Form follows (dys)function: An evolutionary model of the structure of psychopathology

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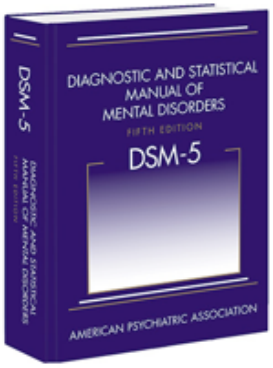
The story so far…



DSM taxonomy:

- **"atheoretical:"** unsatisfactory in the long run
- many diagnostic categories are **highly heterogeneous**
- lacks account of **large-scale comorbidity patterns**
- …

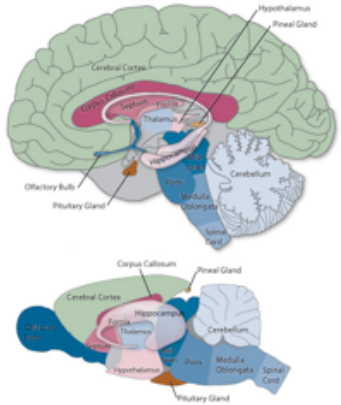
Current answer 1: research domain criteria (RDoC)

NIH National Institute of Mental Health

RDoC Research Domain Criteria Initiative

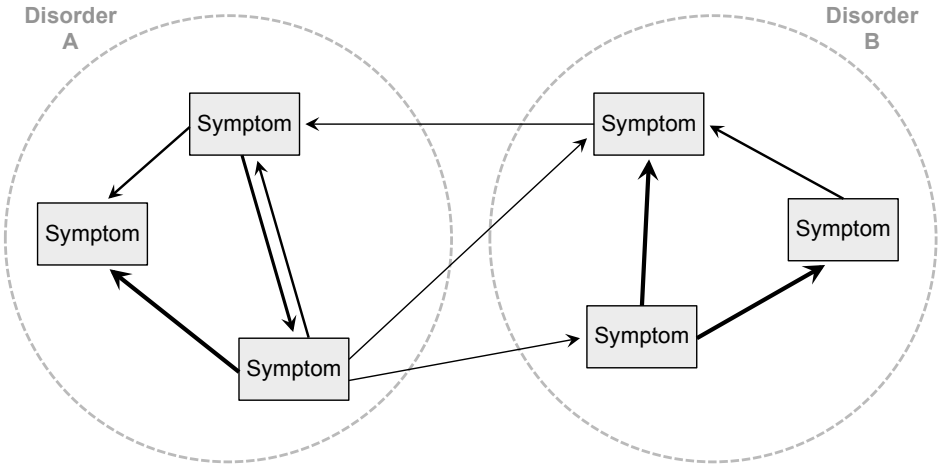
Negative Valence Systems

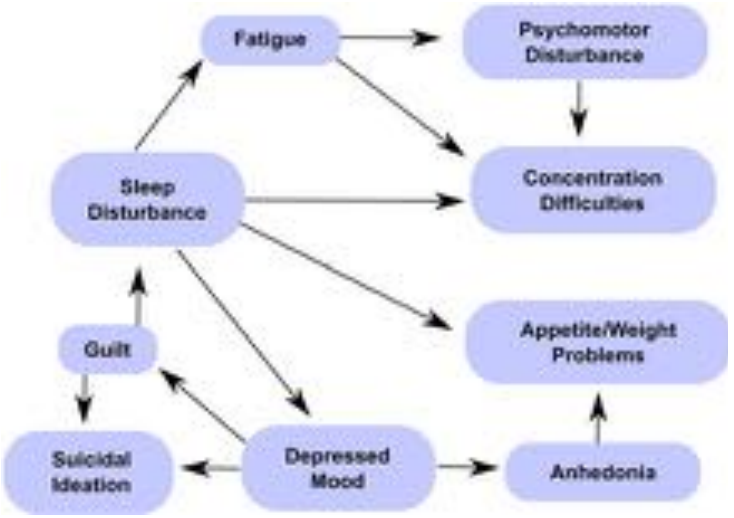
Construct/Subconstruct	Genes Notice	Molecules	Cells	Circuits	Physiology	Behavior	Self-Report
Acute Threat ("Fear")		Elements	Elements	Elements	Elements	Elements	Elements
Potential Threat ("Anxiety")		Elements	Elements	Elements	Elements		Elements
Sustained Threat		Elements	Elements	Elements	Elements	Elements	Elements
Loss		Elements		Elements	Elements	Elements	Elements
Frustrative Nonreward		Elements		Elements		Elements	Elements



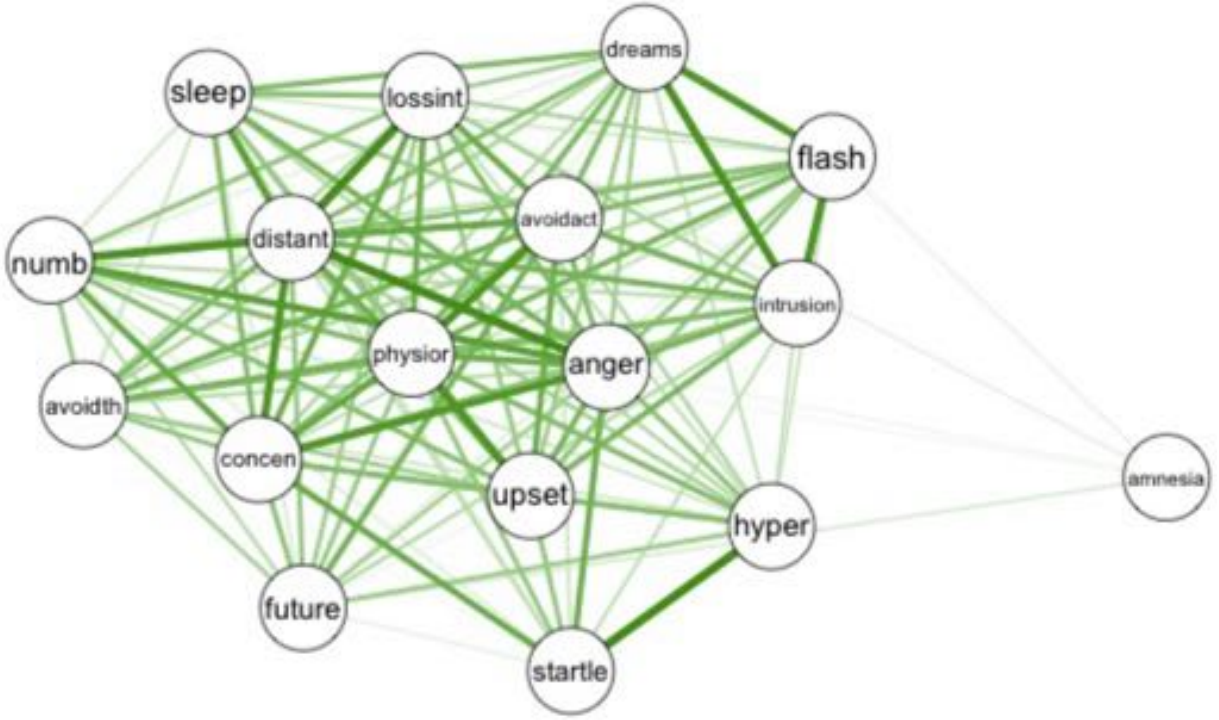
- **bottom-up** approach, focus on brain circuits
- extremely **patchy coverage** (e.g., no mating/sexual behavior)
- still no account of large-scale comorbidity

Current answer 2: Network models

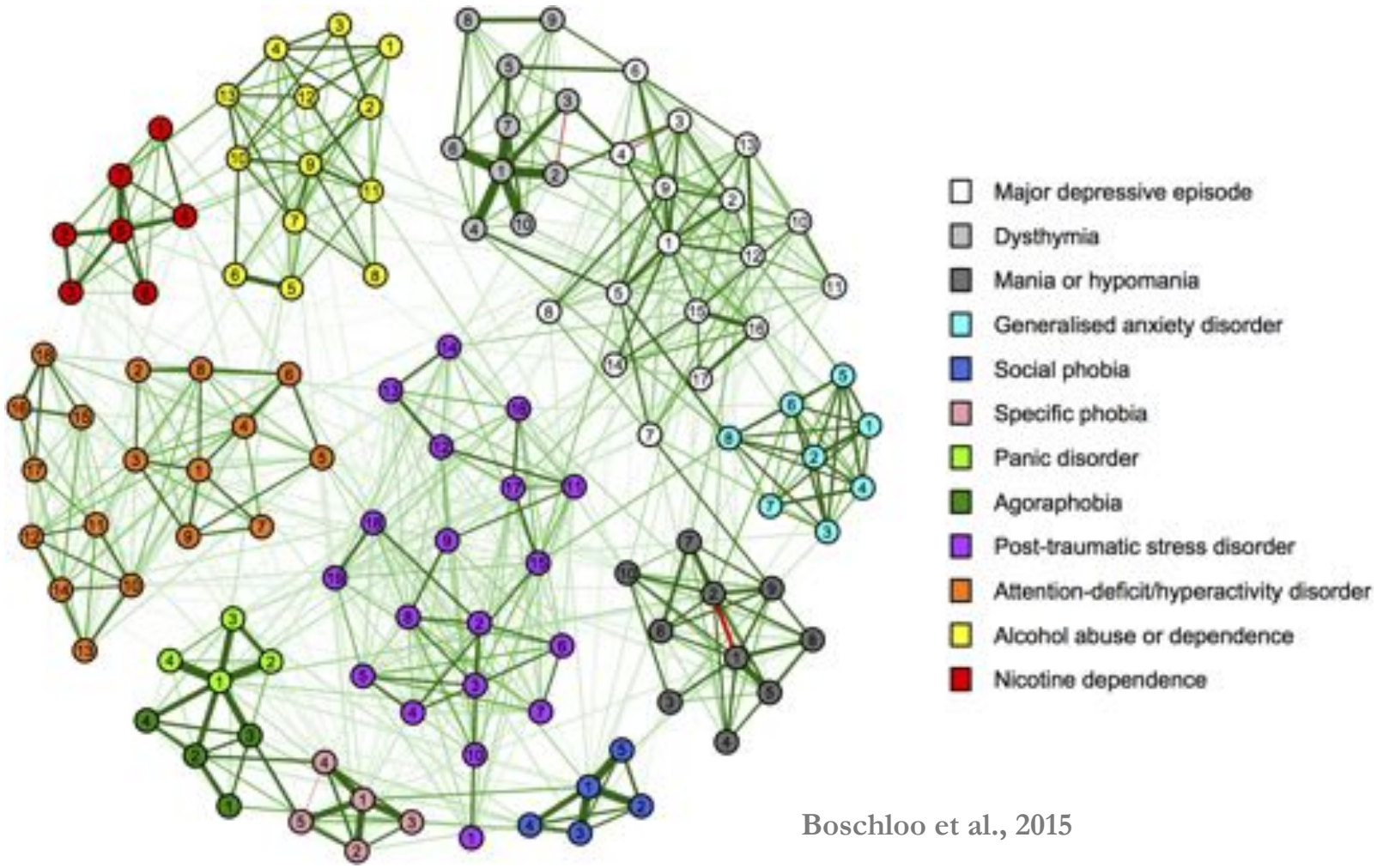




Guloksuz et al., 2017



McNally, 2016

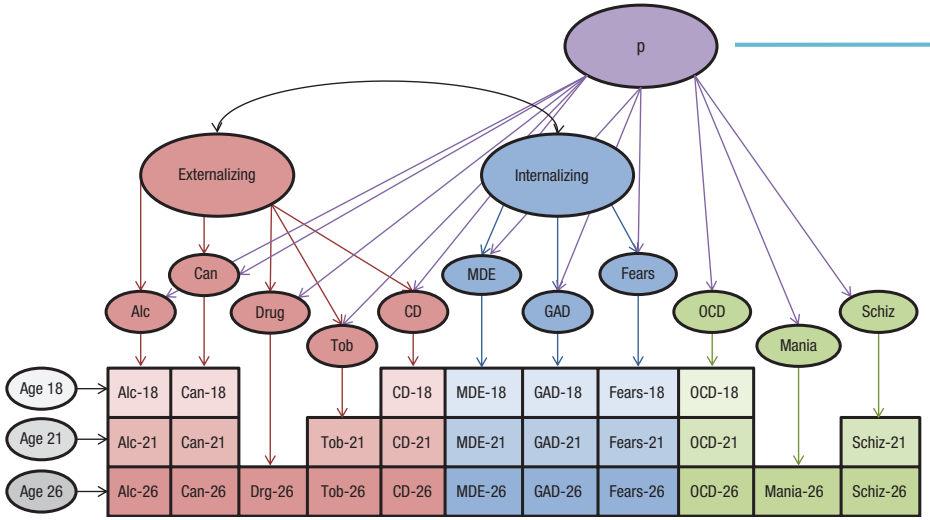


Boschloo et al., 2015

- **methodological difficulties** (replication, stability, strong assumptions…)
- **weak rationale** for "pure" symptom networks (compare with physical disorders)
- large-scale results: not too different from DSM structure…

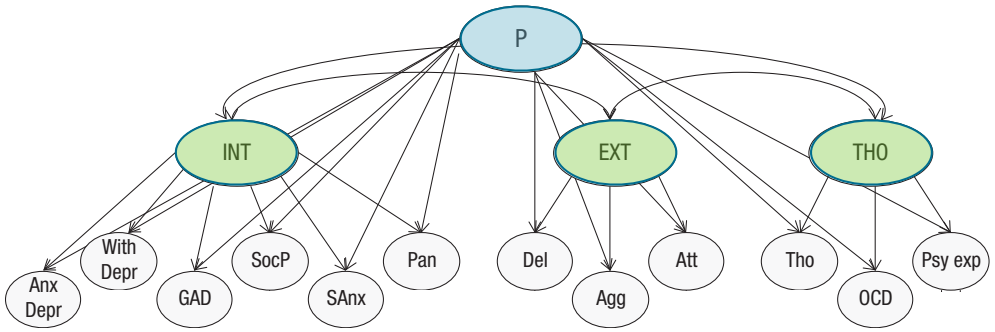
Current answer 3: Transdiagnostic models

Caspi et al. (2014): a **"p factor"** for psychopathology

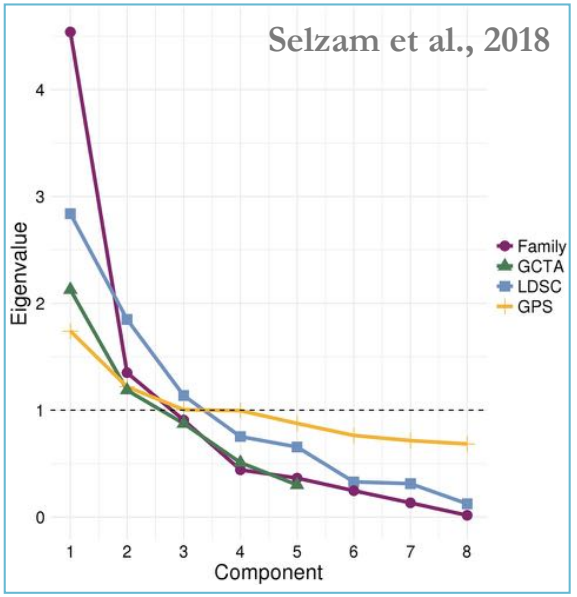


Caspi et al., 2014

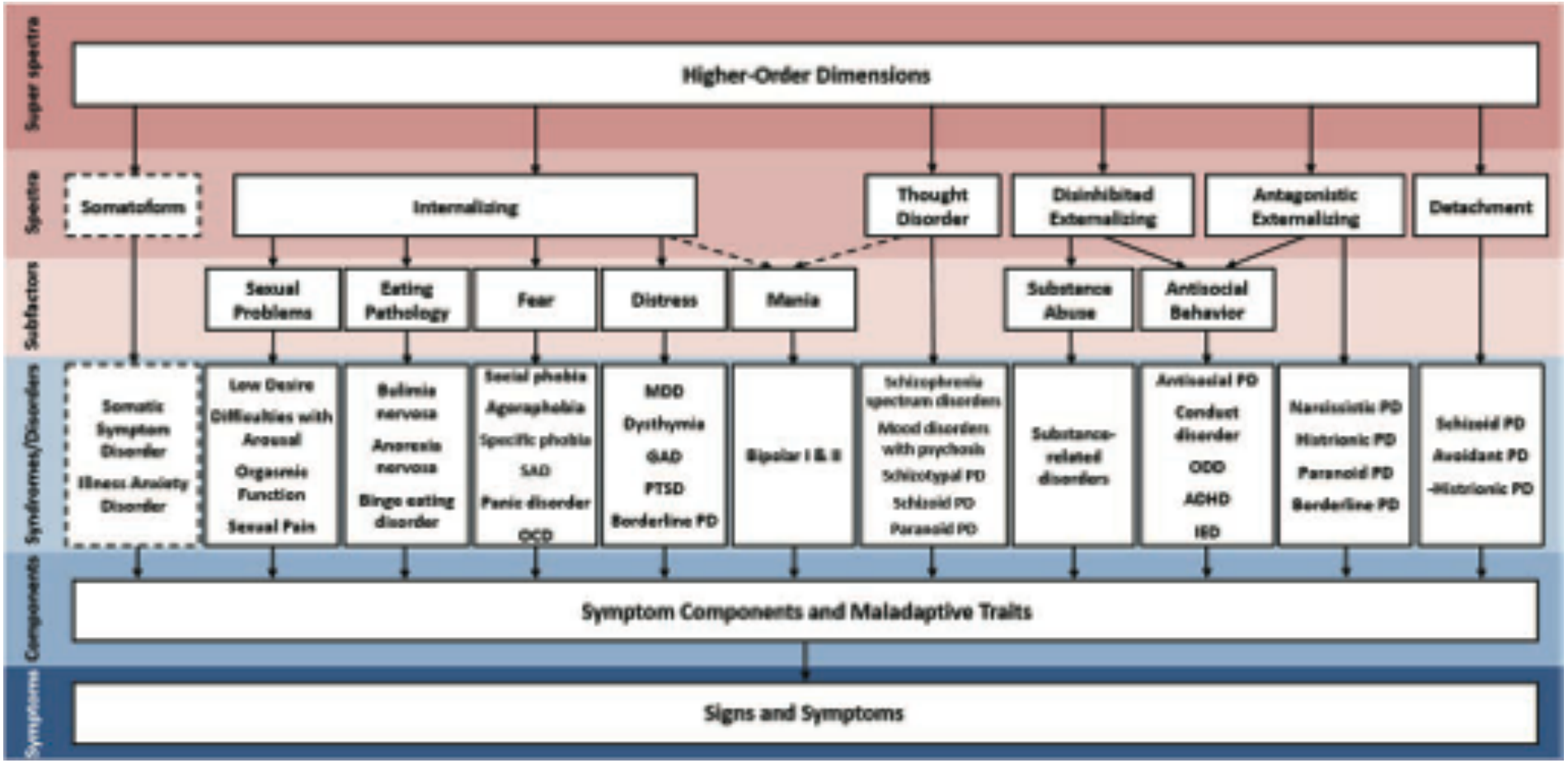
low IQ, neural integrity
low agreeableness
low conscientiousness
high neuroticism
childhood adversity
low SES



Laceulle et al., 2015



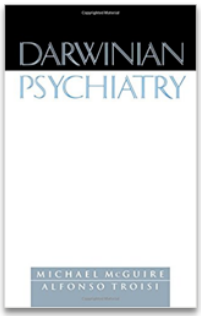
- about **40% heritable**
- found in phenotypic and genetic correlations

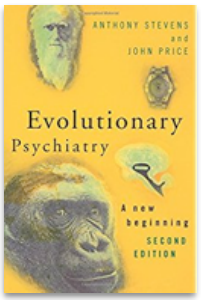


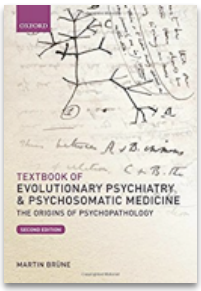
Kotov et al., 2017 (**HiTOP** model)

- powerful description of **large-scale structure**
- explicit links with **normal personality variation**
- **largely inductive:** symptom/syndrome correlations
- ignores **heterogeneity within disorders**

The view from evolution







Undesirable conditions
(broad-sense disorders)

Dysfunctional mechanisms

Harmful dysfunctions
(narrow-sense disorders)

Functional mechanisms

Currently maladaptive
(population level)

[*May also cause dysfunctions*]

Evolutionary mismatches

Currently adaptive
(population level)

Maladaptive outcomes
(individual level)

[*May also cause dysfunctions*]

Developmental mismatches

Maladaptive learning

Maladaptive outcomes of
high-risk strategies

Maladaptive outcomes of
evolutionary conflicts

Errors in defense activation
(false positives/negatives)

Other consequences of
design constraints and trade-offs

Adaptive outcomes
(individual level)

Antisocial, exploitative,
or socially devalued strategies

Aversive defenses

Self-sacrificial adaptations

Other consequences of
health-fitness trade-offs

Del Giudice (2018)
Del Giudice & Ellis (2016)

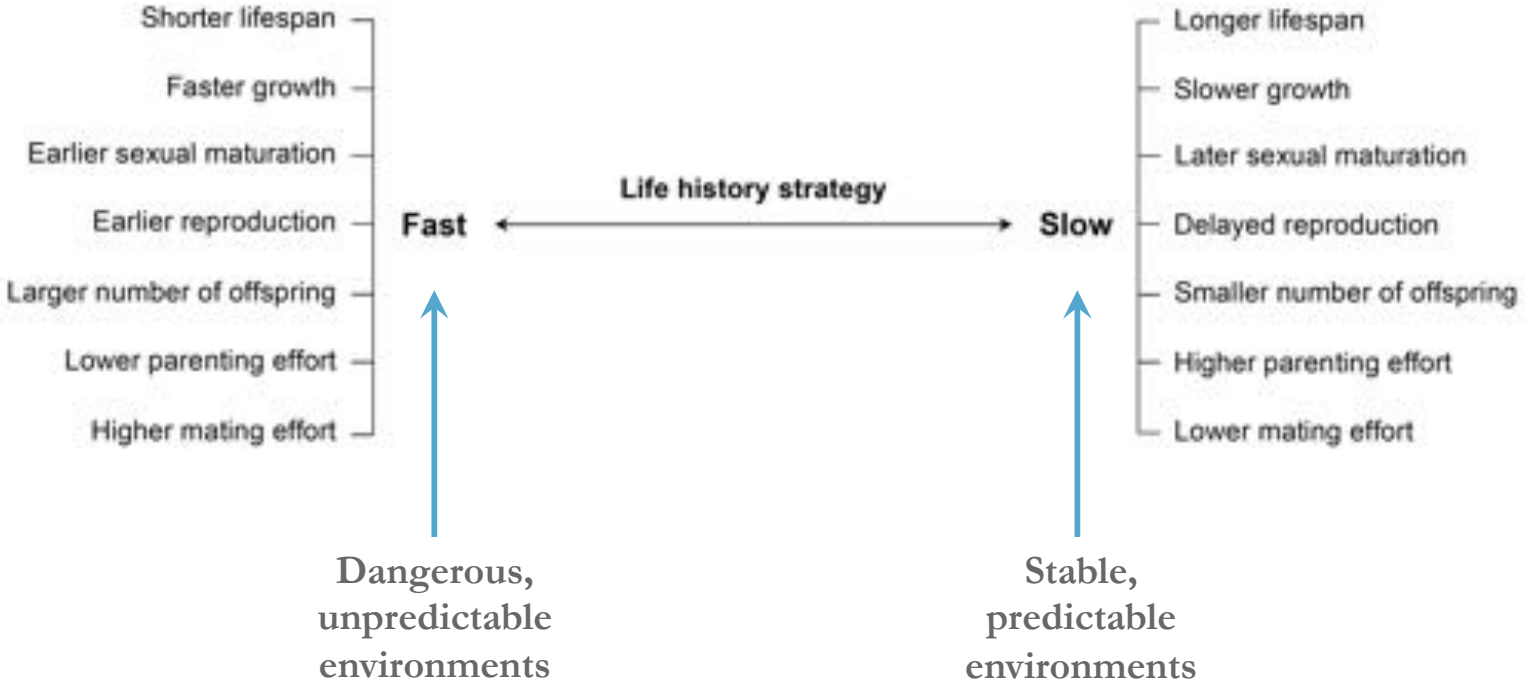
Many key insights but also limitations:

- **lack of integration** among models of specific symptoms/disorders
- few connections with **developmental psychopathology** and **psychiatric genetics**
- no models of **comorbidity, large-scale structure** of mental disorders

A life history perspective

The basic problem: **resource allocation (energy, time…)** among competing components of fitness

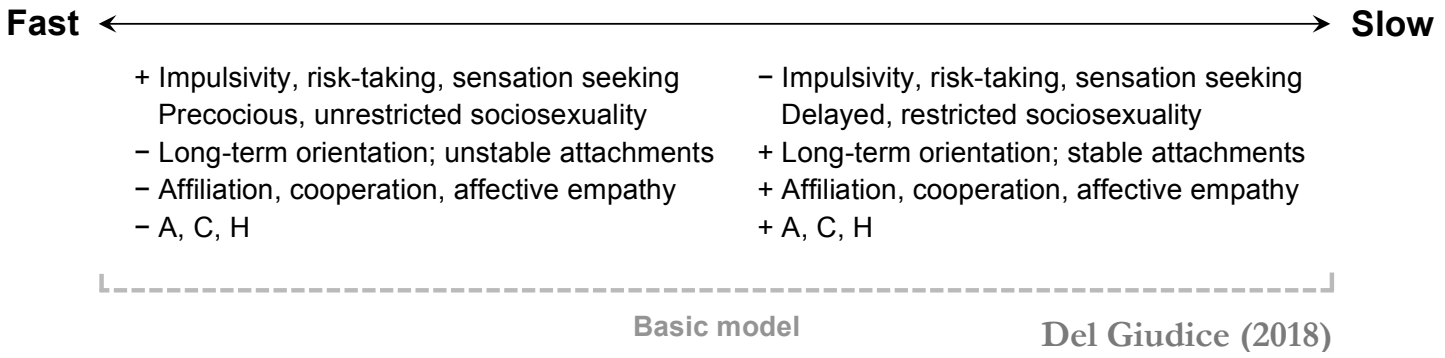
- **somatic effort** (growth, tissue repair, immunity…) vs. **reproductive effort** (mating, parenting)
- **mating** vs. **parenting** effort
- **current** vs. **future** reproduction
- **quantity** vs. **quality** of offspring (survival, growth, mating potential)…



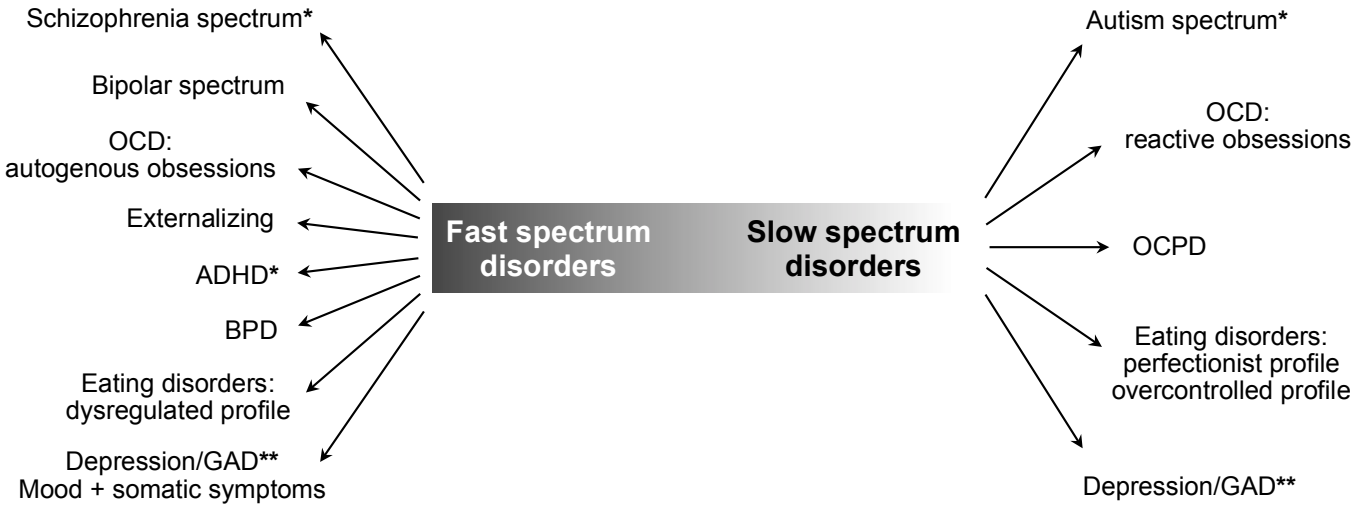
Life history trade-offs shape **development and behavior**

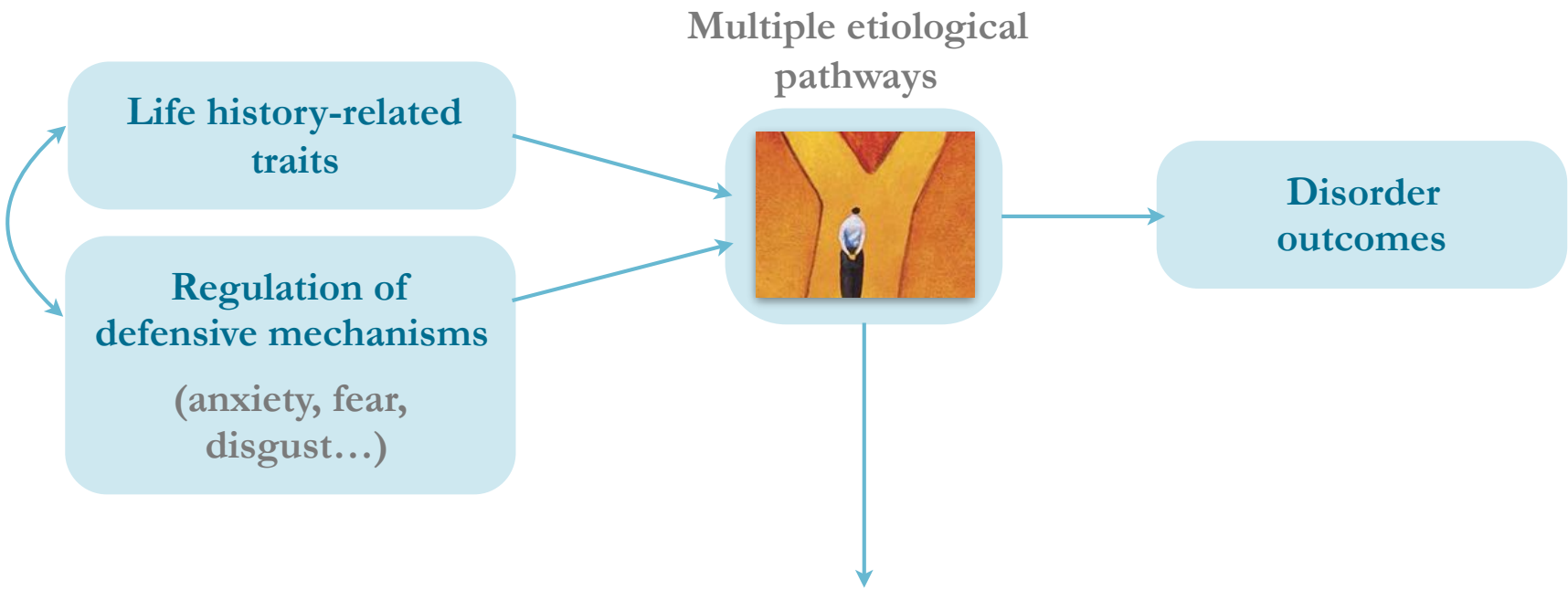
(e.g., Belsky et al., 1991; Del Giudice, 2009, 2014; Figueredo et al., 2005, 2006, 2009; Réale et al., 2010)

Fast-slow continuum as a functional **organizing principle** of individual differences:



From **individual differences** to **psychopathology** (Del Giudice, 2014):





1. Adaptive traits may be **regarded as symptoms**
   - exploitative strategies (e.g., psychopathy)
   - aversive but adaptive defenses (e.g., anxiety)

2. Adaptive traits may be **expressed at maladaptive levels**
   - genetic + environmental factors
   - assortative mating

3. Adaptive strategies may yield **individually maladaptive outcomes**
   - maladaptive learning
   - errors in defense activation ("smoke detector principle;" Nesse, 2001, 2005)

4. Adaptive traits may increase **vulnerability to dysfunctions**
   - e.g., deleterious mutations, pathogens, chronic stress…

Human LH strategies: an extended model

Del Giudice (2018). Evolutionary psychopathology: A unified approach. OUP.

Innovation 1: **dual status hierarchies**

dominance
vs.
prestige

Creativity, courtship skills
(+openness/imagination)

Technical skills
"systemizing"
(–openness/imagination)

(–nurturance/agreeableness)

Indirect parental investment
extended provisioning
wealth inheritance

Innovation 2: Multi-generation **resource transfer**

→ Differentiated **behavioral/cognitive profiles** within fast and slow strategies

[Psychopathy]

Antagonistic/exploitative

Prosocial/caregiving

Lower 5-HT activity ⟷ Higher 5-HT activity

Fast ⟵ ⟶ **Slow**

+ Impulsivity, risk-taking, sensation seeking
  Precocious, unrestricted sociosexuality
− Long-term orientation; unstable attachments
− Affiliation, cooperation, affective empathy
− A, C, H

− Impulsivity, risk-taking, sensation seeking
  Delayed, restricted sociosexuality
+ Long-term orientation; stable attachments
+ Affiliation, cooperation, affective empathy
+ A, C, H

Seductive/creative

+ Mentalistic cognition
+ Verbal ability, verbal and/or artistic creativity
− Aggression
+ O (imagination)

Basic model

Skilled/provisioning

+ Mechanistic cognition
+ Perceptual/rotation ability
− Affiliation
− A, O (imagination)

Extended model
(strategic profiles)

[Narcissism, schizotypy]

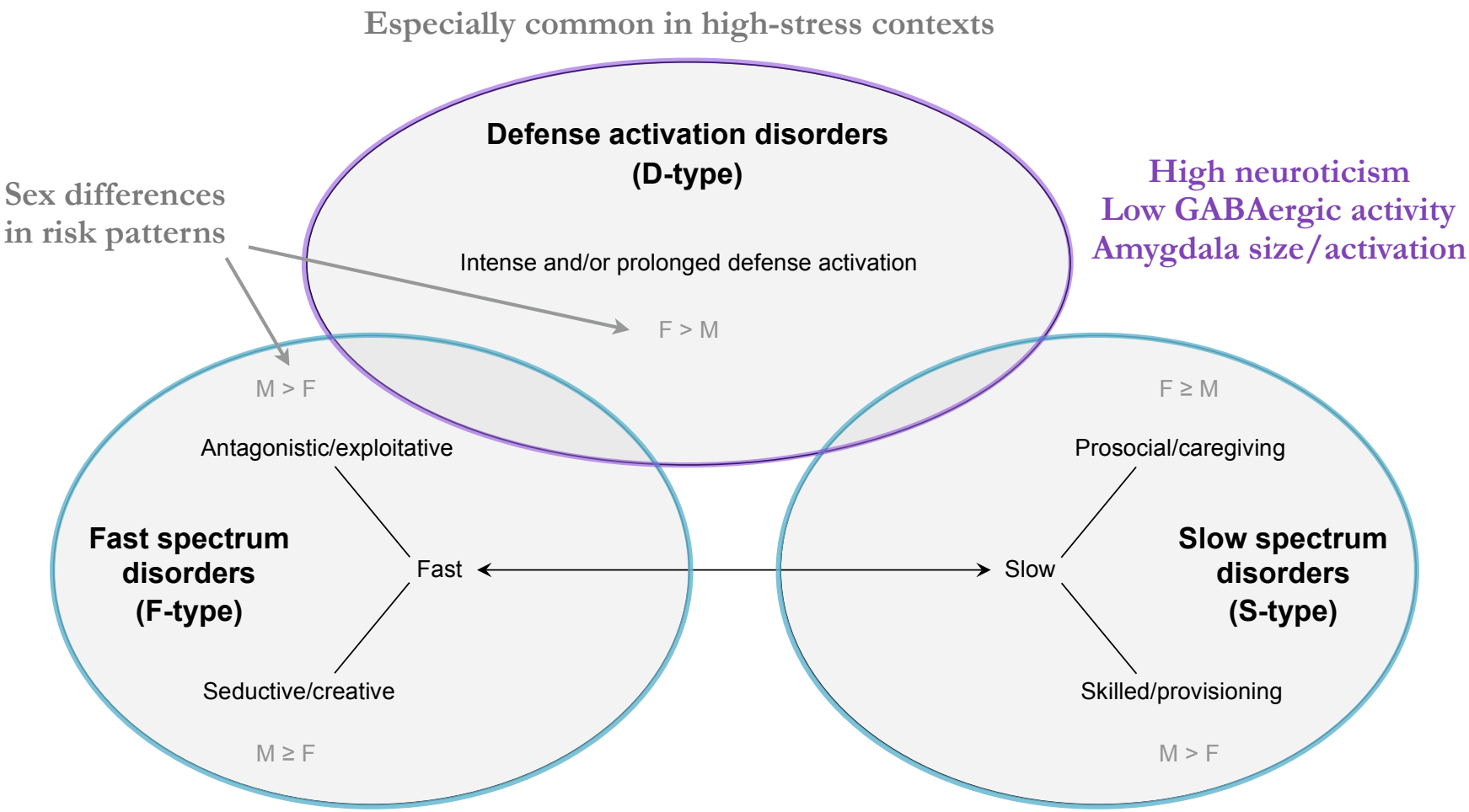
[Autistic-like traits]

High OT activity
High default mode activity (DMN)

Low OT activity
Low default mode activity (DMN)

(+ DA activity, sex hormones, stress physiology…)

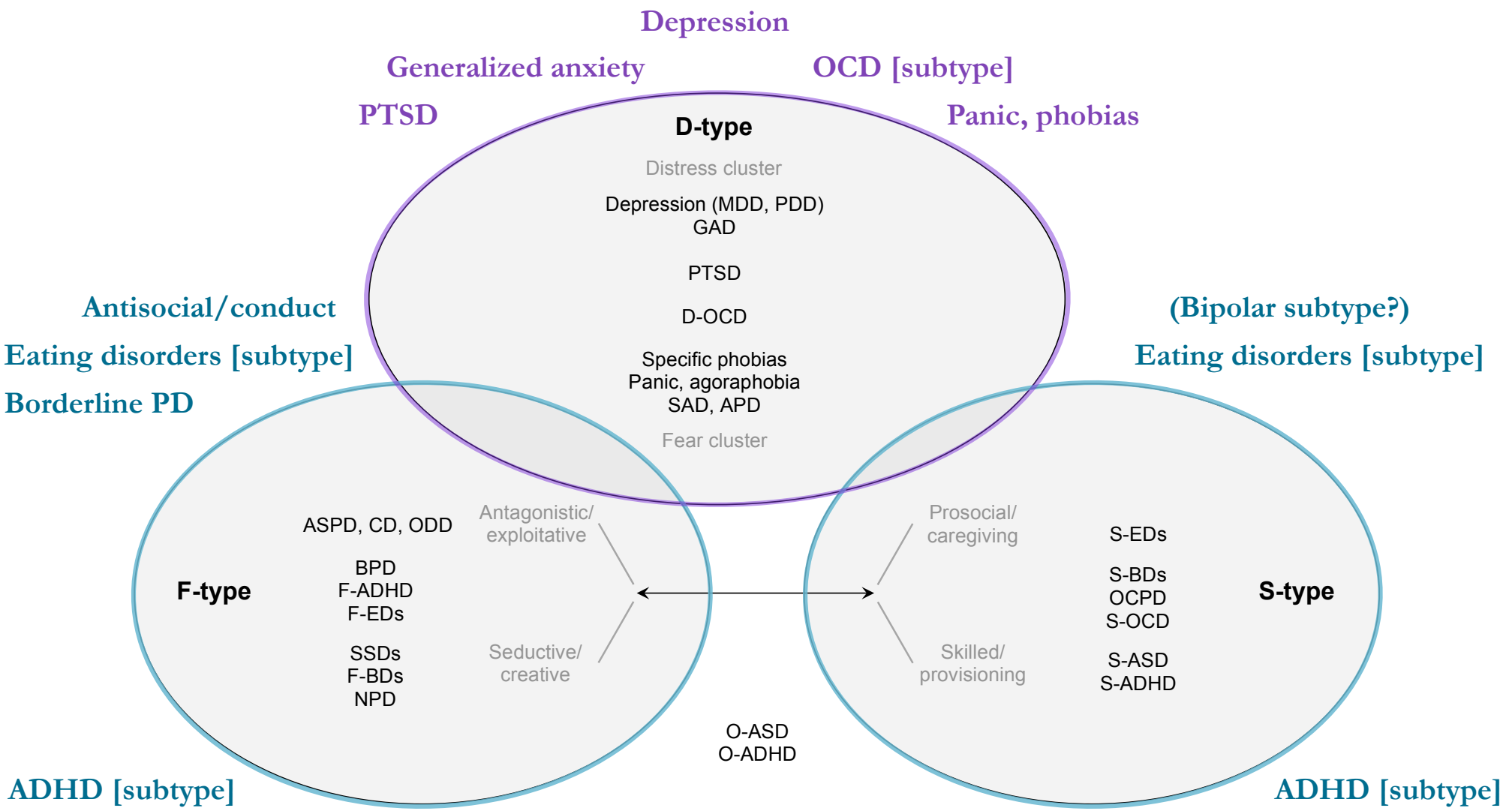
The FSD model



- **F-type, S-type, D-type**: broad clusters of comorbidity with similar **functional correlates**
- D-type disorders: may occur at **both ends** of the continuum (but more frequently with F-type)

Del Giudice (2018)

DSM categories in the FSD model:



Del Giudice (2018)

Example 1: eating disorders

Sexual competition model (Abed, 1998; Ferguson et al., 2011)

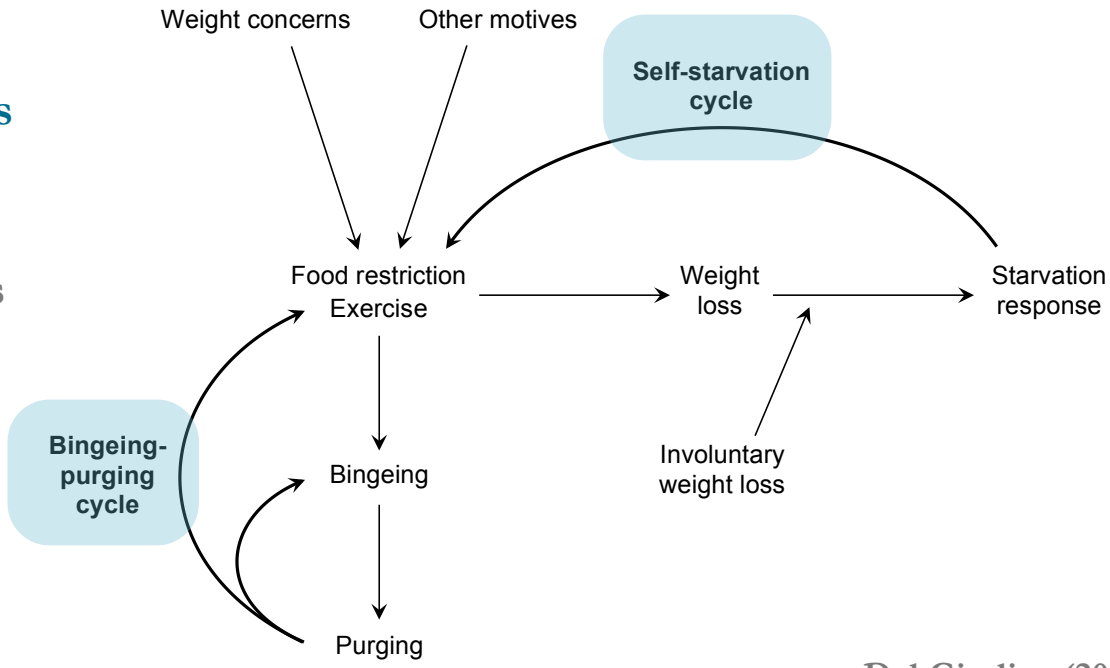
- thinness as cue of **youth** and **reproductive potential**
- social/cultural factors promote runaway competition for thinness
- robust associations with intrasexual competitiveness, mating motives

Limitations:

- historical/cross-cultural evidence of **moral, ascetic motives** (Keel & Klump, 2003)
- evidence of **status competition + perfectionism** in AN symptoms (Faer et al., 2005)

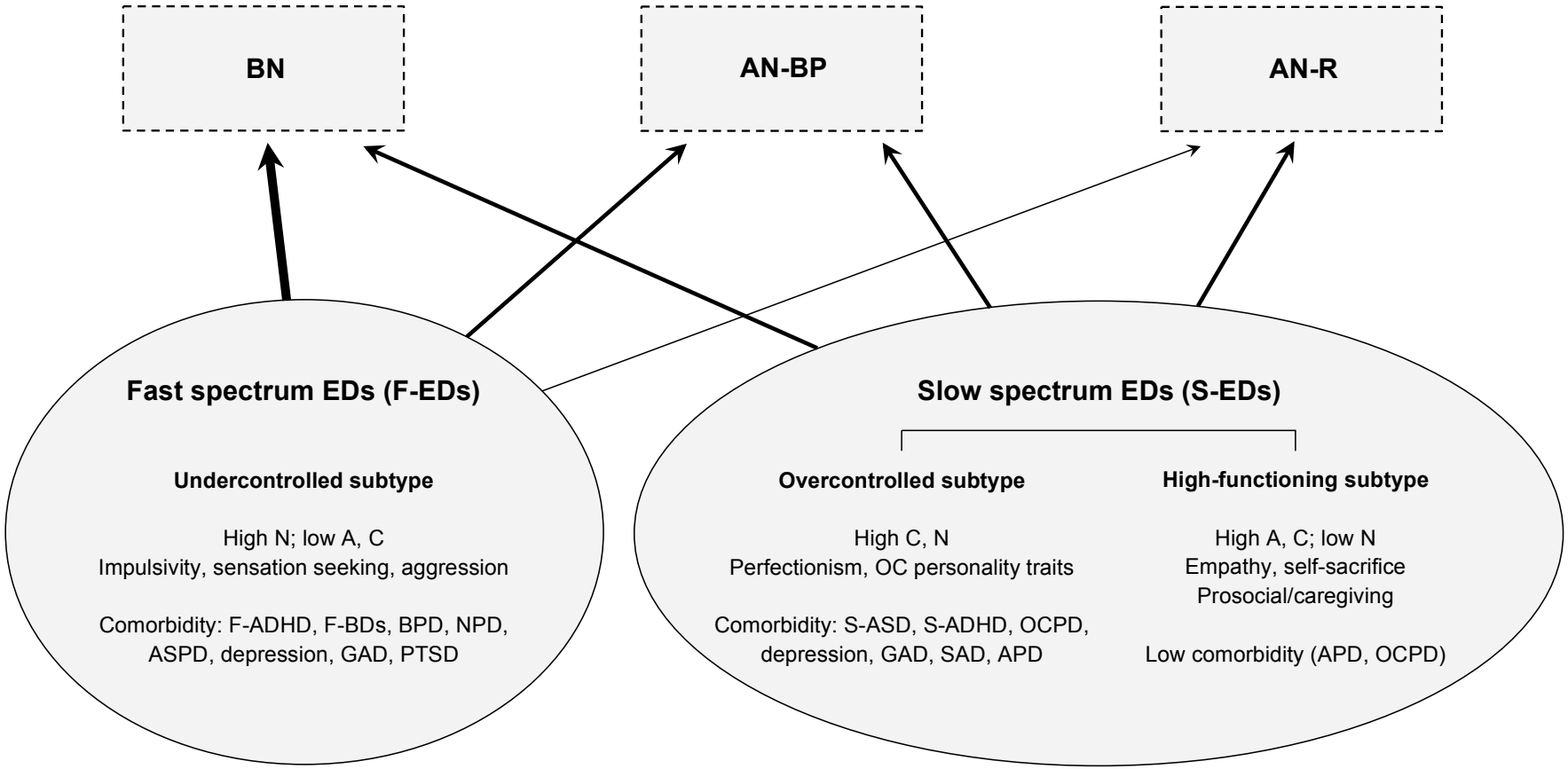
Multiple mechanisms, multiple motivations

Bingeing-purging and **self-starvation**
as common final pathways for ED symptoms
(Fessler, 2002; Dwyer eat al., 2011)



Del Giudice (2018)

FSD classification: two main functional subtypes



- subsumes ED **personality subtypes** (Westen & Harnden-Fischer, 2001; Thompson-Brenner et al., 2005, 2008)

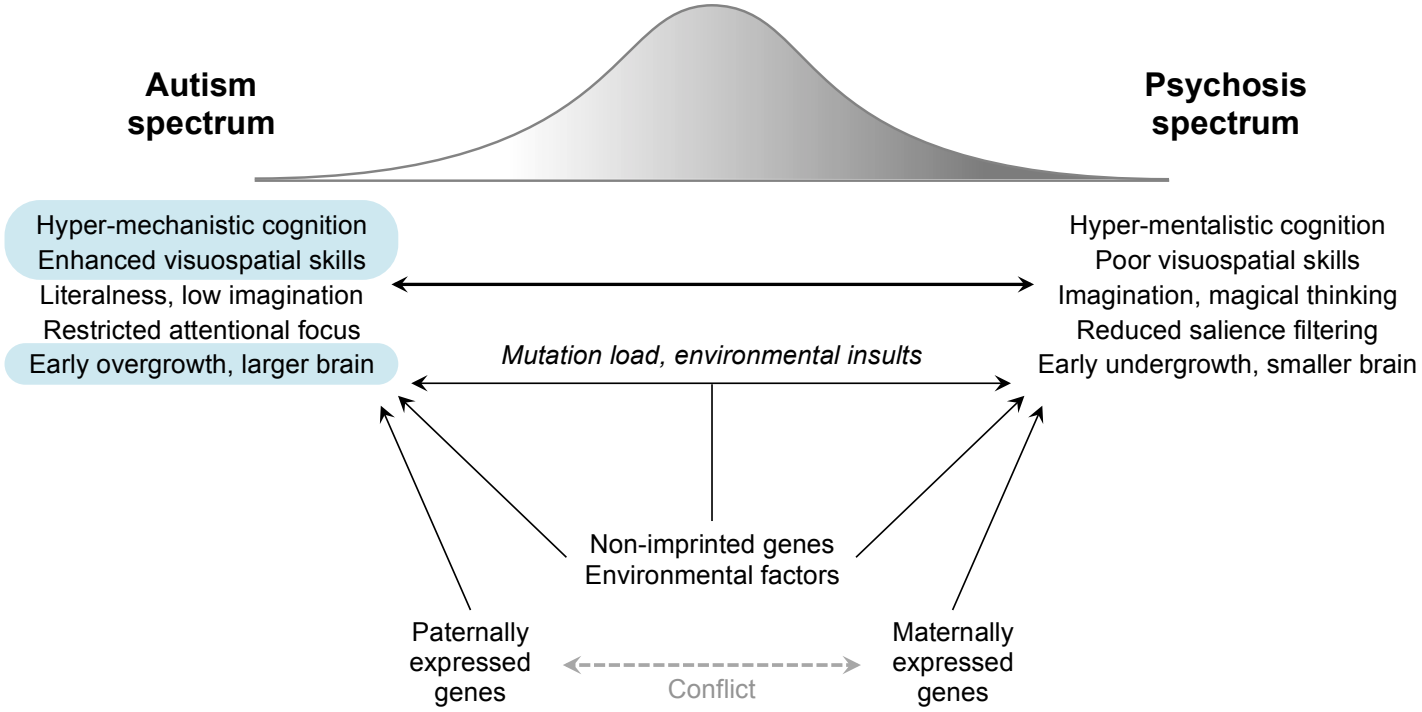
- differential associations with **socioeconomic status, maturation timing,** + other risk factors

- neurobiological implications: e.g., **5-HT** is low in BN and acute AN, but **high in recovered AN** (slow trait marker vs. transient side effect of starvation)

Del Giudice (2018)

Example 2: the autism spectrum

Diametrical model of autism/psychosis (Crespi & Badcock, 2008; Crespi et al., 2010)



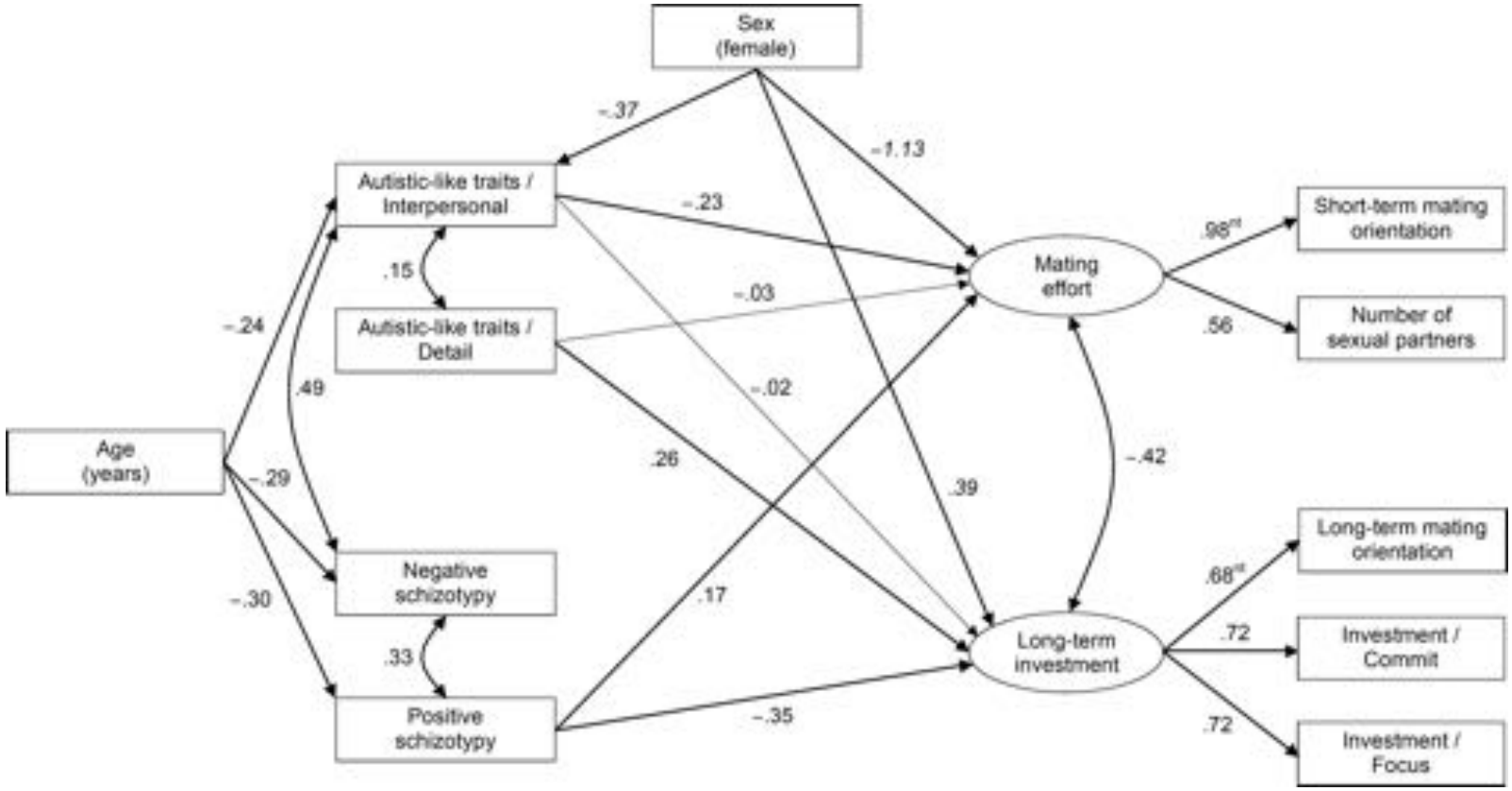
- cognitive development in autism: delays, maintenance of **childhood-typical traits** (Crespi, 2013)
- byproduct of **recent selection** for visuospatial skills/problem solving? (Crespi, 2016)
- genetic associations with **higher IQ** (Clarke et al., 2016; Hagenaars et al., 2016)
- roles for rare deleterious mutations (especially: low IQ) **AND** common genetic variants (high IQ)

Autistic-like traits →

+ long-term mating motivation
– short-term mating motivation
+ moral/sexual disgust

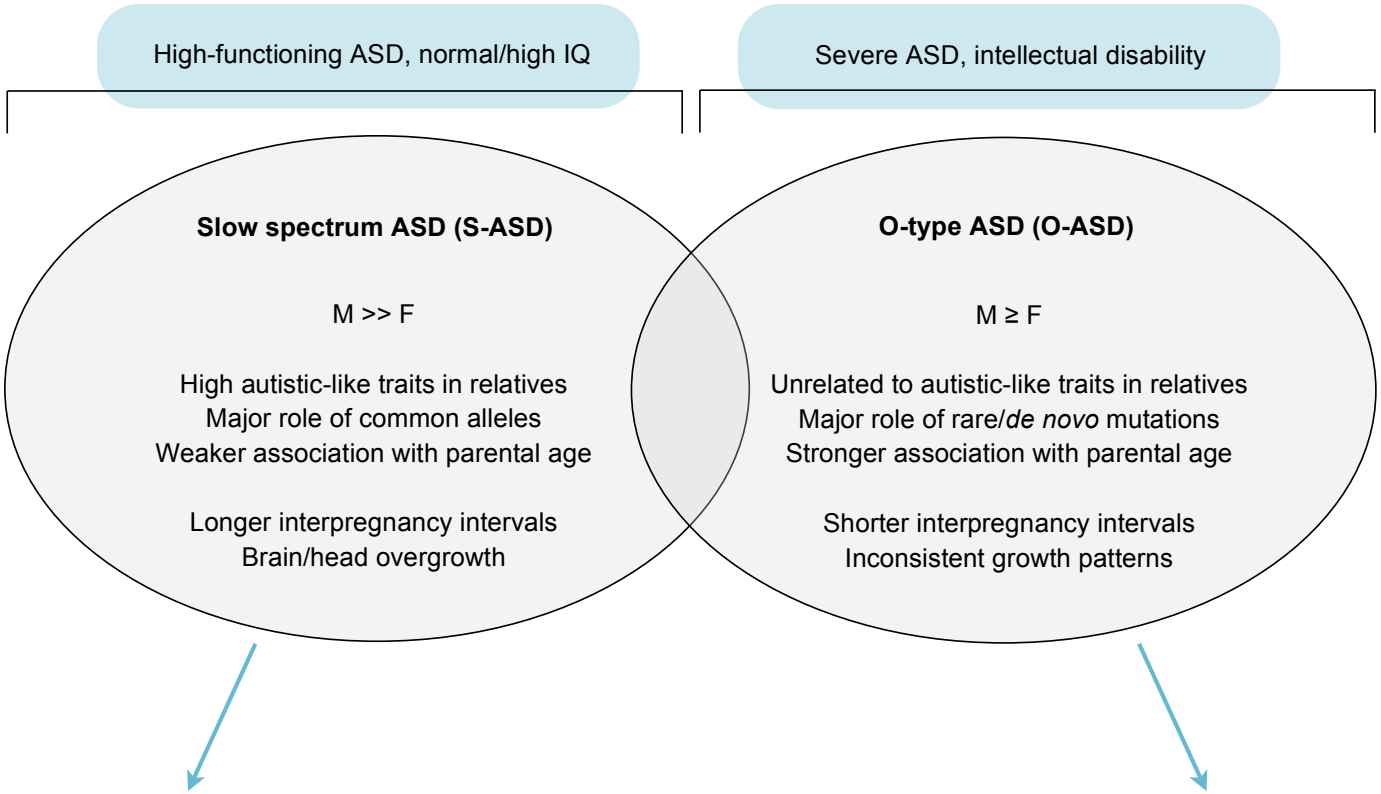
– impulsivity, risk-taking

+ conscientiousness
– extraversion
– openness (imagination)
– agreeableness



Del Giudice et al. (2010, 2014)

FSD classification: overlapping subtypes



Autistic-like traits as **slow LH variant**
(skilled/provisioning profile)

Just **one of several** "extreme male brains!"
(Baron-Cohen, 2003)

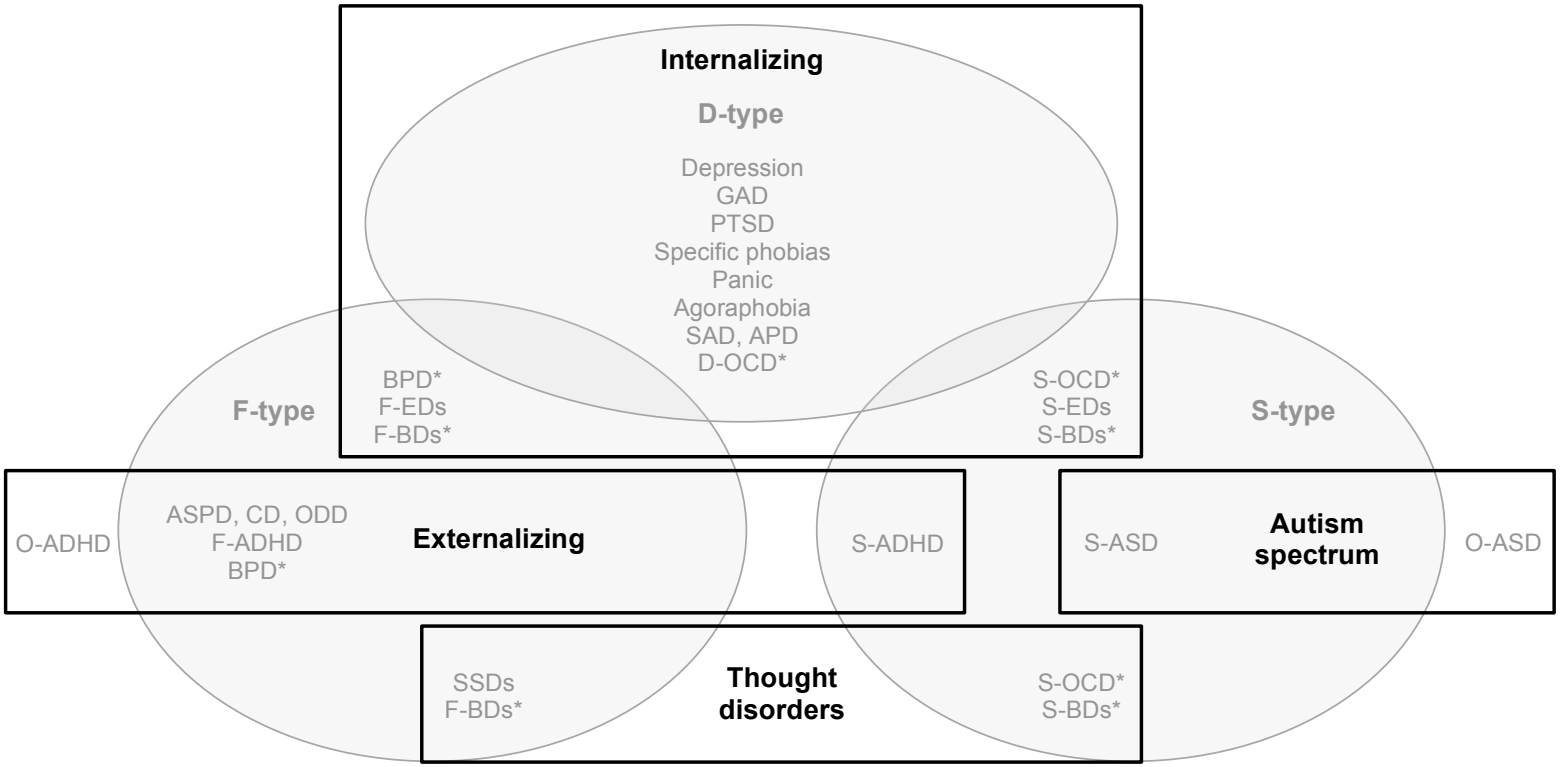
Early overgrowth, long interpregnancy interval:
high maternal investment

S-ASD: gradient from adaptive to maladaptive
(overexpression, cliff-edged fitness?)

O-ASD: pure **harmful dysfunction**
(driven by rare mutations)

Del Giudice (2018)

FSD model vs. standard transdiagnostic model

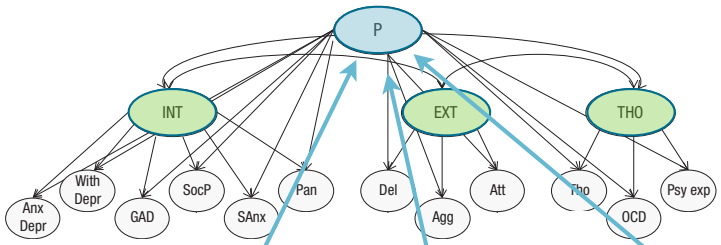


Why the differences?

- many DSM disorders contain **functionally distinct subtypes** (not considered in the standard model)
- some subtypes are functionally **unrelated** to personality variation (e.g., most severe ASD cases)
- standard factor analysis misses **nonlinear associations** (e.g., D-type disorders elevated at both ends)

Del Giudice (2018)

What is the p factor?



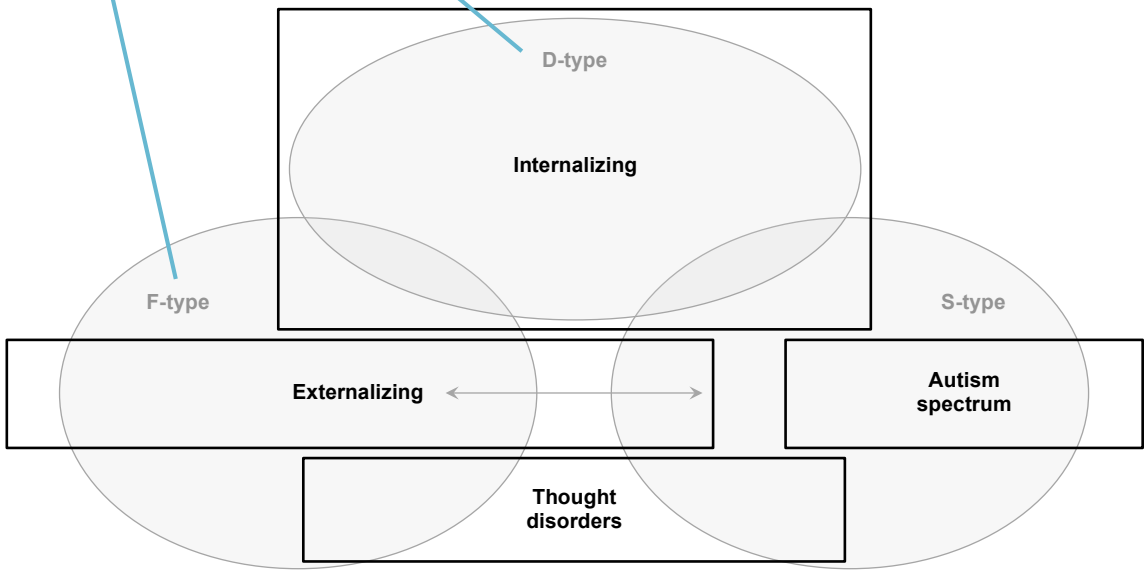
p factor =

Fast LH (low A, low C, impulsivity)

+ defense upregulation (high N)

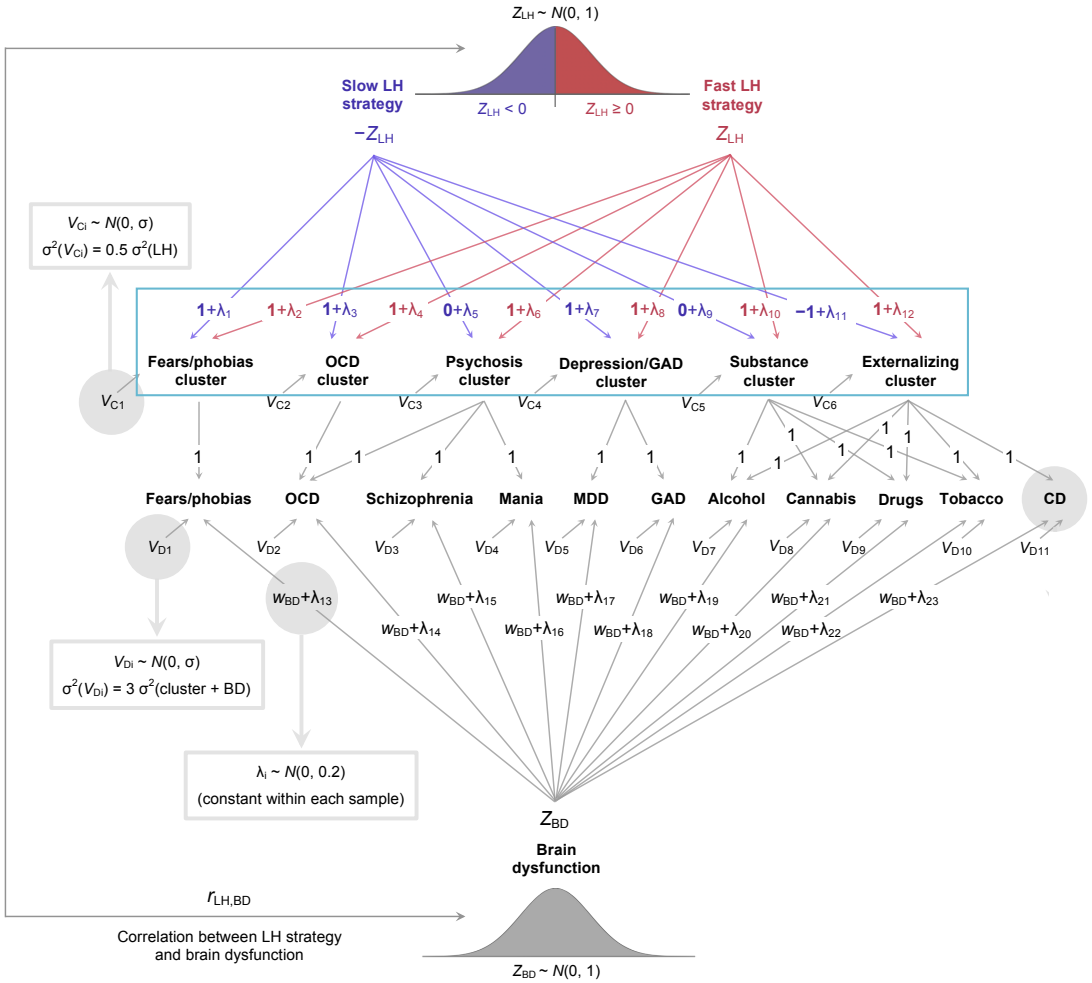
+ low cognitive ability (low IQ)

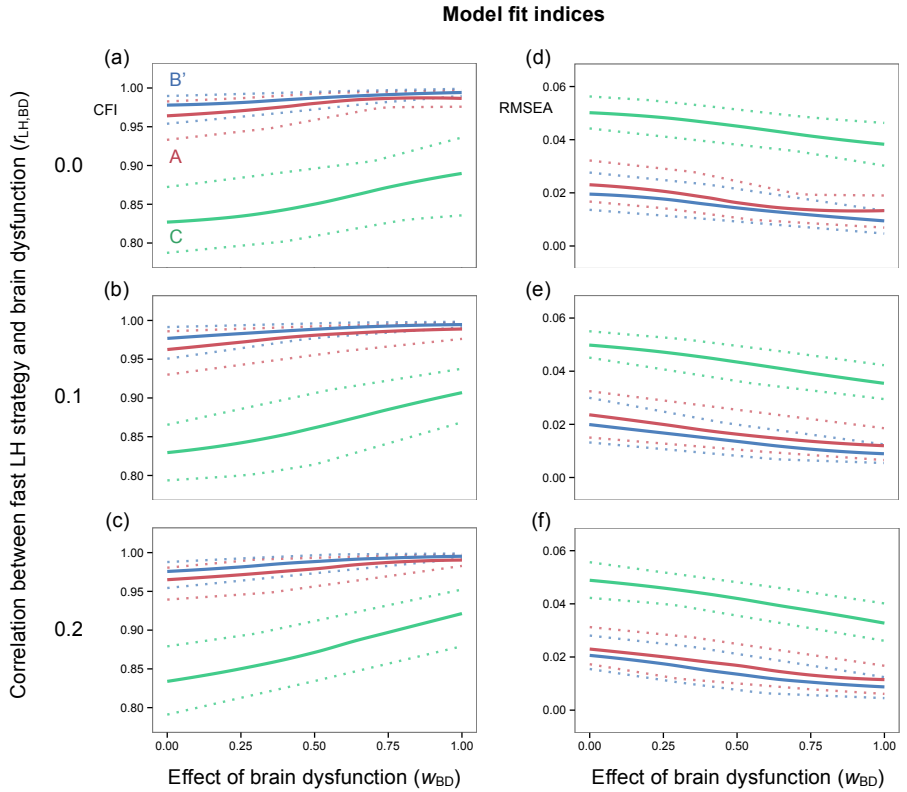
Low IQ
Neurological impairment



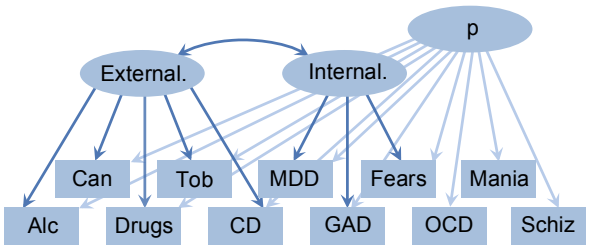
A unitary p factor may emerge from **functionally (and statistically!) independent** dimensions of variation

Simulation study: Del Giudice (2016)



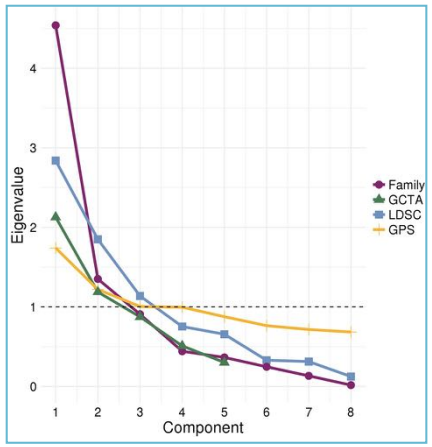


Model **B'**: hierarchical / bifactor

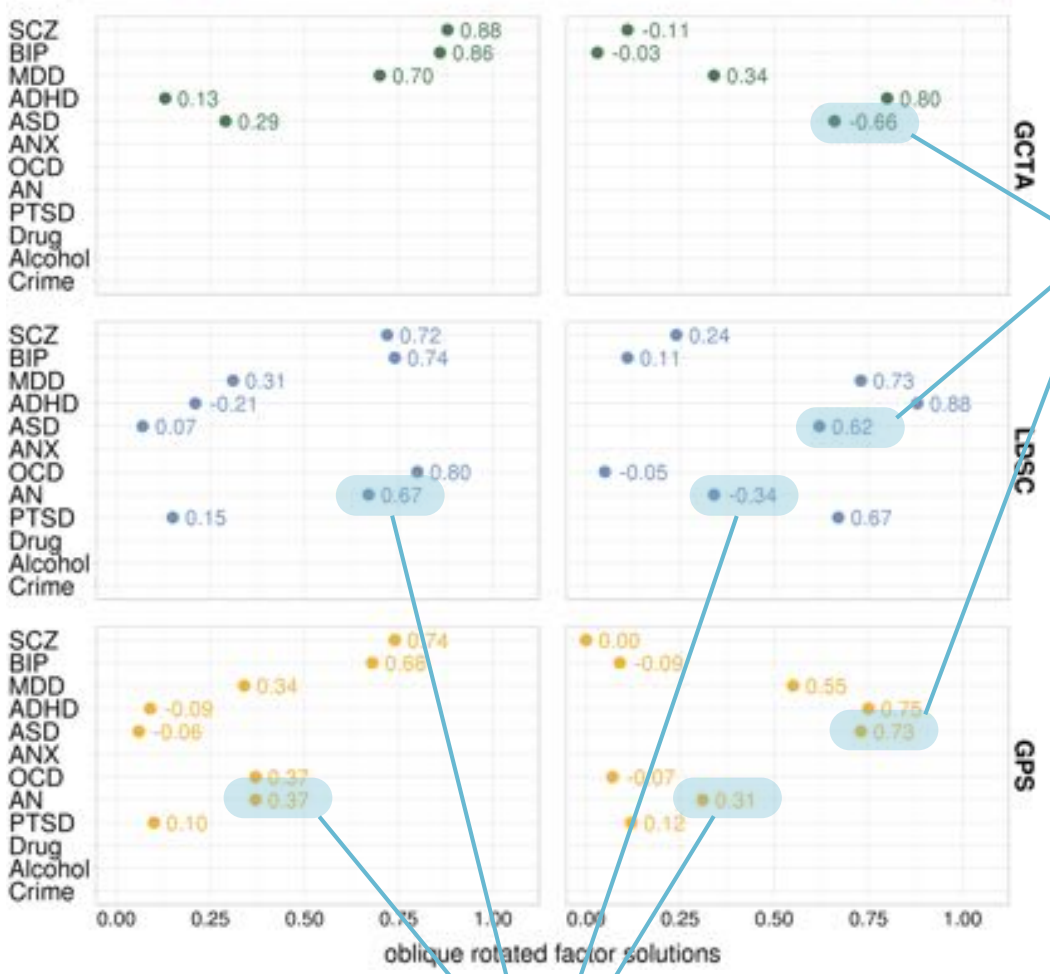


when split into **two factors**…

Polygenic p factor



Selzam et al., 2018



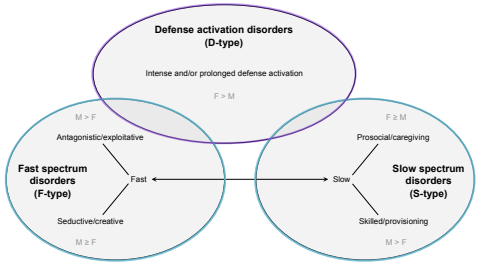
ASD dissociates
(common alleles)

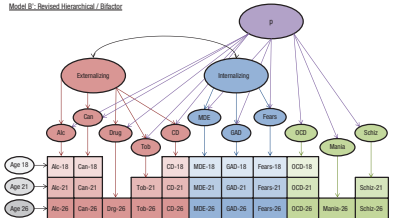
cross-loadings
for **AN**

In conclusion…

A life history approach may help **overcome fragmentation** in evolutionary psychopathology

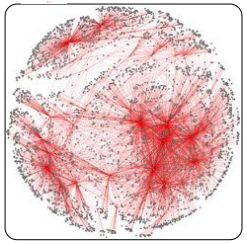
The framework provides the foundation for an **alternative classification system**

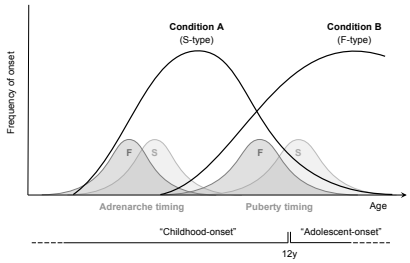




The FSD model successfully reproduces the **large-scale structure** of mental disorders

Potential for deeper integration with **behavior genetics, individual differences, computational models**





Implications for **epidemiology** and **developmental psychopathology**

Thank you!

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