Comorbidity Prevalence and Treatment Outcome In Children and Adolescents With ADHD

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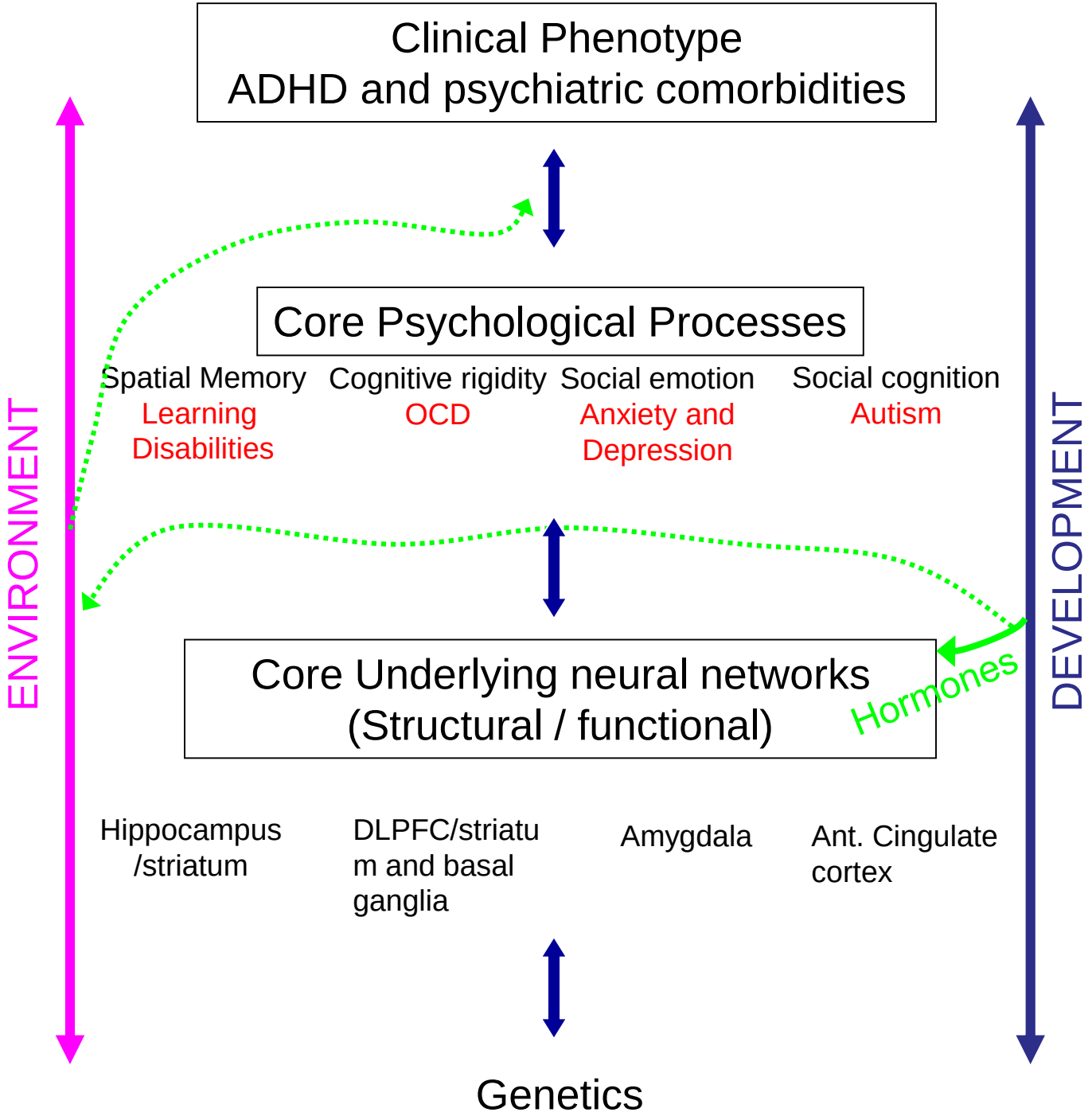
ULTIMATE GOALS

- Recognition
- Development and timing
- Risk (and protective) factors and vulnerability
- PREDICT

  - outcome

  - treatment response
- *“DIAGNOSE”*

INTERMEDIATE STEP: UNDERSTAND MECHANIMS

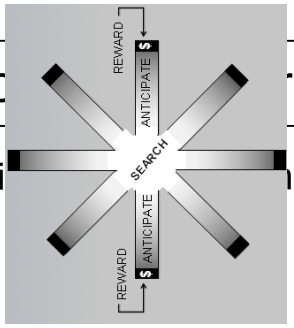

Clinical Phenotype
ADHD and psychiatric comorbidities
Core Psychological Processes
Spatial Memory
Learning Disabilities
Cognitive rigidity
OCD
Social emotion
Anxiety and Depression
Social cognition
Autism
ENVIRONMENT
DEVELOPMENT
Core Underlying neural networks
(Structural / functional)
Hormones
Hippocampus /striatum
DLPFC/striatum and basal ganglia
Amygdala
Ant. Cingulate cortex
Genetics

C s
DEPRE CTION

ENVIRONMENT

DEVELOPMENT

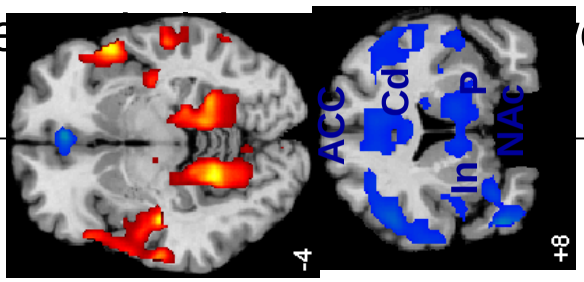
Core Psyc rocesses



Spatial Memory Valence/sali notion Social cognition

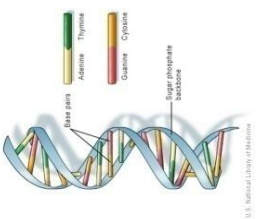
Hormones

Core orks

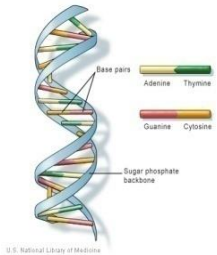


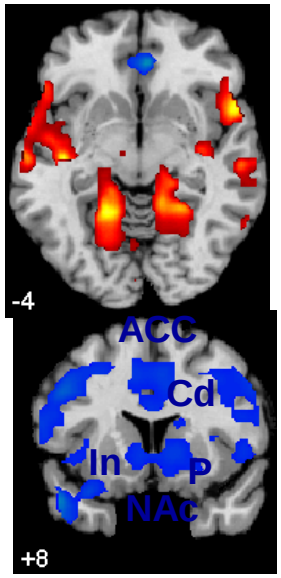
Hippocampus
/striatum

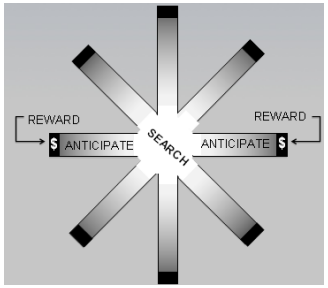
Ant. Cingulate
cortex



ENVIRONMENT







DEVELOPMENT

Genotype

Endophenotype

Neuropsychological phenotype

Clinical phenotype

Message

✓Clinical pictures that have different characteristics may share neurobilogical alterations

✓The study of endophenotypes shared in multiple conditions

❖**individualized therapy**

❖evolution of psychiatric genetic studies

This model could overcome the categorical approach for a more dimensional approach

CHALLENGES
About Sample

- Patient Heterogeneity (severity, intellectual disability, gender)
- Lack of reliable markers to predict response or side effects
- Different treatment for different developmental stages

CHALLENGES
About method

- Outcome Measures (Questionnaire and scale not always reliable)
- Cross-sectional studies can help to understand comorbidity in ADHD ?
- Limited public awareness of treating ADHD children with medication

CHALLENGES
About method

Difficulty defining the natural history and developmental correlates of a disease process

• Common assumption in cross-sectional studies:

•Members of differing age cohorts who have the same diagnosis belong to the same larger population of subjects with the same biological illness

• Corollary is that younger subjects will, with time, resemble their older counterparts

•Untrue in most cases

•Most illnesses that onset earlier differ from their counterparts that onset later -- in phenomenology, familial risk comorbidities, and natural history

The lesson we have learned from previous studies

First point: there is a need to stratify patients based on clinical features and establish longitudinal development trajectories

Second point: there is need to stratify patients based on biological pathway (i.e. different patient subgroups may have different mechanism and respond to different treatments)

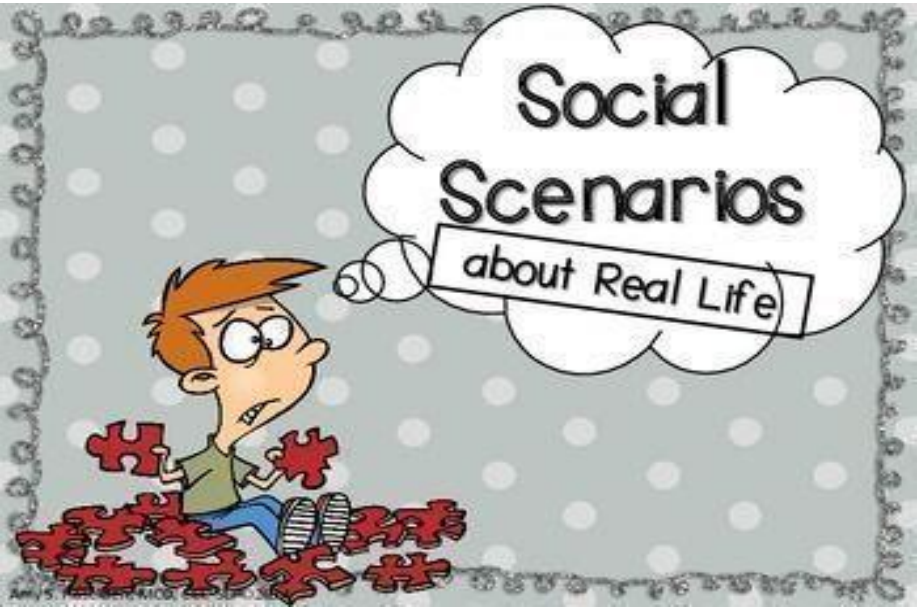
Third: Difficulty distinguishing findings of core pathological processes from epiphenomena or compensatory responses (adaptive changes are difficult to distinguish from the events that incite them)

Extend studies to progressively younger age groups prior to ADHD onset

- identify trait rather than state markers of CNS functioning that predispose individuals to particular illnesses or comorbidity
- identify these markers prior to emergence of compensatory & epiphenomenal effects
- study longitudinally before, during, and after onset to disentangle trait, state, and compensatory effects

Drug treatment **Behavioral intervention**

Always keep in mind
Where we are and where we are going



about real life…

The discussion is open

- Recognition
- Development and timing
- Risk (and protective) factors and vulnerability
- PREDICT

  - outcome

  - treatment response
- *Open Challenges*

INTERMEDIATE STEP:
UNDERSTAND MECHANIMS