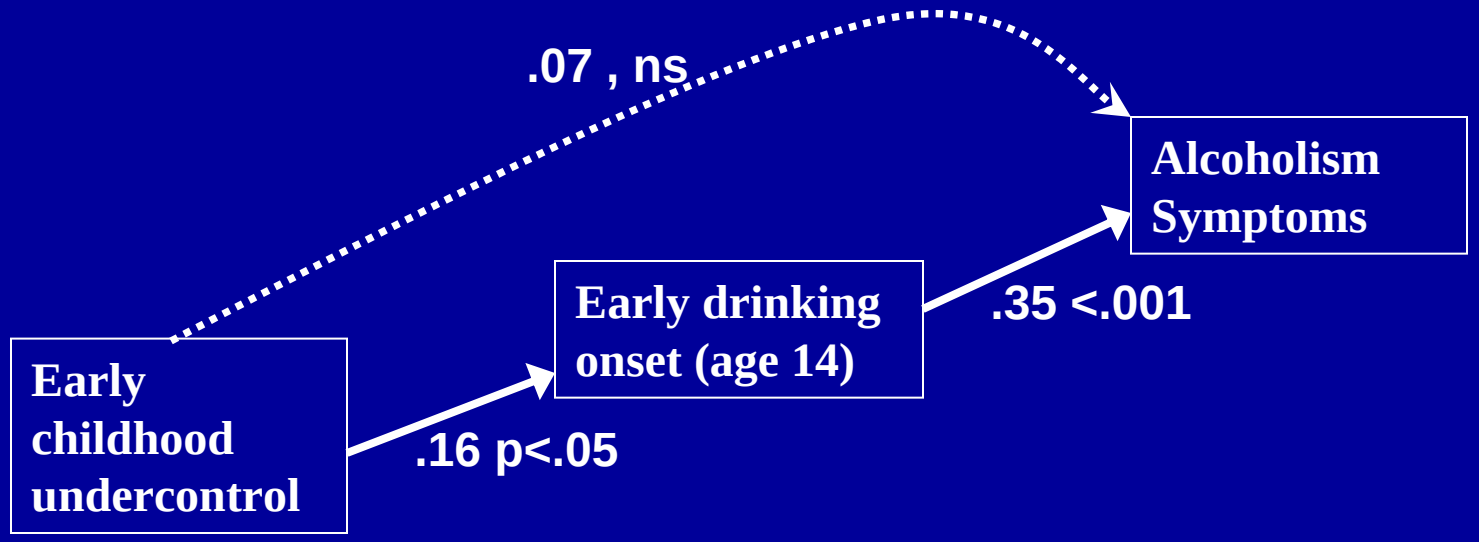


Early negative affectivity effects mediated through social disarray/stress

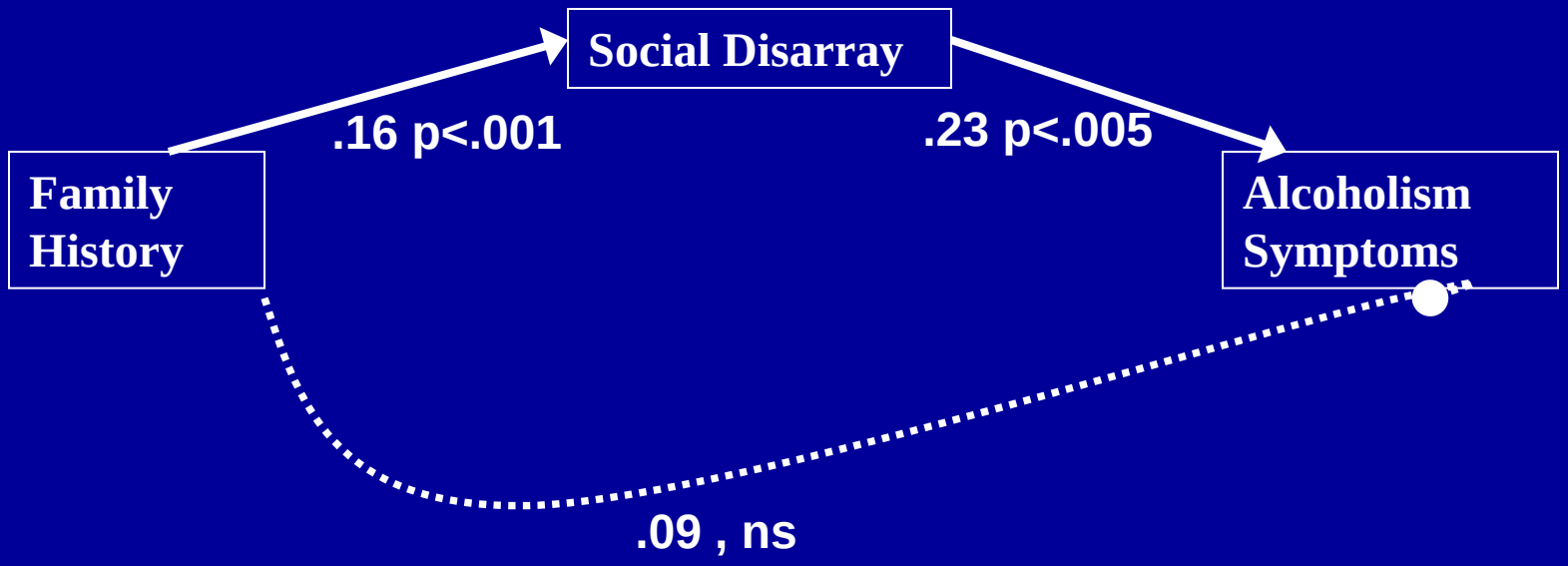


Mediated relationship is significant (Sobel test)

Undercontrol risk transmission is also mediated by early alcohol involvement

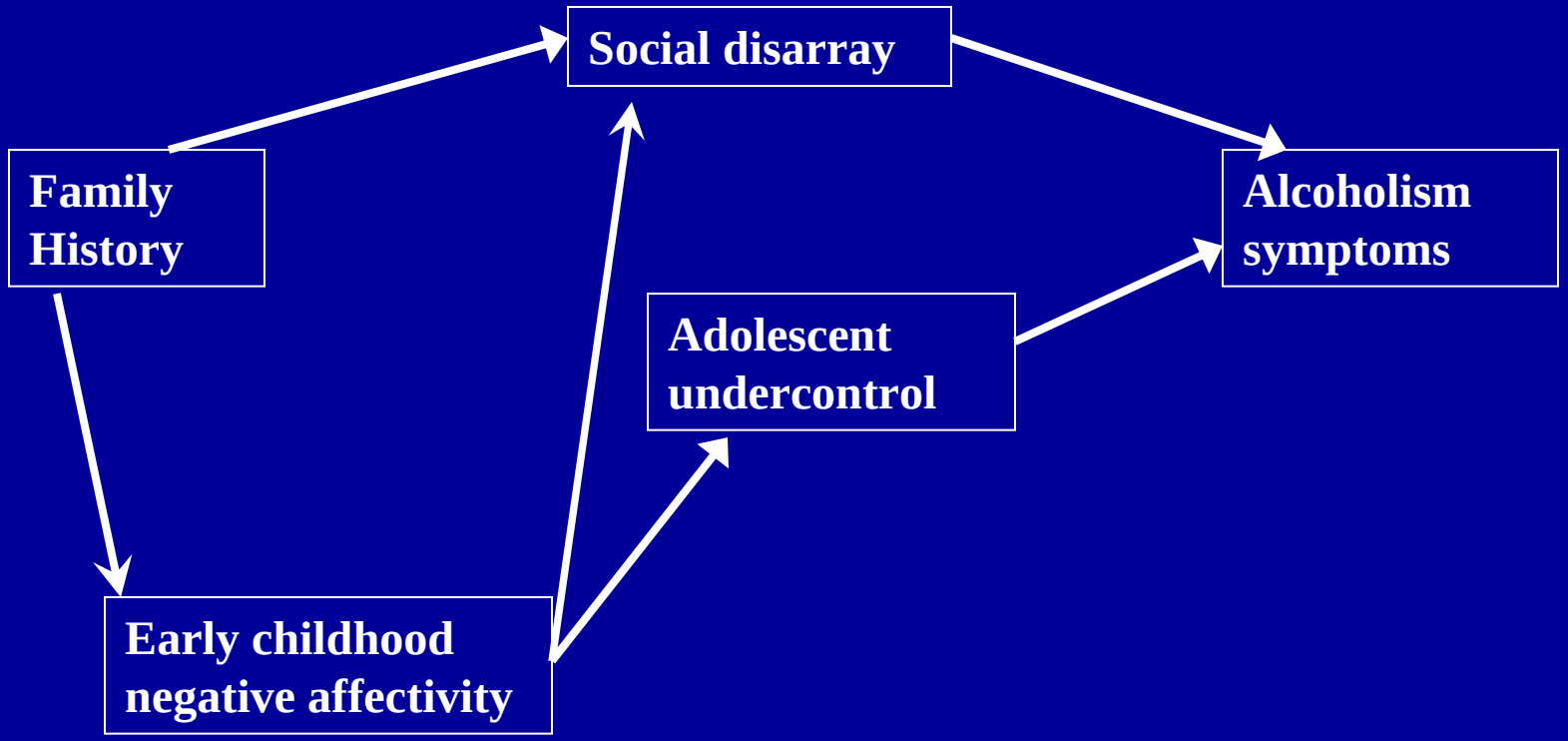


Contextual mediation of family risk

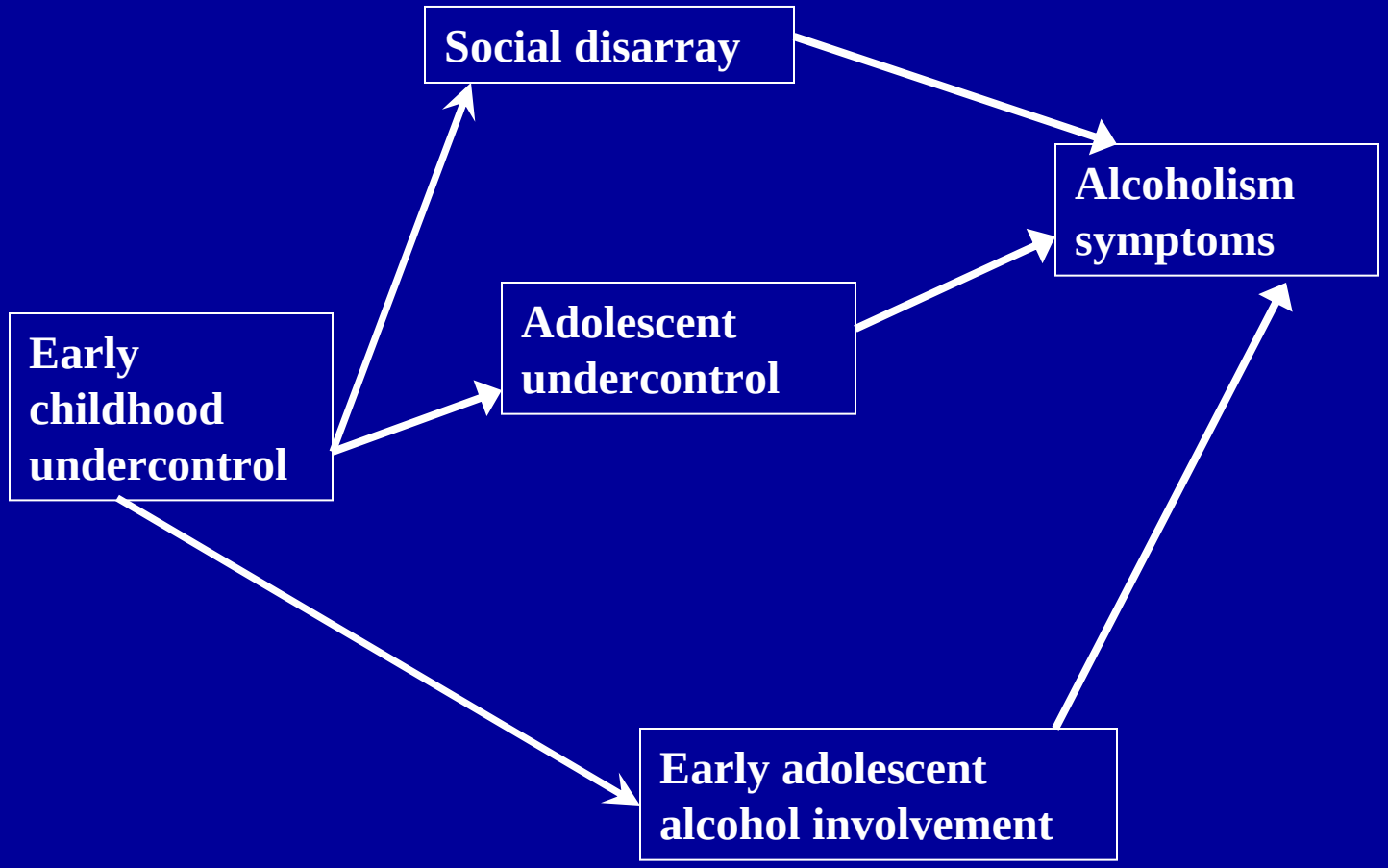


The multiple pathways of risk development that lead to problem drinking and alcohol use disorder

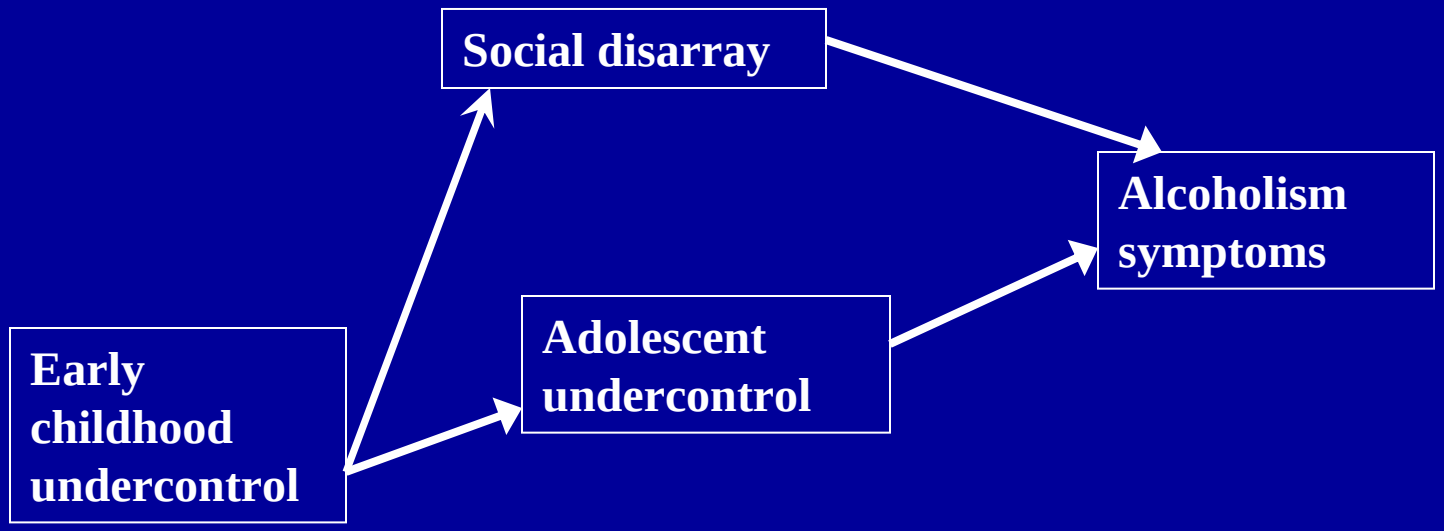
The family history of alcoholism pathway



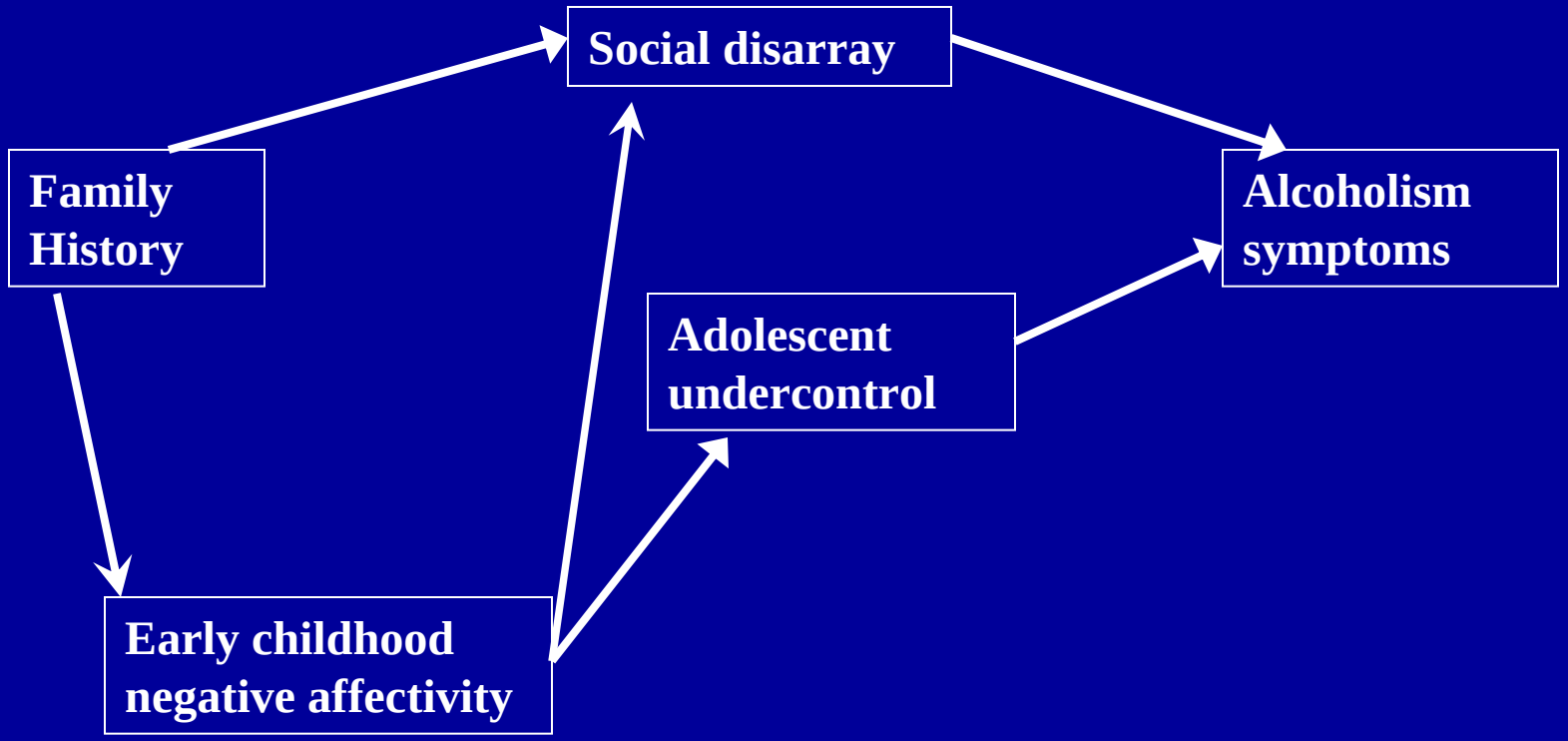
The early alcohol involvement pathway



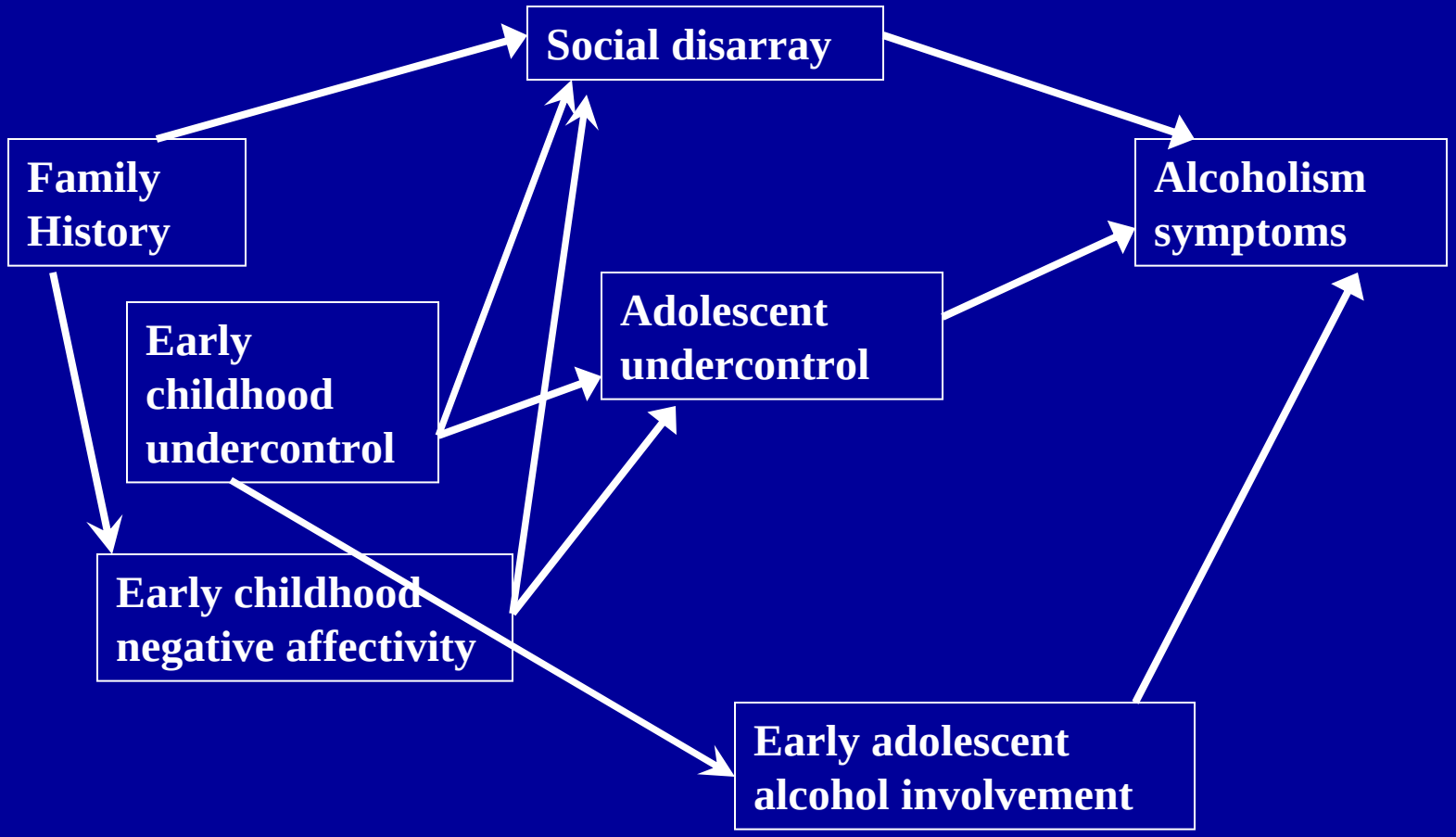
The undercontrol pathway



The negative affectivity pathway



Developmental mediation and domain aggregation in the final common pathways to the endpoint phenotype

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EXIT ONLY

Developmental models of drinking:

Where should they begin?

Where do they end?

What should be done about the evidence we now have?

Social Policy Considerations

Although AUD has been recognized as a chronic, recurring disorder, the ability to identify risk long before clinical onset has generally been regarded as not possible. Our findings--in concert with at least 6 other longitudinal studies worldwide -now indicate this is feasible.

Why then is it not a part of the dominant paradigm of the field? Recent reviews indicate that some intervention techniques are effective in reducing risk long term. Other challenges remain however.

Massive cost considerations.

Identification of venues for early identification without increasing stigma.

The need to identify feasible pass-through points for intervention.