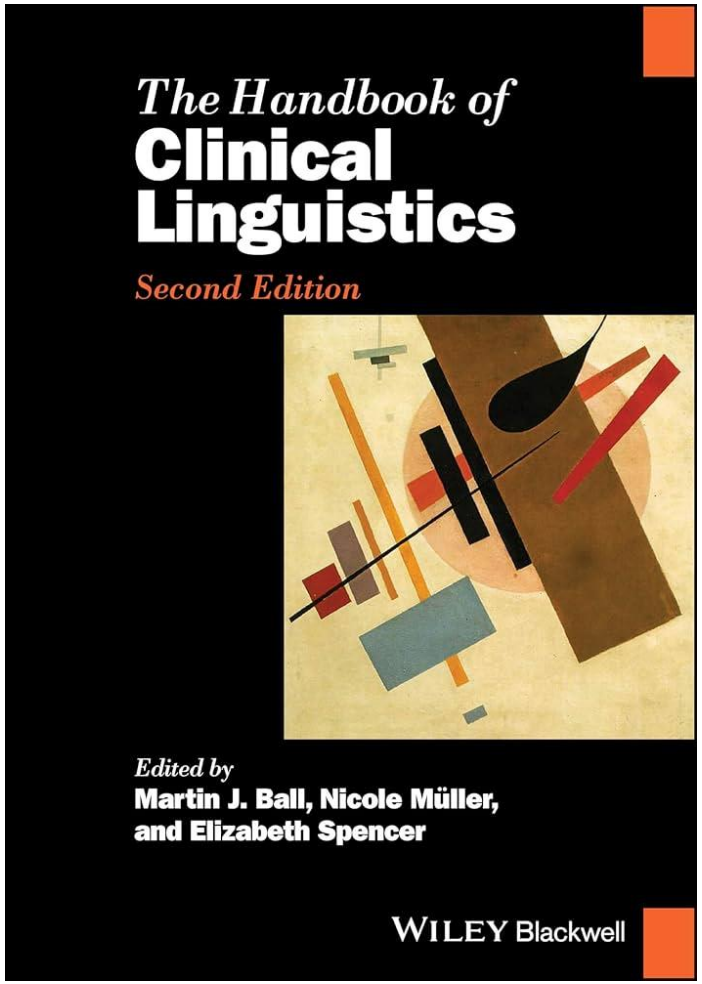
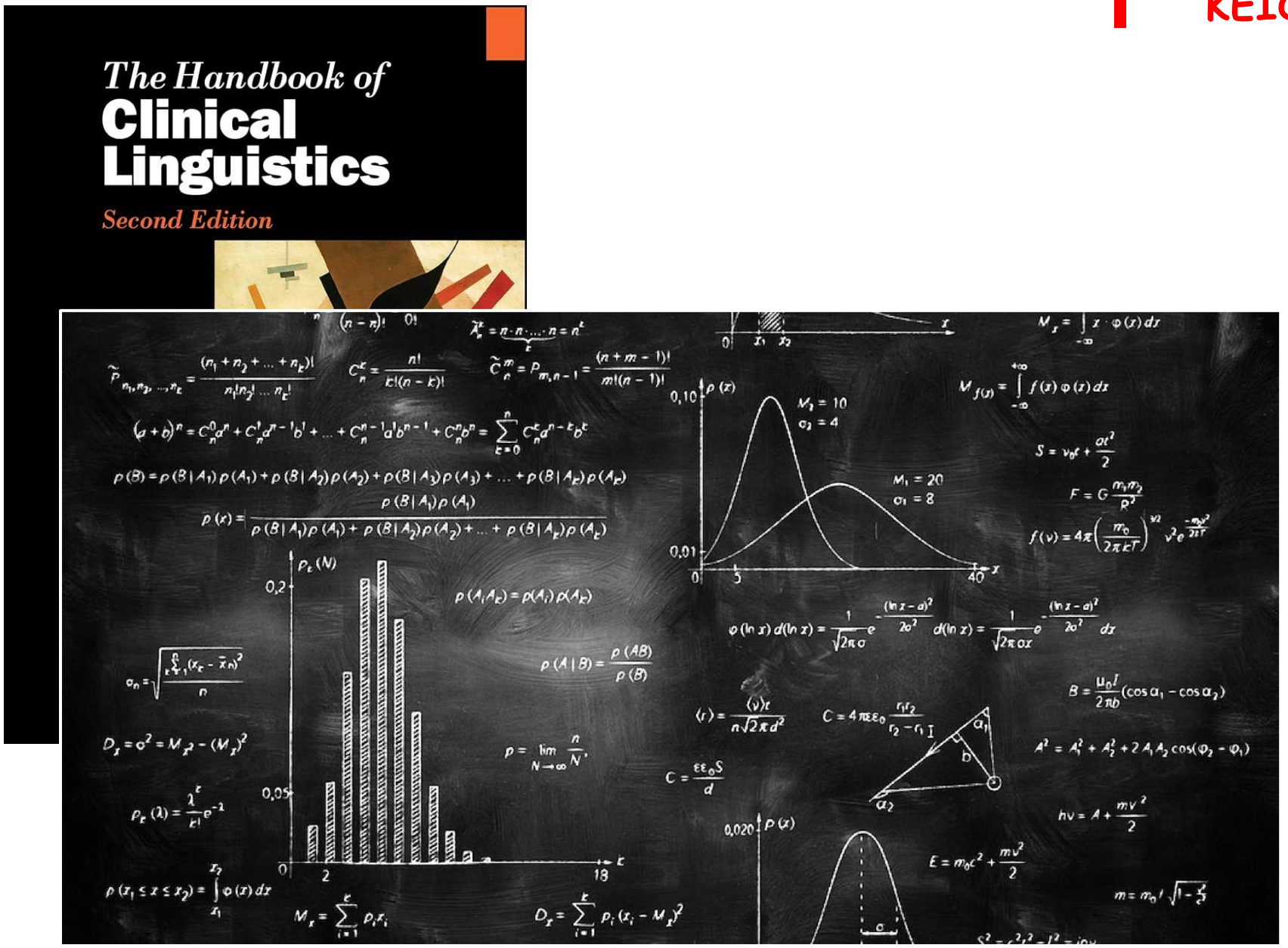
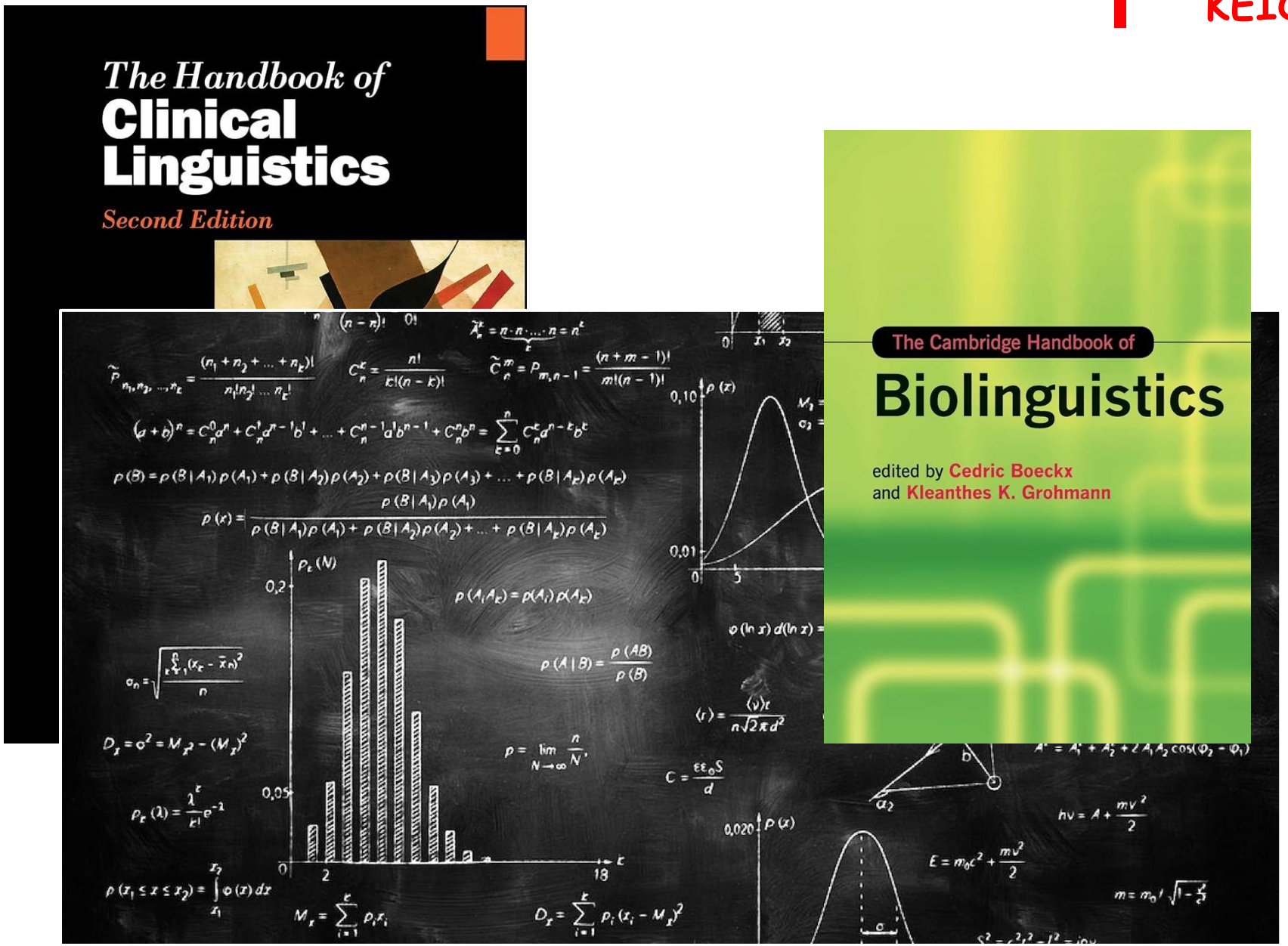
Language disorders through the lens of biolinguistics

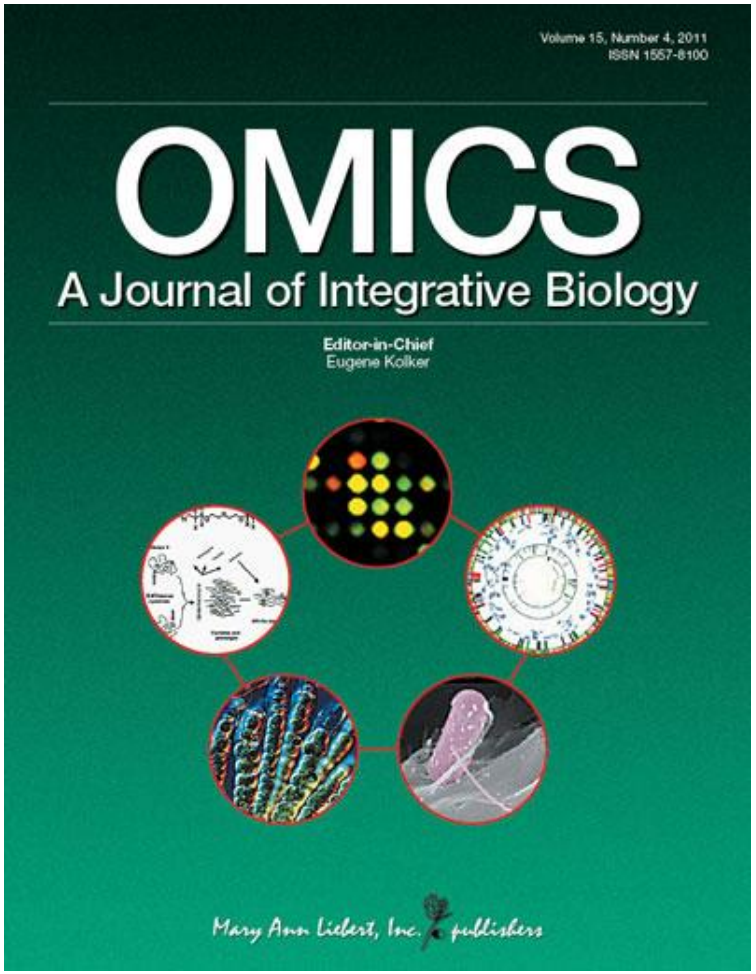
INVITED TALK 1

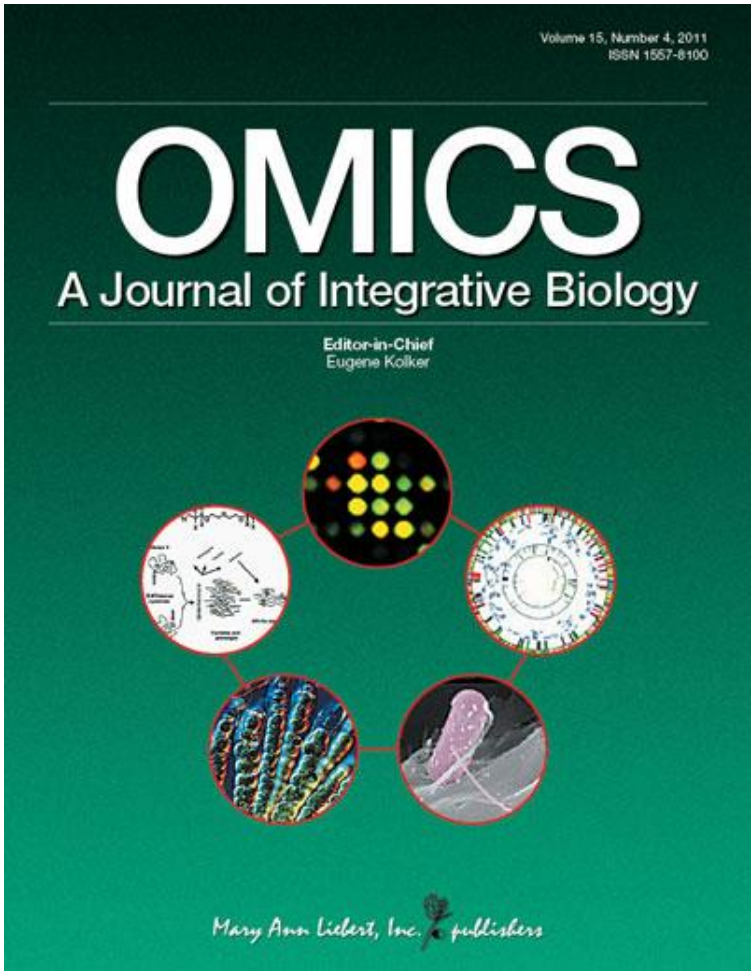
Antonio Benítez Burraco
Department of Spanish, Linguistics, and Theory of Literature (Linguistics)
University of Seville (Spain)

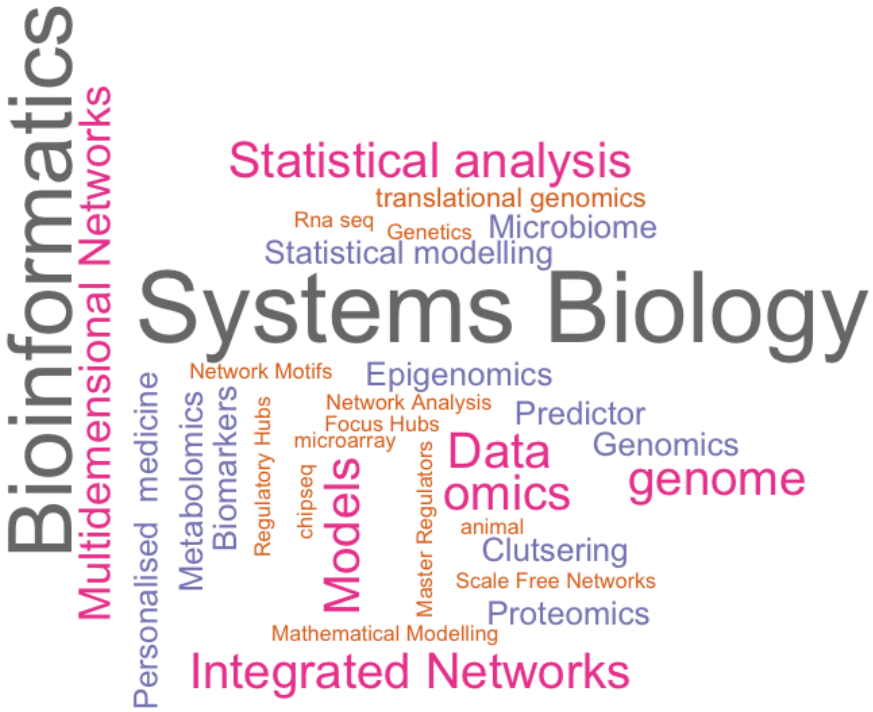

The Handbook of
Clinical
Linguistics
Second Edition
Edited by
Martin J. Ball, Nicole Müller,
and Elizabeth Spencer
WILEY Blackwell

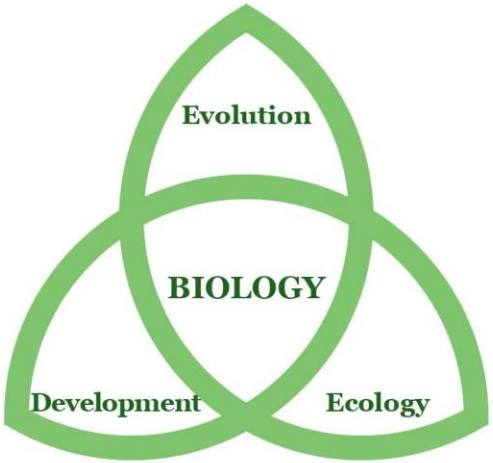

The Handbook of
Clinical
Linguistics
Second Edition

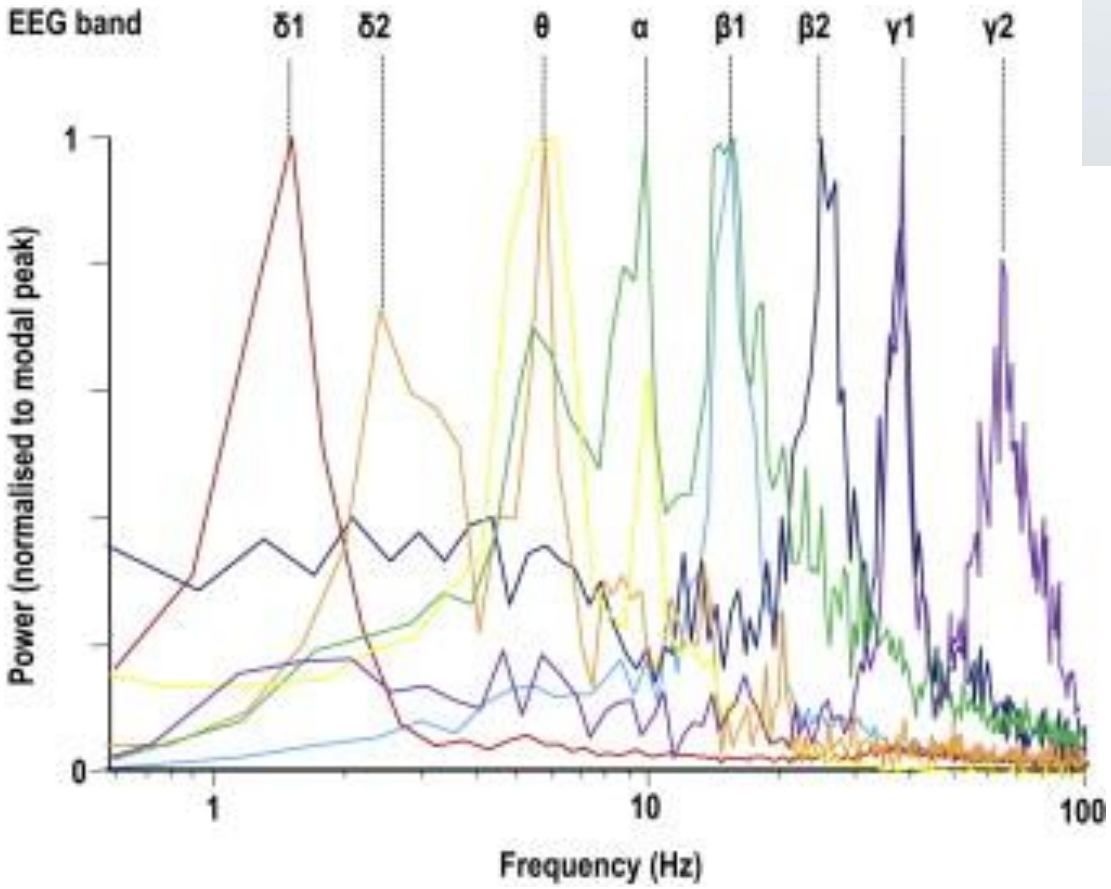

The Handbook of
Clinical
Linguistics
Second Edition
The Cambridge Handbook of
Biolinguistics
edited by Cedric Boeckx
and Kleanthes K. Grohmann


Volume 15, Number 4, 2011
ISSN 1557-8100
OMICS
A Journal of Integrative Biology
Editor-in-Chief
Eugene Kolker
Mary Ann Liebert, Inc. publishers

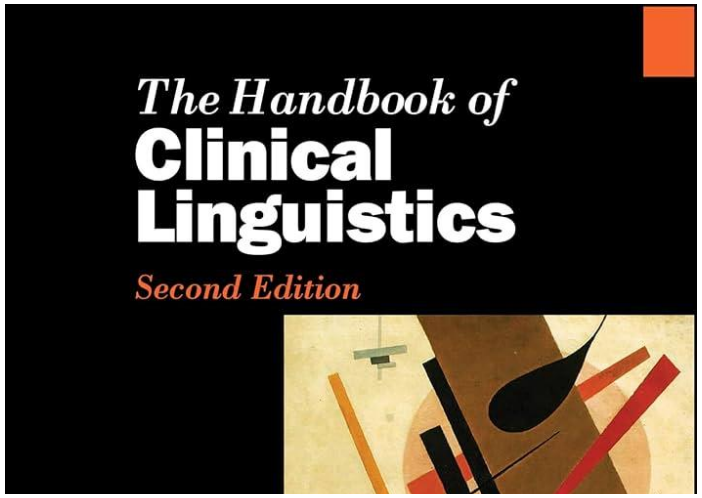

Volume 15, Number 4, 2011
ISSN 1557-8100
OMICS
A Journal of Integrative Biology
Editor-in-Chief
Eugene Kolker
Mary Ann Liebert, Inc. publishers

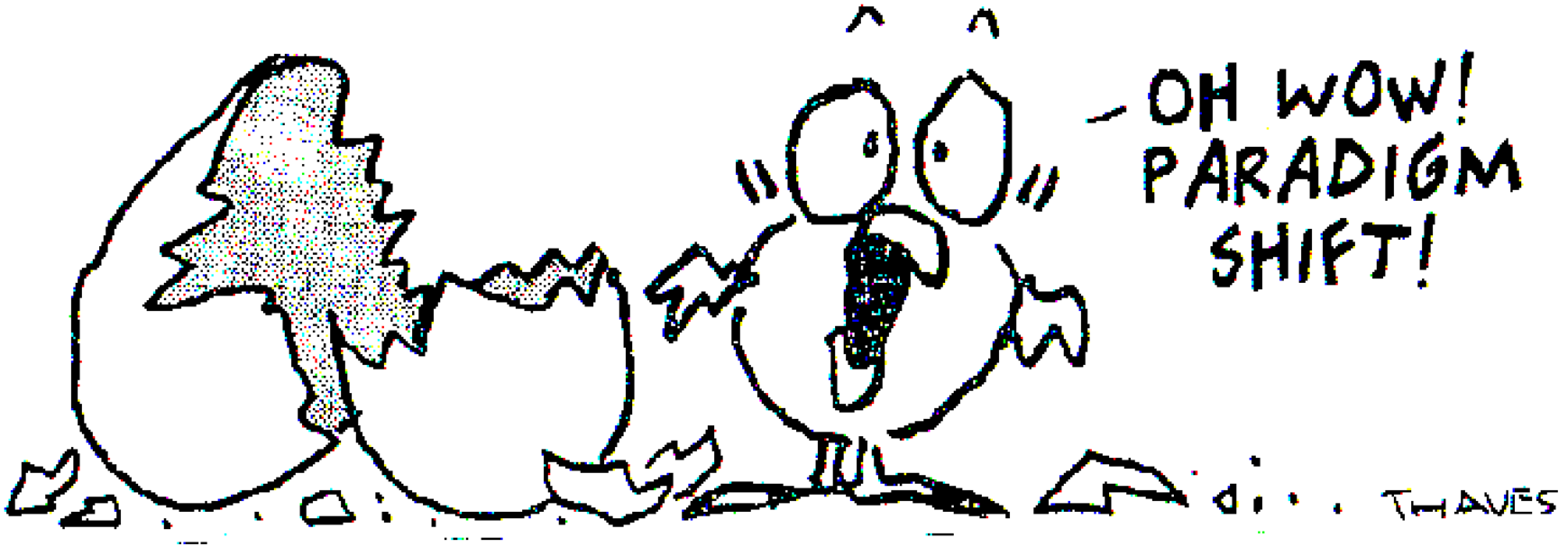

Bioinformatics
Multidemensional Networks
Personalised medicine
Statistical analysis
translational genomics
Rna seq
Genetics
Microbiome
Statistical modelling
Systems Biology
Network Motifs
Epigenomics
Network Analysis
Predictor
Focus Hubs
microarray
Data
Genomics
genome
omics
Metabolomics
Biomarkers
Regulatory Hubs
chipseq
Models
Master Regulators
animal
Clutsering
Scale Free Networks
Proteomics
Mathematical Modelling
Integrated Networks


Evolution
BIOLOGY
Development
Ecology


EEG band
δ1
δ2
θ
α
β1
β2
γ1
γ2
1
0
Power (normalised to modal peak)
1
10
100
Frequency (Hz)


Adobe Stock | #510336870


The Handbook of
Clinical
Linguistics
Second Edition

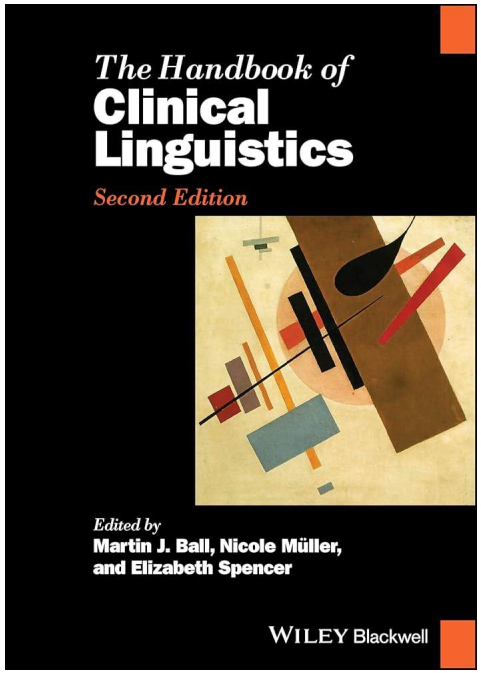

OH WOW!
PARADIGM
SHIFT!
THAVES

HOW I HAVE STRUCTURED MY TALK

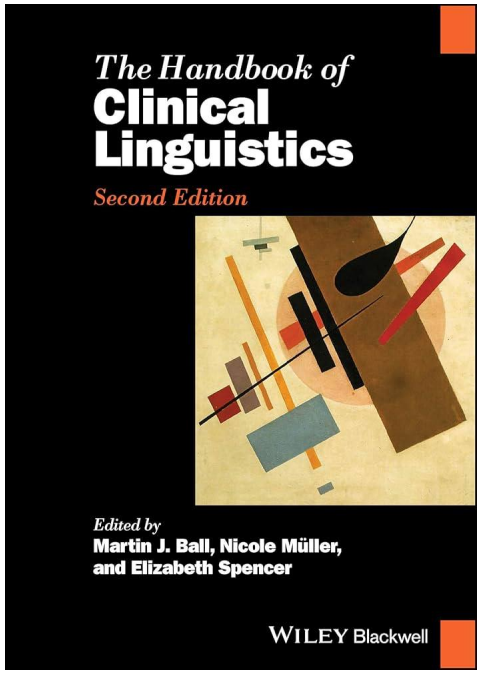
1. Introduction
2. Clinical linguistics: struggling with symptoms and causes
3. Bridging genes to language (and vice versa)
4. Putting the focus on brain oscillations
5. Conclusions and future prospects

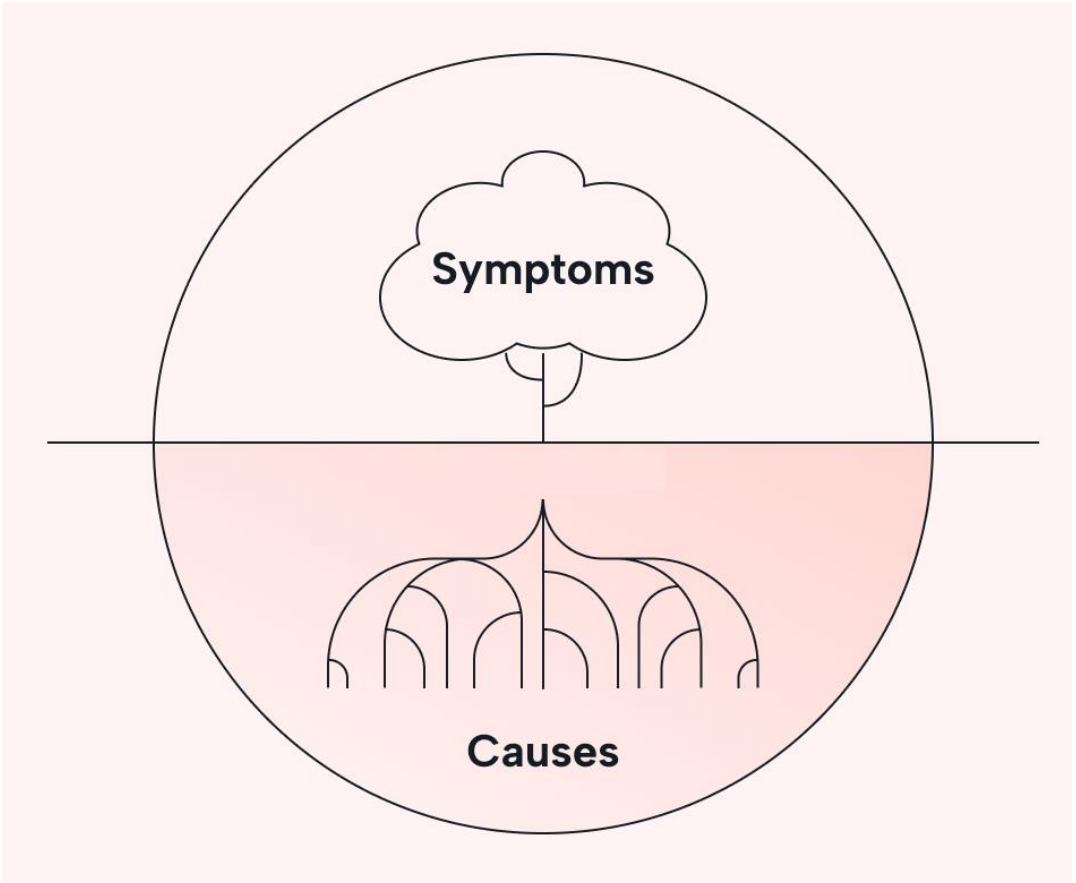
HOW I HAVE STRUCTURED MY TALK

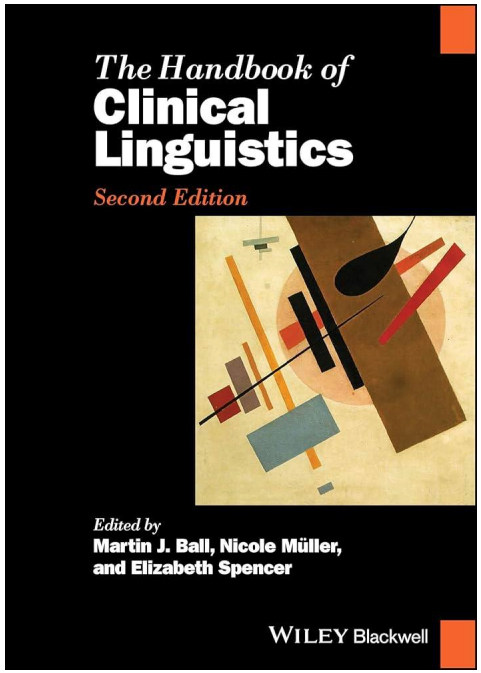
1. Introduction
2. Clinical linguistics: struggling with symptoms and causes
3. Bridging genes to language (and vice versa)
4. Putting the focus on brain oscillations
5. Conclusions and future prospects

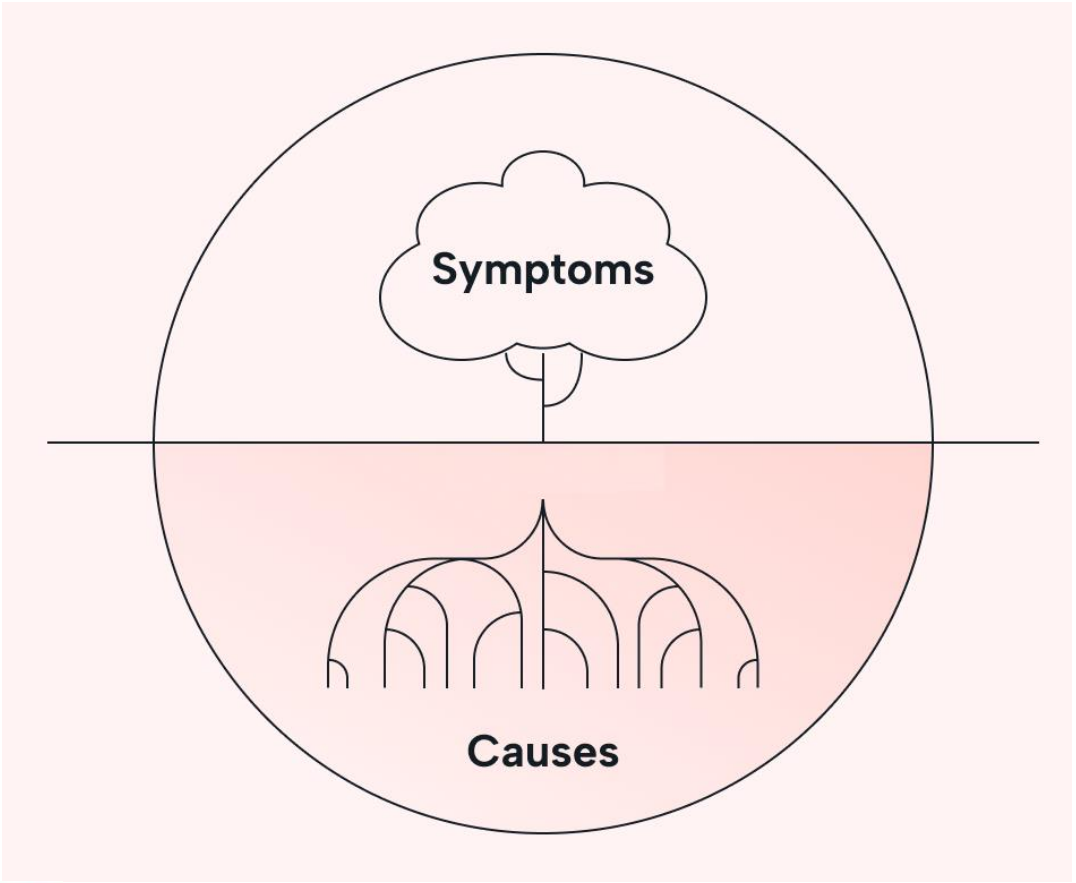


1. Description
2. Diagnosis
3. Etiology
4. Research
5. Therapy

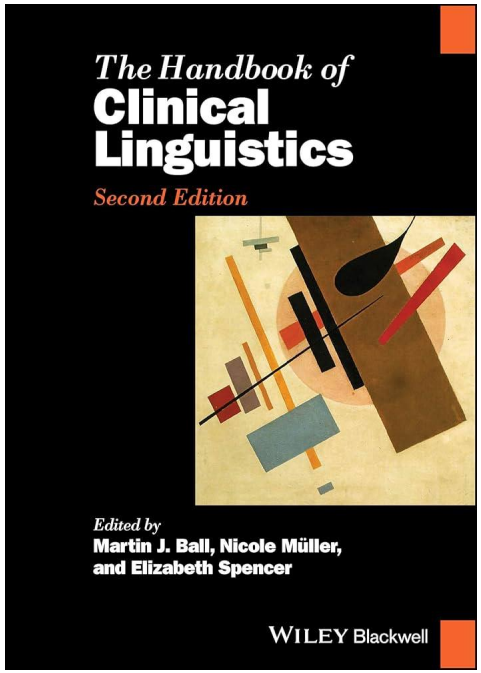

The Handbook of
Clinical Linguistics
Second Edition
Edited by
Martin J. Ball, Nicole Müller,
and Elizabeth Spencer
WILEY Blackwell

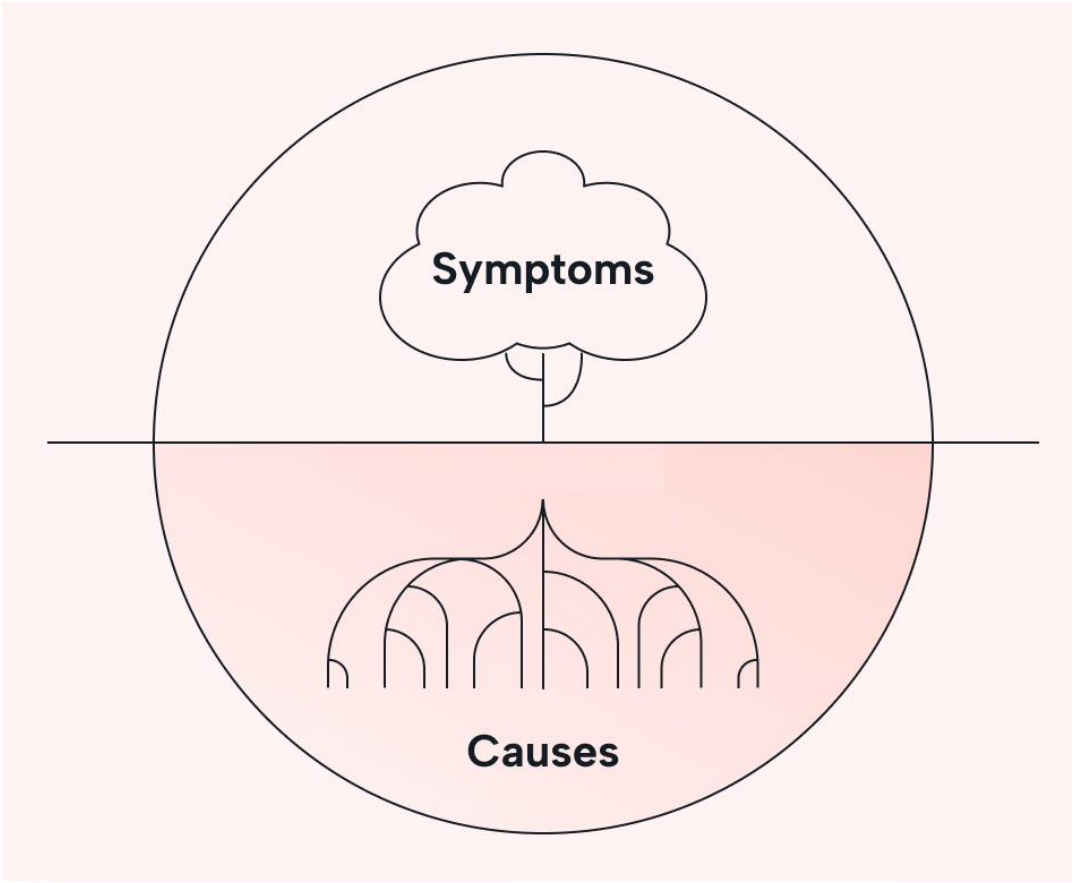

Symptoms
Causes


The Handbook of
Clinical
Linguistics
Second Edition
Edited by
Martin J. Ball, Nicole Müller,
and Elizabeth Spencer
WILEY Blackwell

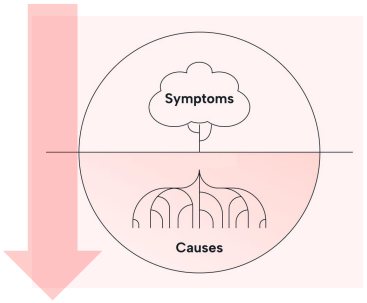

Symptoms
Causes


Developmental Language
Disorder (DLD)


The Handbook of
Clinical Linguistics
Second Edition
Edited by
Martin J. Ball, Nicole Müller,
and Elizabeth Spencer
WILEY Blackwell


Symptoms
Causes


Developmental Language
Disorder (DLD)

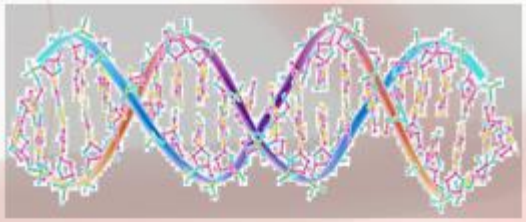

Symptoms
Causes

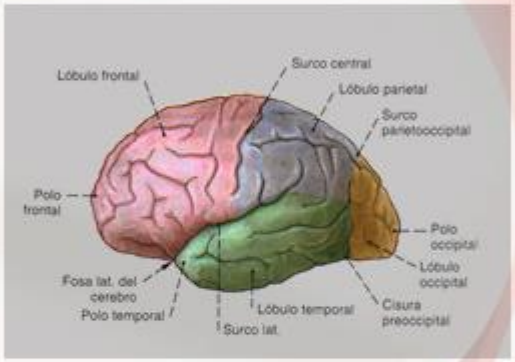
A

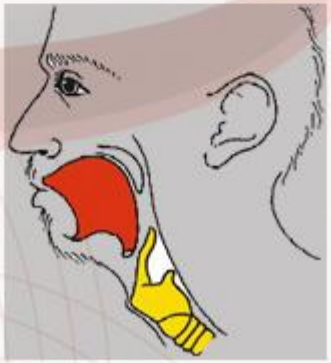
B

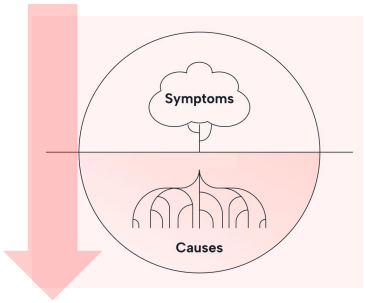
C

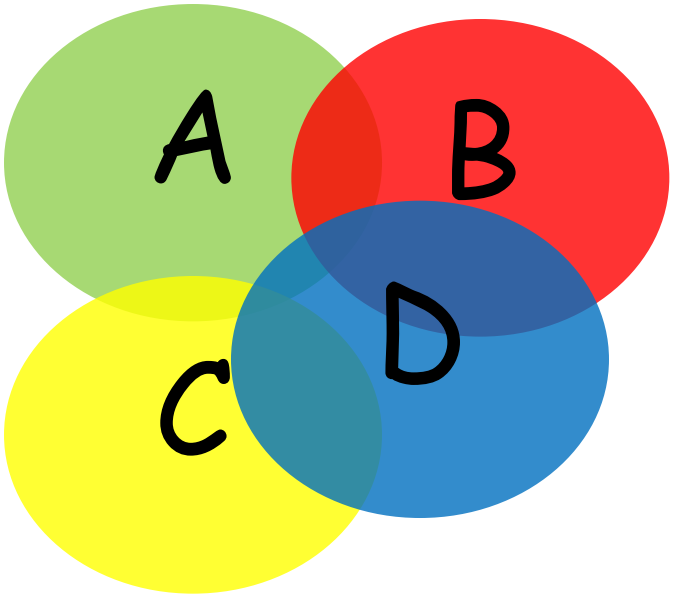
D

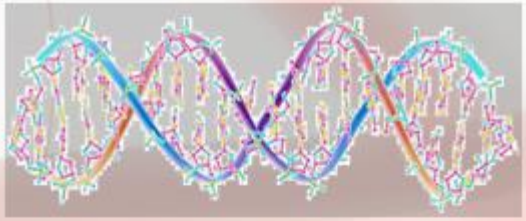


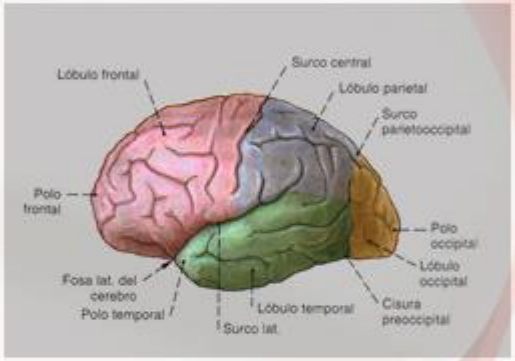

Lóbulo frontal
Surco central
Lóbulo parietal
Surco parietooccipital
Polo frontal
Polo occipital
Lóbulo occipital
Fosa lat. del cerebro
Polo temporal
Lóbulo temporal
Surco lat.
Cisura preoccipital

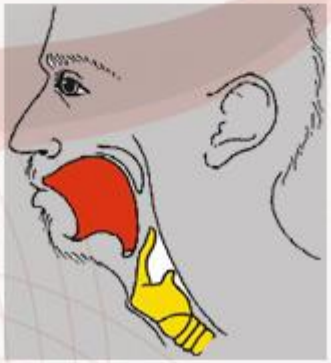


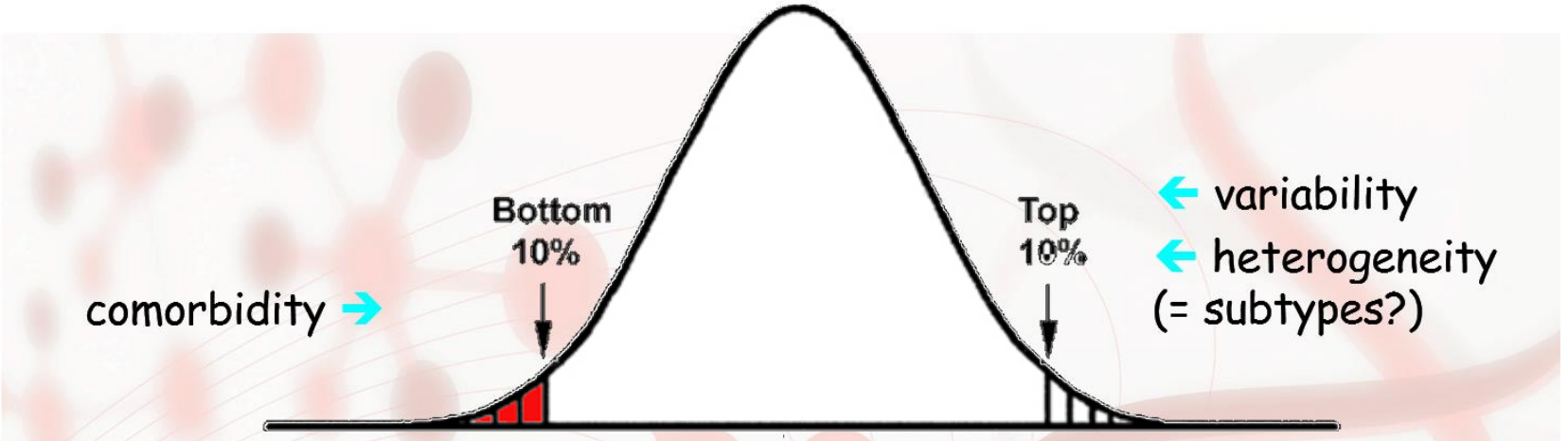

Symptoms
Causes

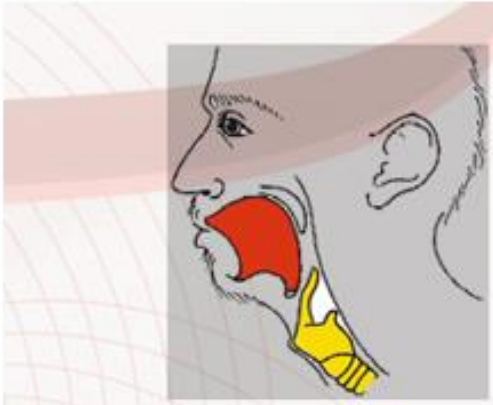

A
B
C
D

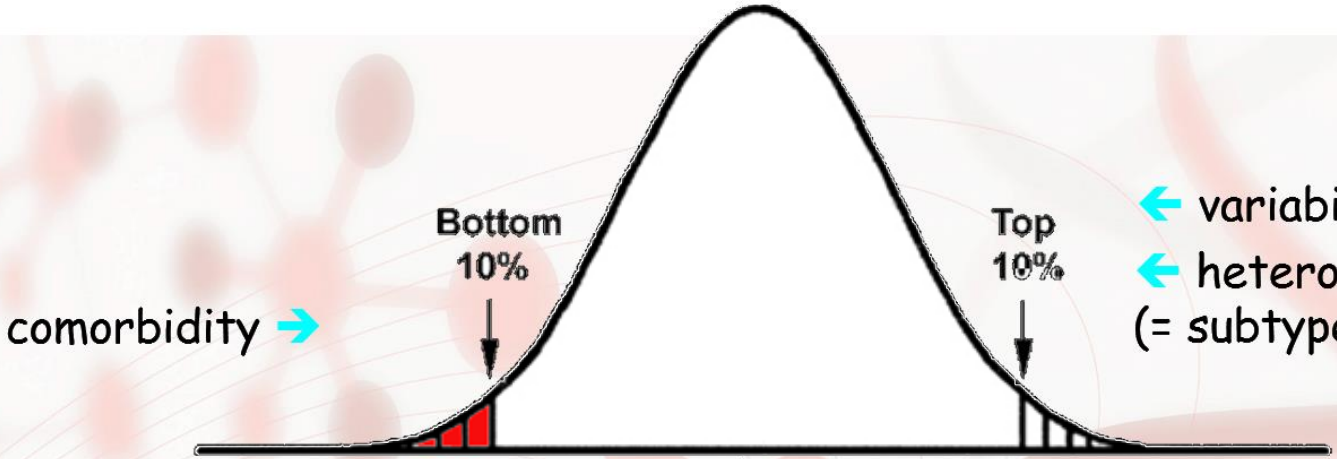



Lóbulo frontal
Surco central
Lóbulo parietal
Surco parietooccipital
Polo frontal
Polo occipital
Lóbulo occipital
Fosa lat. del cerebro
Polo temporal
Lóbulo temporal
Surco lat.
Cisura preoccipital




Bottom
10%
Top
10%
comorbidity
variability
heterogeneity
(= subtypes?)
The same symptoms are found in different disorders
Symptoms changes from one subject to another



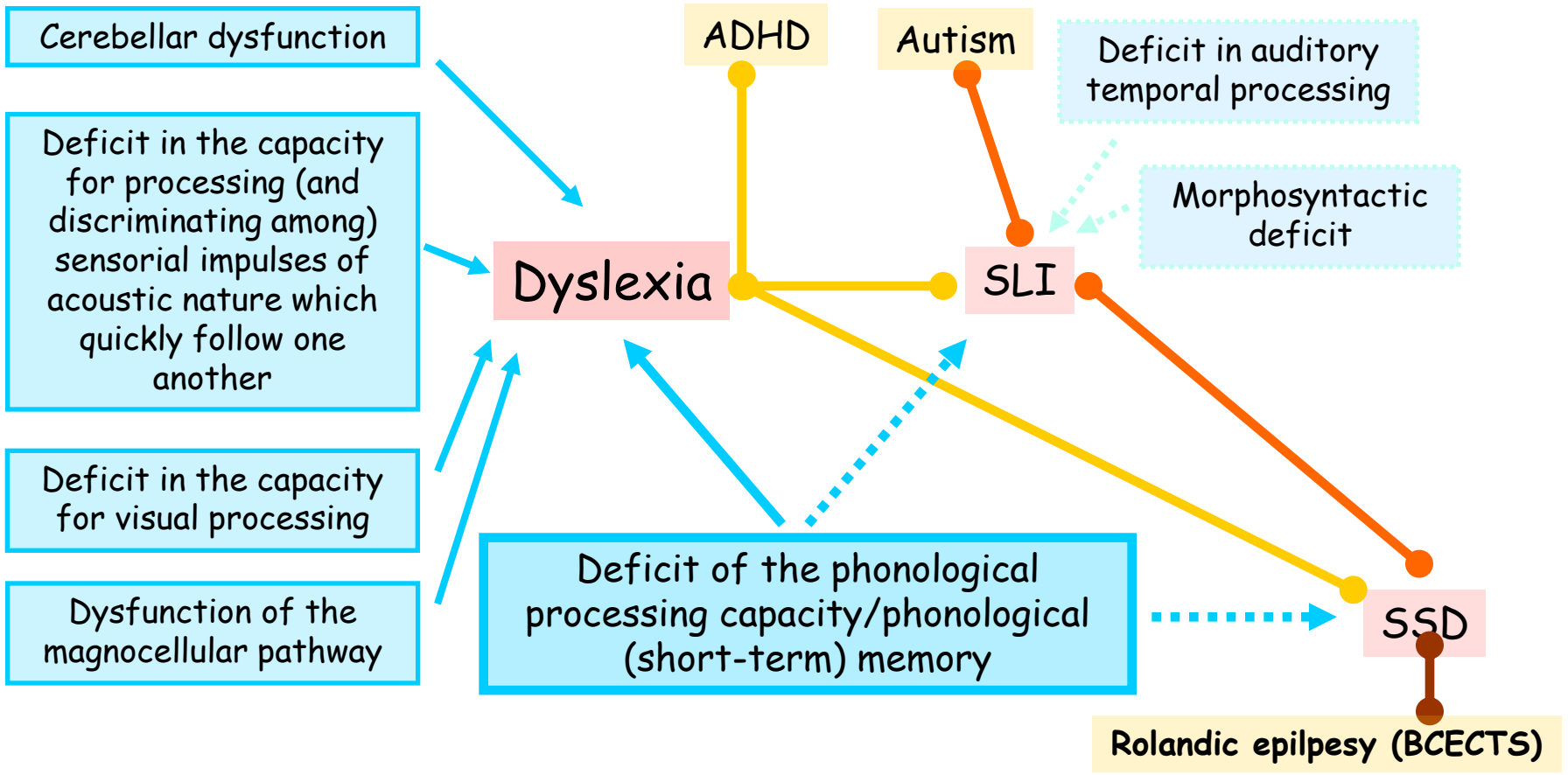


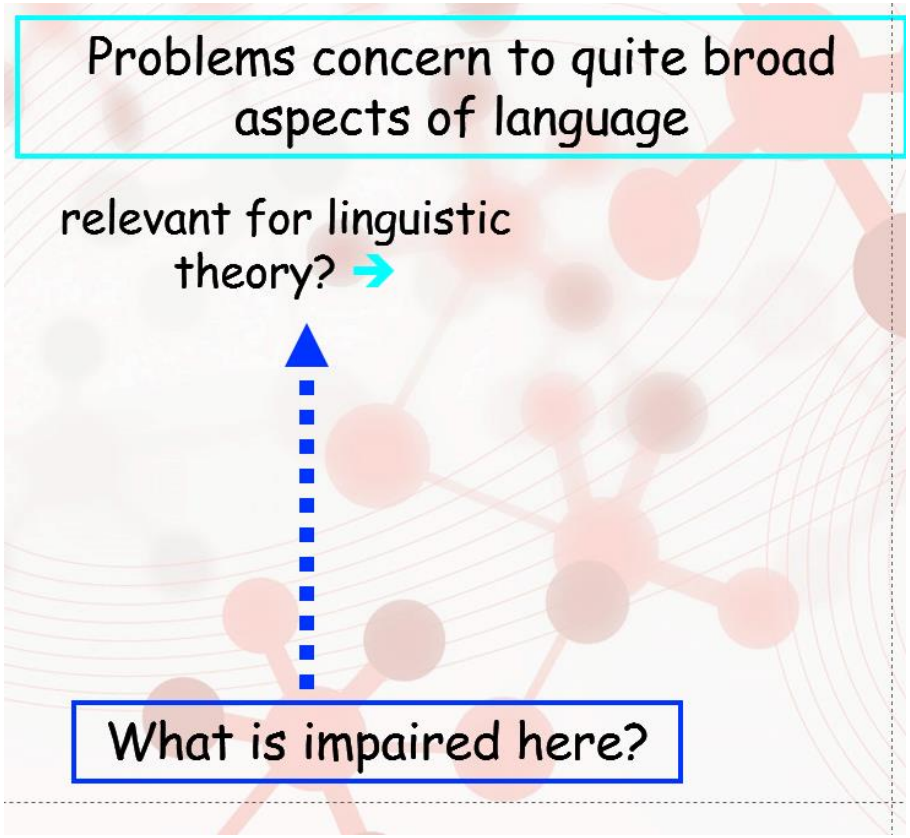
The same symptoms are found in different disorders

Symptoms changes from one subject to another

One (broad) cognitive deficit causing different disorders?

Different deficits contributing to the same disorder?


Cerebellar dysfunction
Deficit in the capacity for processing (and discriminating among) sensorial impulses of acoustic nature which quickly follow one another
Deficit in the capacity for visual processing
Dysfunction of the magnocellular pathway
ADHD
Autism
Deficit in auditory temporal processing
Morphosyntactic deficit
Dyslexia
SLI
Deficit of the phonological processing capacity/phonological (short-term) memory
SSD
Rolandic epilpesy (BCECTS)
comorbid disorders
underlying deficit


Problems concern to quite broad aspects of language
relevant for linguistic theory?
What is impaired here?

SLI
Research Group

Rapin and Allen (1983)[20]

Phonologic syntactic syndrome (with or without oromotor dysfunction)
Severe expressive syndrome with good comprehension
Verbal auditory agnosia (phonetic decoding deficit)
Syntactic-pragmatic syndrome
Semantic-pragmatic syndrome without autism ("cocktail party" speech)
Mute autistic syndrome
Autistic syndrome with echolalia

Problems concern to quite broad aspects of language

relevant for linguistic theory? →

What is impaired here?

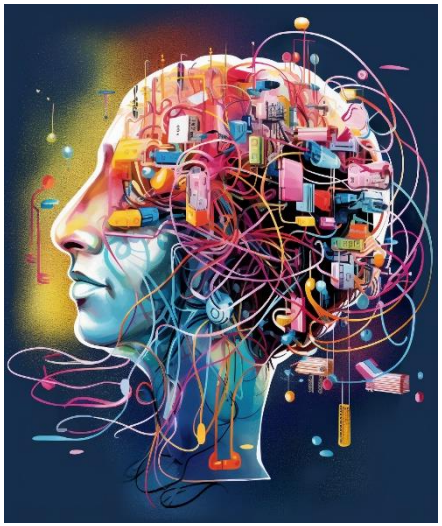
Developmental Language Disorder (DLD)

Problems concern to quite broad aspects of language

relevant for linguistic theory? →

What is impaired here?

Developmental Language Disorder (DLD)



Problems concern to quite broad aspects of language

relevant for linguistic theory? →

What is impaired here?

Symptoms change during the life span

Development is worth considering!

Environment too!

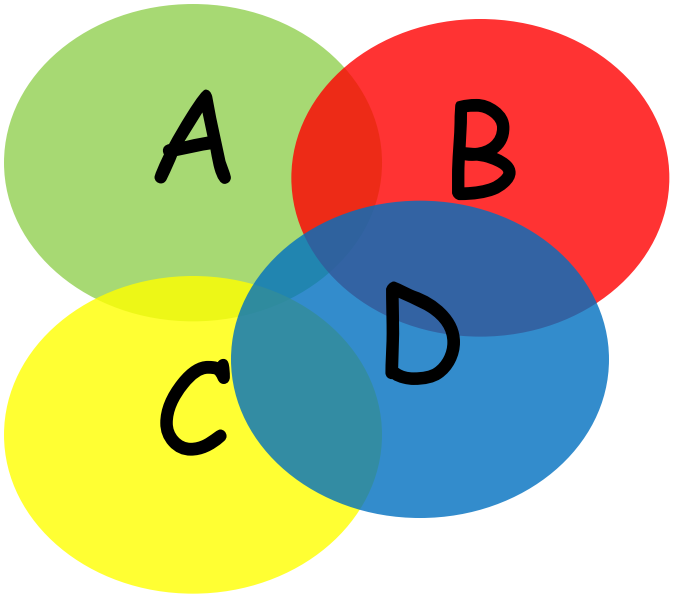
"To understand developmental outcomes, it is vital to identify full developmental trajectories, to assess how progressive change occurs from infancy onwards, and how parts of the developing system may interact with other parts differently at different times across ontogenesis"

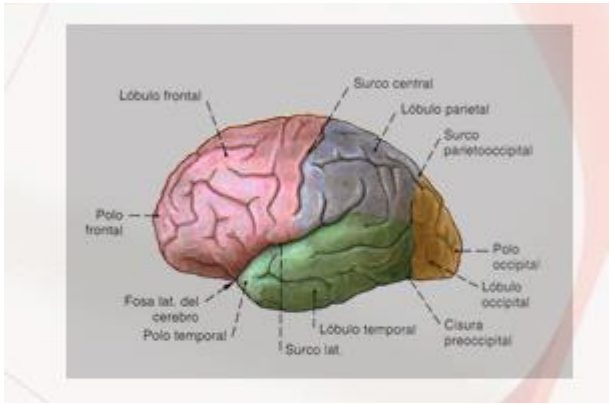
Karmiloff-Smith (2009: 58)

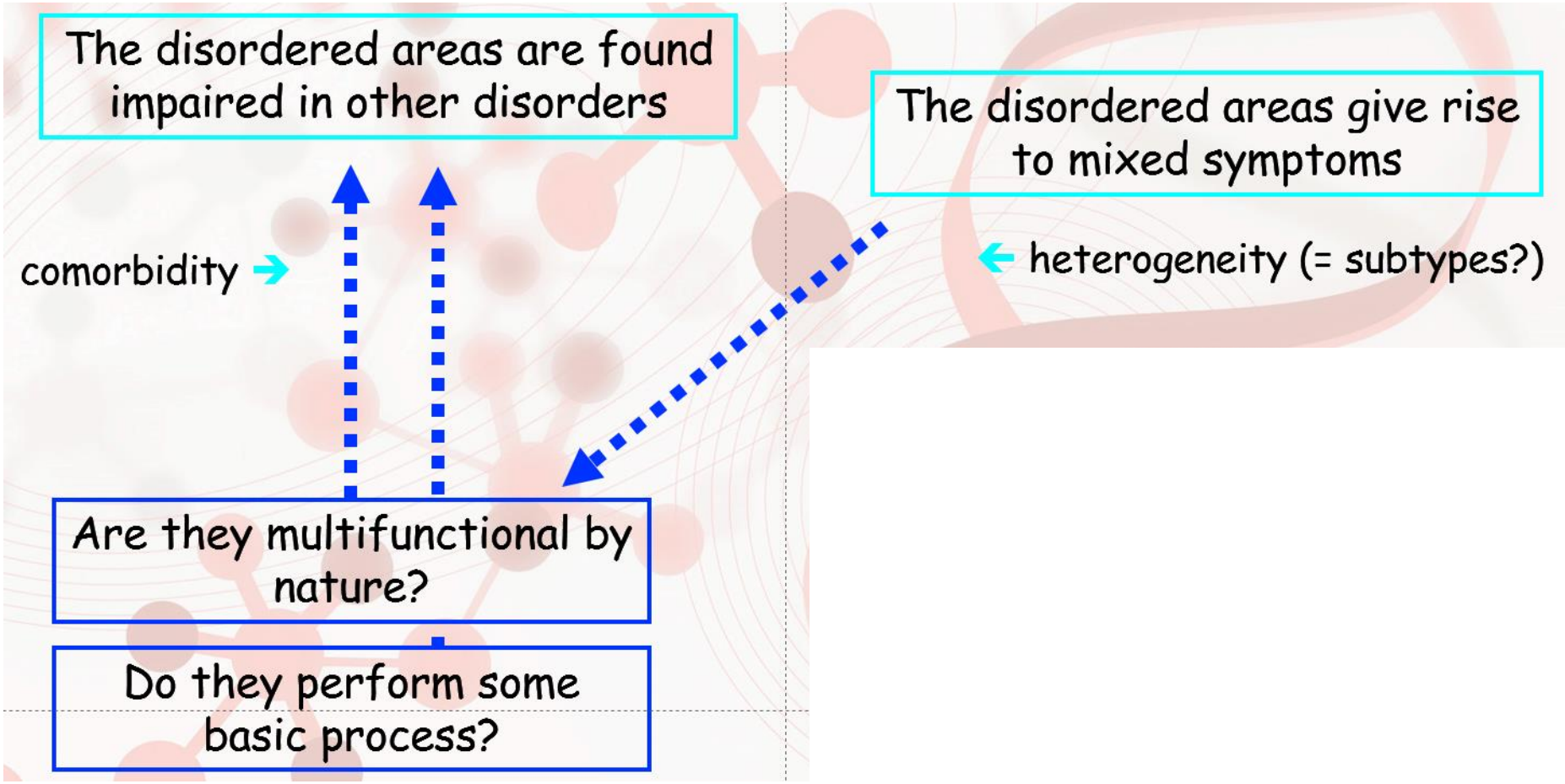
"To understand developmental outcomes, it is vital to identify full developmental trajectories, to assess how progressive change occurs from infancy onwards, and how parts of the developing system may interact with other parts differently at different times across ontogenesis"

Karmiloff-Smith (2009: 58)

➔ Disordered systems are adaptative ← compensations


A
B
C
D


Lóbulo frontal
Surco central
Lóbulo parietal
Surco parietooccipital
Polo frontal
Polo occipital
Lóbulo occipital
Fosa lat. del cerebro
Polo temporal
Lóbulo temporal
Surco lat.
Cisura preoccipital


The disordered areas are found impaired in other disorders
The disordered areas give rise to mixed symptoms
comorbidity
heterogeneity (= subtypes?)
Are they multifunctional by nature?
Do they perform some basic process?

Ontological Incommensurability Problem (OIP):

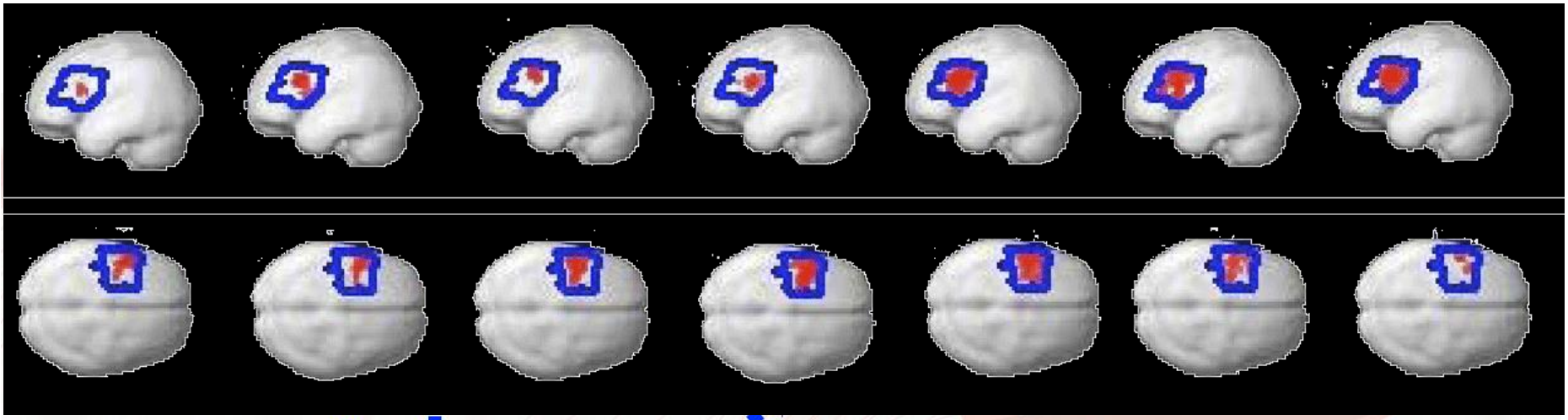
The units of linguistic computation and the units of neurological computation are incommensurable = the former cannot be reduced or matched up with the latter

principled relationships

Linguistics	Neuroscience
Fundamental elements of representation (at a given analytic level)	
distinctive feature	dendrites, spines
syllable	neuron
morpheme	cell-assembly/ensemble
noun phrase	population
clause	cortical column
Fundamental operations on primitives (at a given analytic level)	
concatenation	long-term potentiation (LTP)
linearization	receptive field
phrase-structure generation	oscillation
semantic composition	synchronization

Independent development

arbitrary relationships

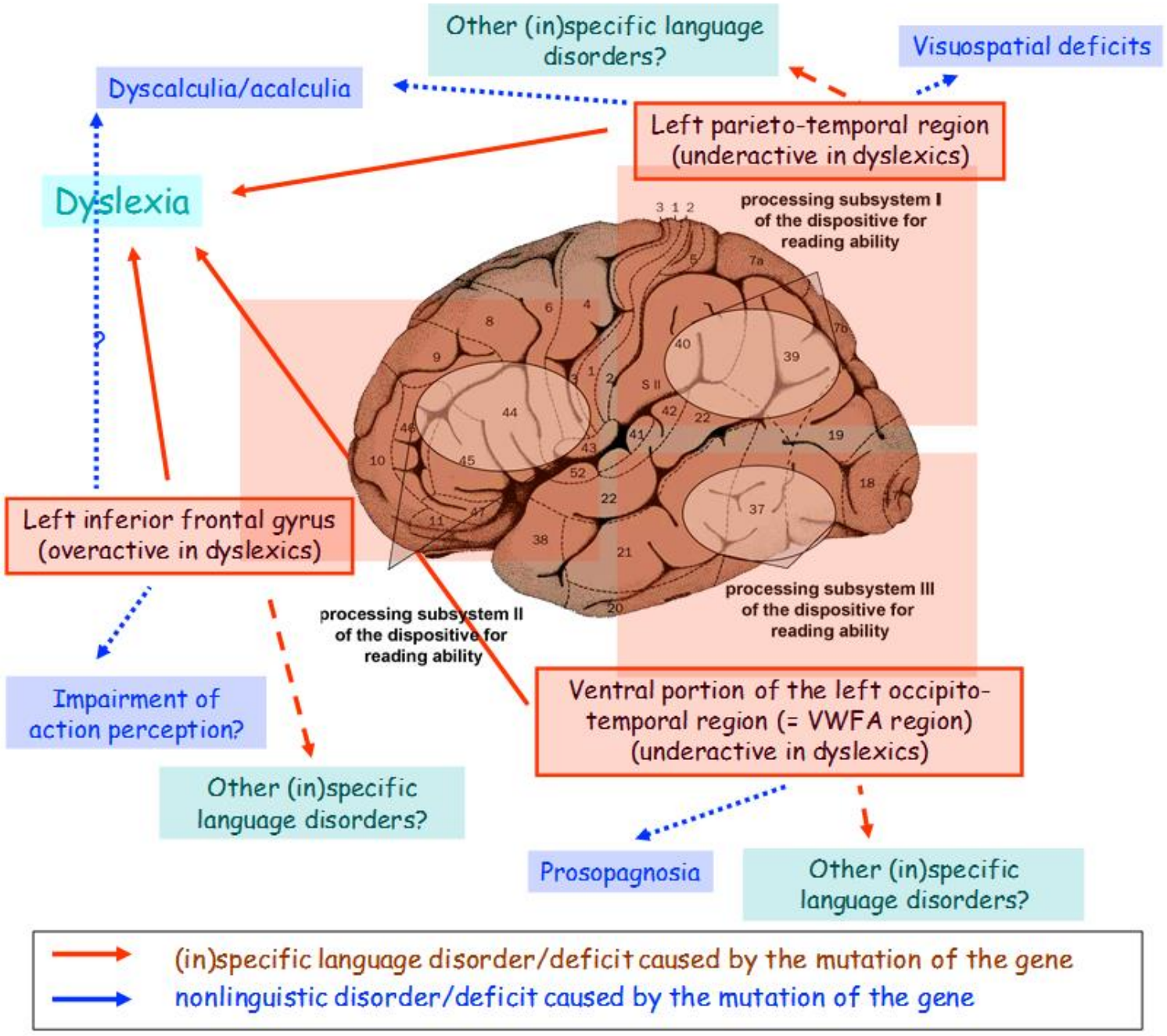


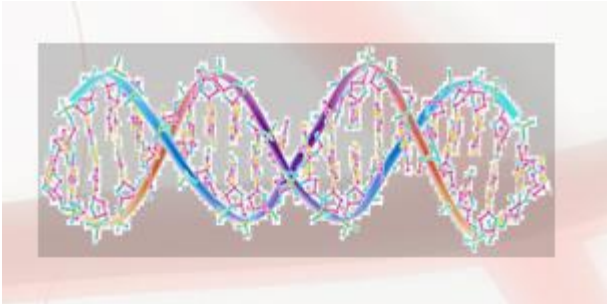
Are they multifunctional by nature?

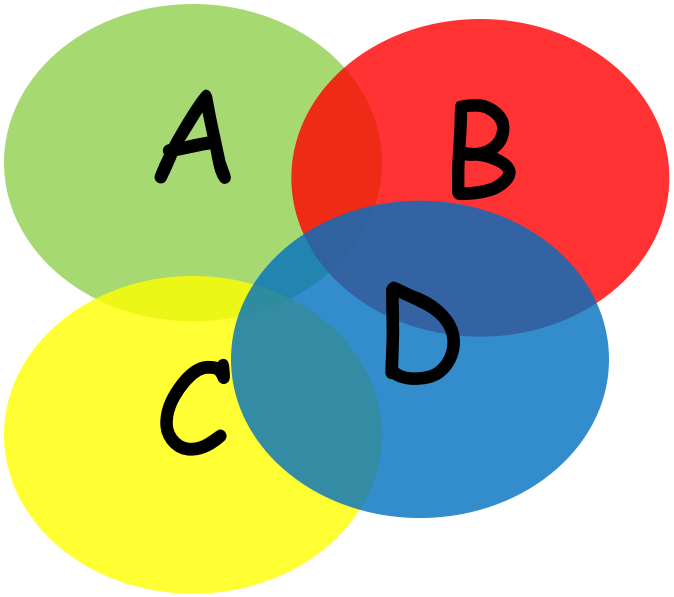
Do they perform some basic process?

The boundaries of the involved areas are different from one subject to another

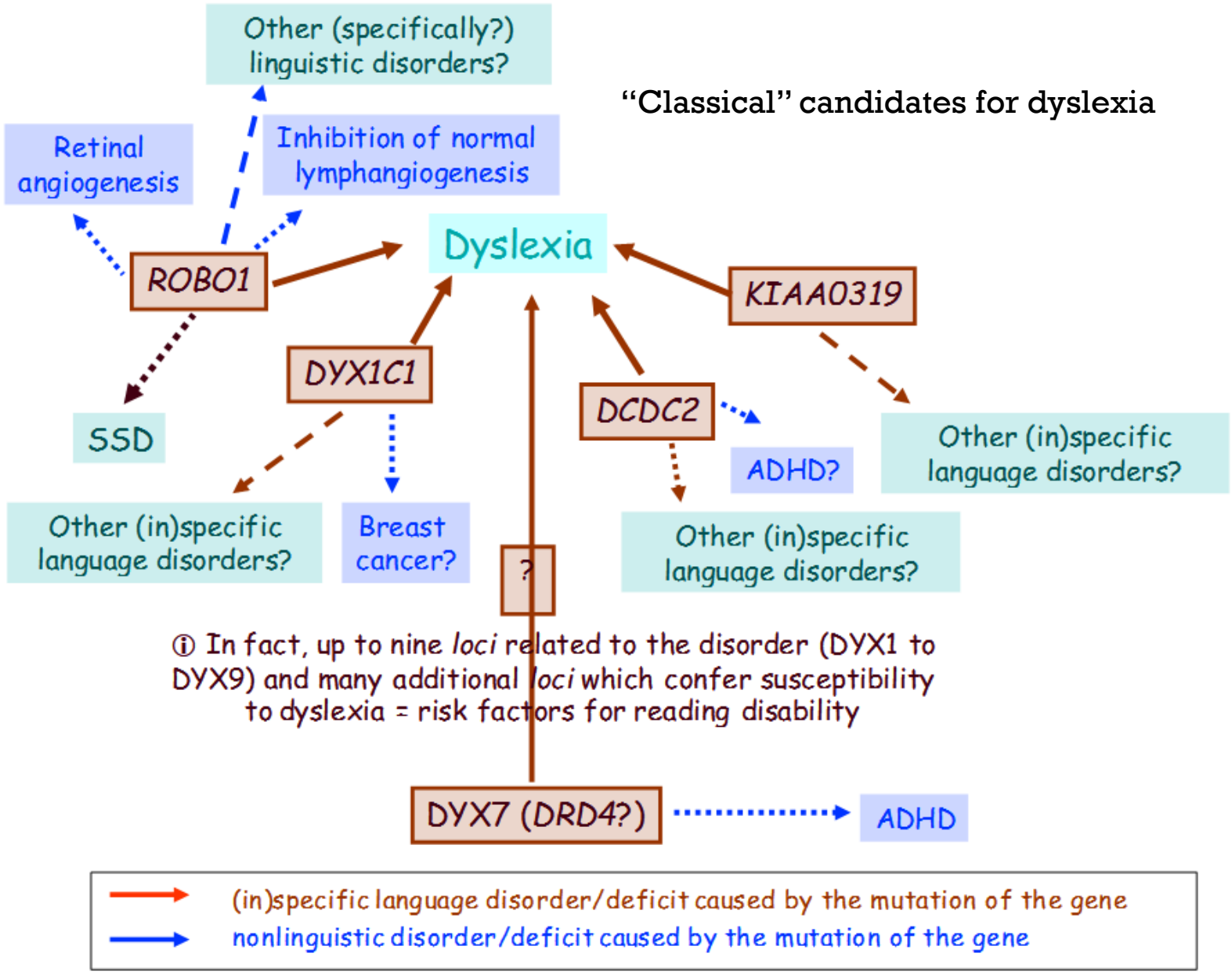
← variability

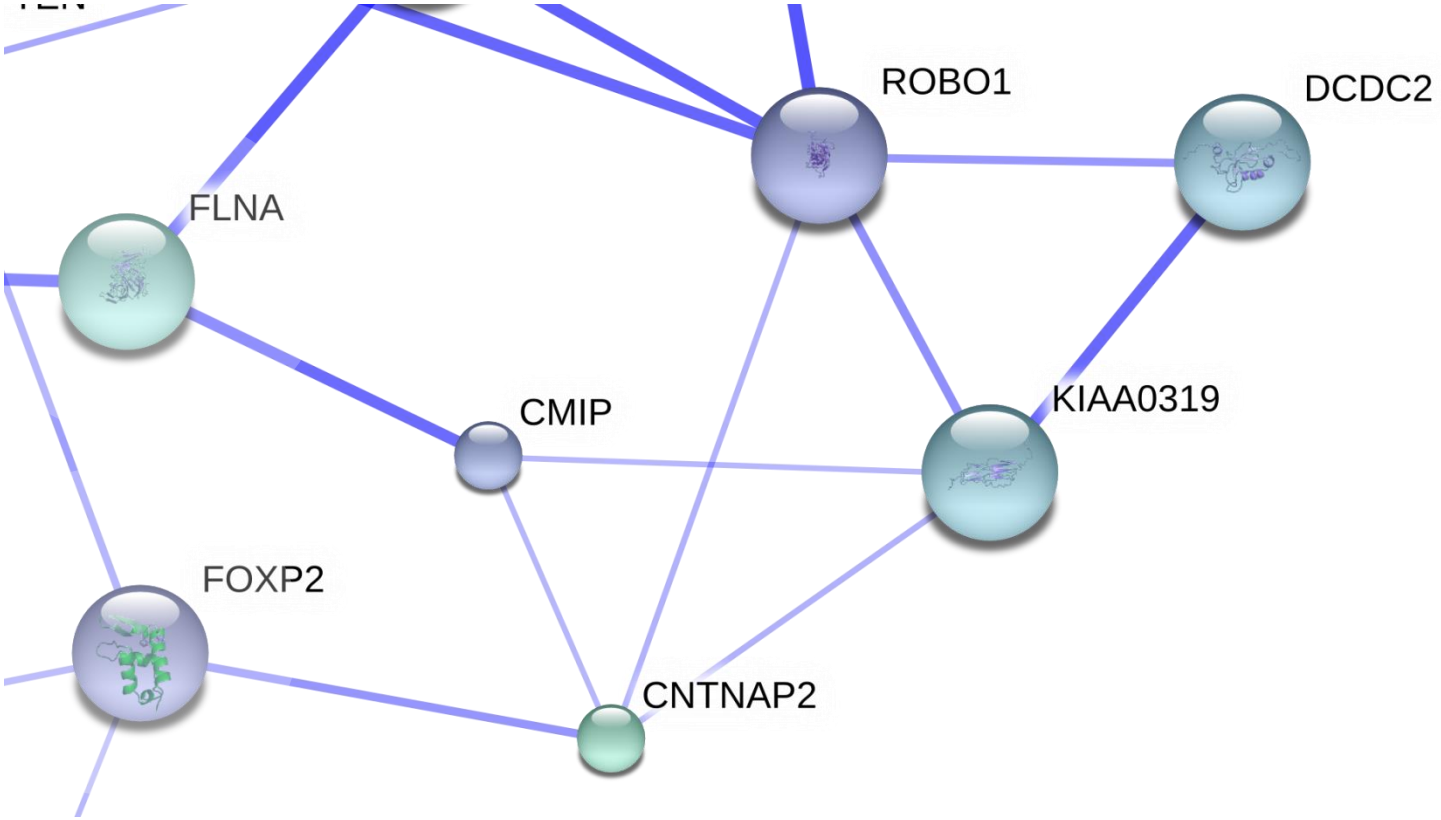

Other (in)specific language disorders?
Visuospatial deficits
Dyscalculia/acalculia
Left parieto-temporal region (underactive in dyslexics)
Dyslexia
processing subsystem I of the dispositive for reading ability
Left inferior frontal gyrus (overactive in dyslexics)
processing subsystem II of the dispositive for reading ability
processing subsystem III of the dispositive for reading ability
Impairment of action perception?
Other (in)specific language disorders?
Ventral portion of the left occipito-temporal region (= VWFA region) (underactive in dyslexics)
Prosopagnosia
Other (in)specific language disorders?
(in)specific language disorder/deficit caused by the mutation of the gene
nonlinguistic disorder/deficit caused by the mutation of the gene



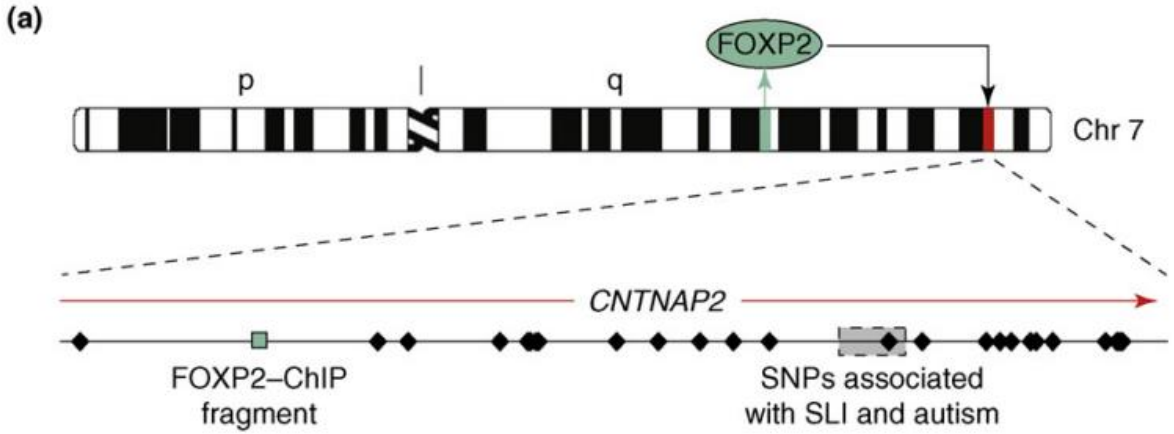

A
B
C
D

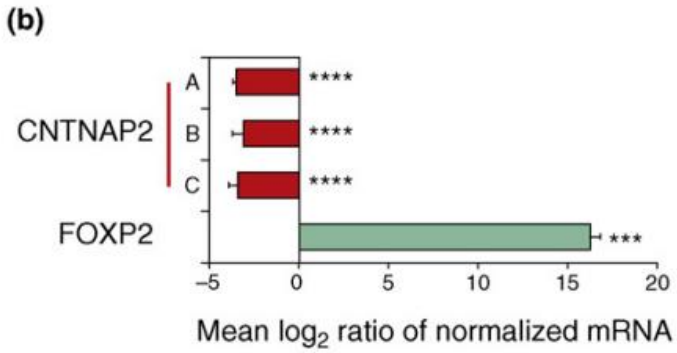
- polygenism
- polimorphism ← neurotypical population
- variable penetrance
- phenocopy
- pleiotropy
- different partners → different disorders

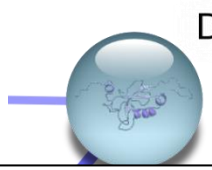

"Classical" candidates for dyslexia
Other (specifically?) linguistic disorders?
Retinal angiogenesis
Inhibition of normal lymphangiogenesis
Dyslexia
ROBO1
KIAA0319
DYX1C1
DCDC2
SSD
ADHD?
Other (in)specific language disorders?
Other (in)specific language disorders?
Breast cancer?
?
Other (in)specific language disorders?
ⓘ In fact, up to nine loci related to the disorder (DYX1 to DYX9) and many additional loci which confer susceptibility to dyslexia = risk factors for reading disability
DYX7 (DRD4?)
ADHD
(in)specific language disorder/deficit caused by the mutation of the gene
nonlinguistic disorder/deficit caused by the mutation of the gene


ROBO1
DCDC2
FLNA
CMIP
KIAA0319
FOXP2
CNTNAP2

CNTNAP2 as a candidate for dyslexia







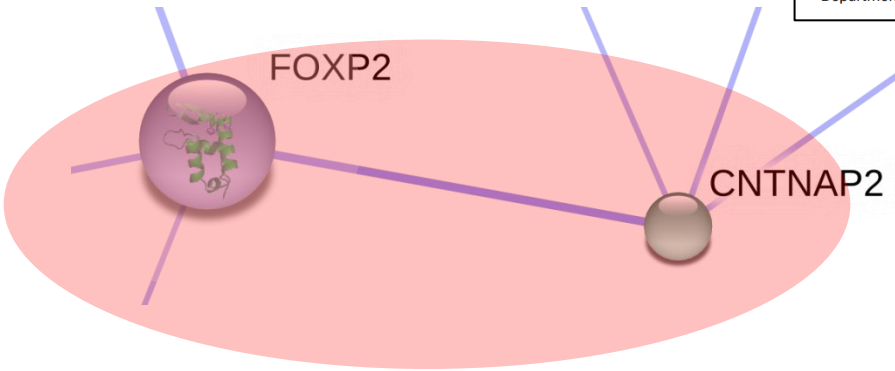
Review

Cell PRESS

FOXP2 as a molecular window into speech and language

Simon E. Fisher[1] and Constance Scharff[2]

[1] Wellcome Trust Centre for Human Genetics, University of Oxford, Roosevelt Drive, Oxford, OX3 7BN, UK
[2] Department of Animal Behavior, Institute of Biology, Freie Universität, Berlin, Germany



CNTNAP2 as a candidate for dyslexia

- Psycomotor delay ***
- Epilepsy ***
- ASD **
- Other NPsy
- ID moderate to profound *
- Hyporeflexia *
- Failure to thrive
- Comorbidities
- Dysmorphysms
- Language impairment *

Human Genetics (2023) 142:909–925
https://doi.org/10.1007/s00439-023-02552-2

ORIGINAL INVESTIGATION

Check for updates

Genotype–phenotype correlation in contactin-associated protein-like 2 (*CNTNAP-2*) developmental disorder

Gianluca D'Onofrio[1] · Andrea Accogli[2,3] · Mariasavina Severino[4] · Haluk Caliskan[5] · Tomislav Kokotović[5,6] · Antonela Blazekovic[7] · Kristina Gotovac Jercic[7,8] · Silvana Markovic[9] · Tamara Zigman[10] · Krnjak Goran[11] · Nina Barišić[10] · Vlasta Duranovic[12] · Ana Ban[12] · Fran Borovecki[7,8] · Danijela Petković Ramadža[10] · Ivo Barić[10] · Walid Fazeli[13] · Peter Herkenrath[13] · Carla Marini[14] · Roberta Vittorini[15] · Vykuntaraju Gowda[16] · Arjan Bouman[17] · Clarissa Rocca[18] · Issam Azmi Alkhawaja[19] · Bibi Nazia Murtaza[20] · Malik Mujaddad Ur Rehman[21] · Chadi Al Alam[22] · Gisele Nader[22] · Maria Margherita Mancardi[23] · Thea Giacomini[23] · Siddharth Srivastava[24] · Javeria Raza Alvi[25] · Hoda Tomoum[26] · Sara Matricardi[27] · Michele Iacomino[28] · Antonella Riva[1] · Marcello Scala[1] · Francesca Madia[28] · Angela Pistorio[29] · Vincenzo Salpietro[17] · Carlo Minetti[1] · Jean-Baptiste Rivière[30,31] · Myriam Srour[32,33] · Stephanie Efthymiou[17] · Reza Maroofian[17] · Henry Houlden[17] · Sonja Catherine Vernes[34,35] · Federico Zara[1,27] · Pasquale Striano[1,36] · Vanja Nagy[5,6]

Received: 18 February 2023 / Accepted: 3 April 2023 / Published online: 14 May 2023

Normal population	Early communicative behaviour
	Early communicative behaviour
	Early communicative behaviour

CNTNAP2 as a candidate for dyslexia

European Journal of Human Genetics (2014) 22, 171–178

www.nature.com/ejhg

REVIEW

Shining a light on *CNTNAP2*: complex functions to complex disorders

Pedro Rodenas-Cuadrado[1], Joses Ho[1,2] and Sonja C Vernes*[,1,3]

Other (specifically?)

nature genetics

Article

https://doi.org/10.1038/s41588-022-01192-y

Discovery of 42 genome-wide significant loci associated with dyslexia

Received: 28 August 2021

Accepted: 23 August 2022

Published online: 20 October 2022

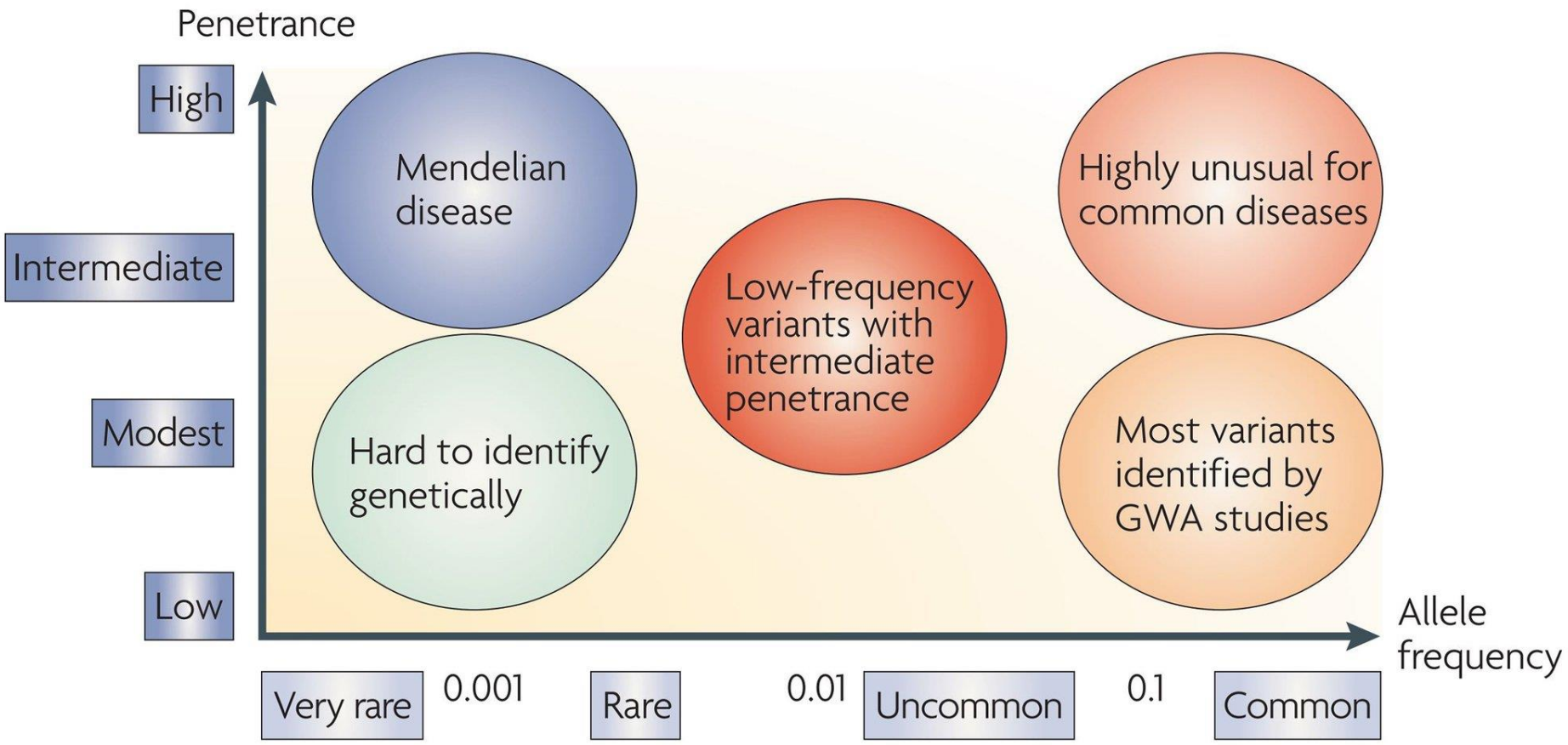
Check for updates

Catherine Doust[1], Pierre Fontanillas[2], Else Eising[3], Scott D. Gordon[4], Zhengjun Wang[5], Gökberk Alagöz[3], Barbara Molz[3], 23andMe Research Team*, Quantitative Trait Working Group of the GenLang Consortium*, Beate St Pourcain[3,6,7], Clyde Francks[3,6], Riccardo E. Marioni[8], Jingjing Zhao[5], Silvia Paracchini[9], Joel B. Talcott[10], Anthony P. Monaco[11], John F. Stein[12], Jeffrey R. Gruen[13], Richard K. Olson[14,15], Erik G. Willcutt[14,15], John C. DeFries[14,15], Bruce F. Pennington[16], Shelley D. Smith[17], Margaret J. Wright[18], Nicholas G. Martin[4], Adam Auton, Timothy C. Bates[1], Simon E. Fisher[3,6] and Michelle Luciano[1]

(in)specific language disorder/deficit caused by the mutation of the gene

nonlinguistic disorder/deficit caused by the mutation of the gene

Other (specifically?)



(in)specific language disorder/deficit caused by the mutation of the gene

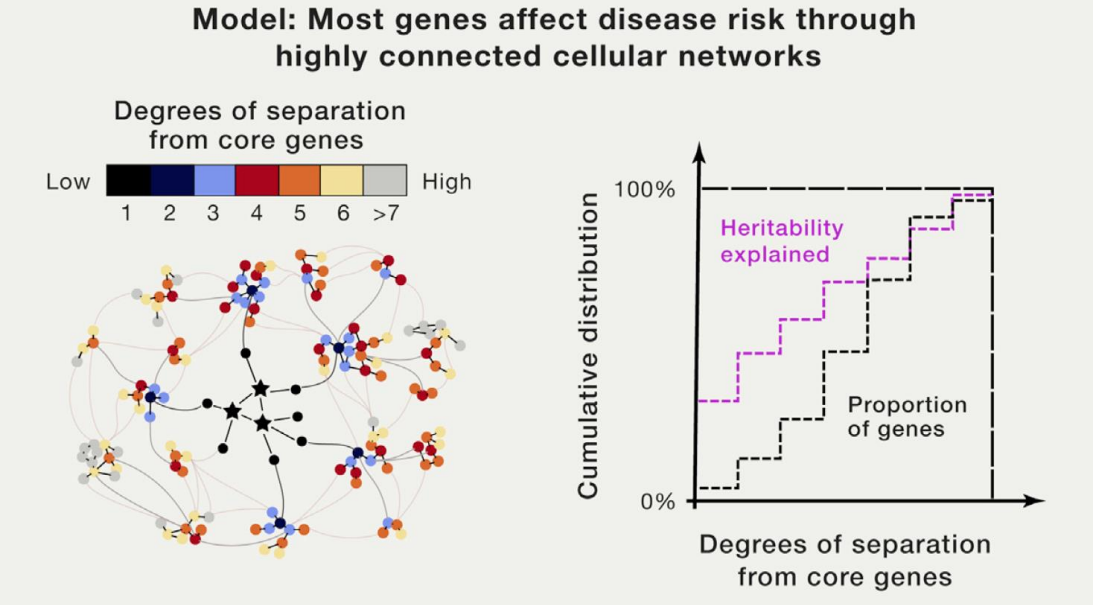
→ nonlinguistic disorder/deficit caused by the mutation of the gene

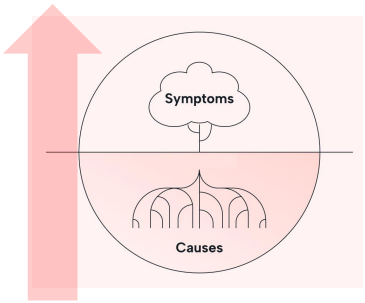
Leading Edge
Perspective

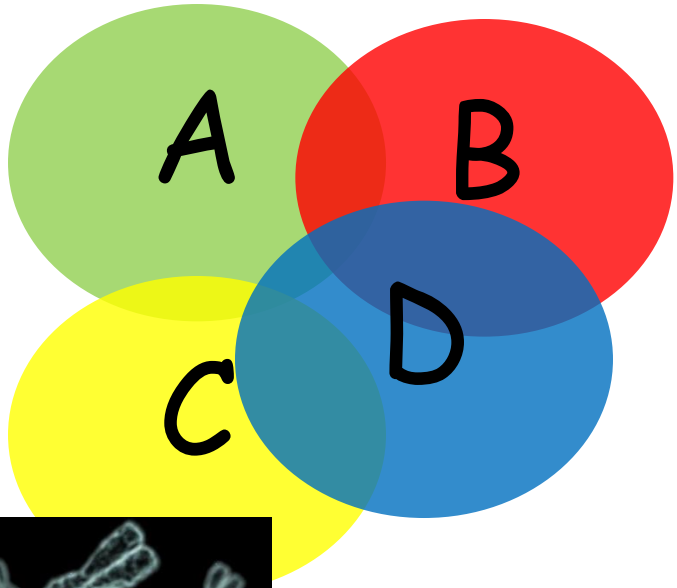
Cell

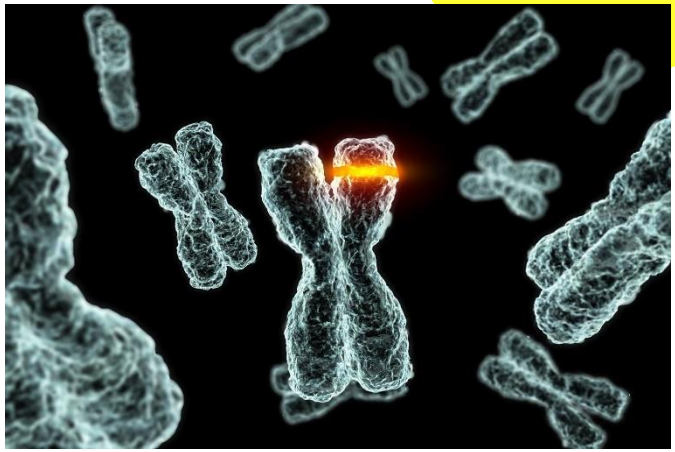
An Expanded View of Complex Traits: From Polygenic to Omnigenic

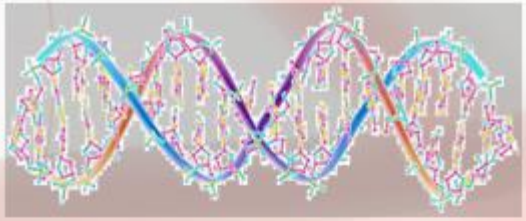
Evan A. Boyle,[1,*] Yang I. Li,[1,*] and Jonathan K. Pritchard[1,2,3,*]
[1]Department of Genetics
[2]Department of Biology
[3]Howard Hughes Medical Institute
Stanford University, Stanford, CA 94305, USA
*Correspondence: eaboyle@stanford.edu (E.A.B.), yangili@stanford.edu (Y.I.L.), pritch@stanford.edu (J.K.P.)
http://dx.doi.org/10.1016/j.cell.2017.05.038

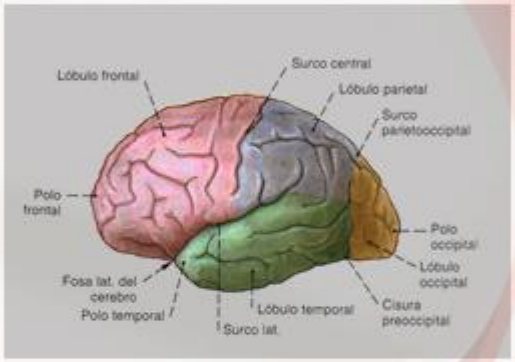


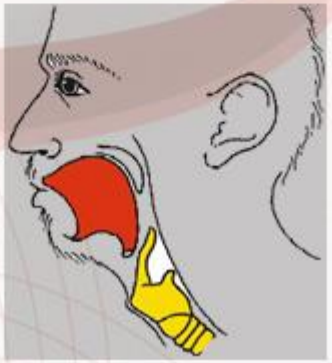

Symptoms
Causes

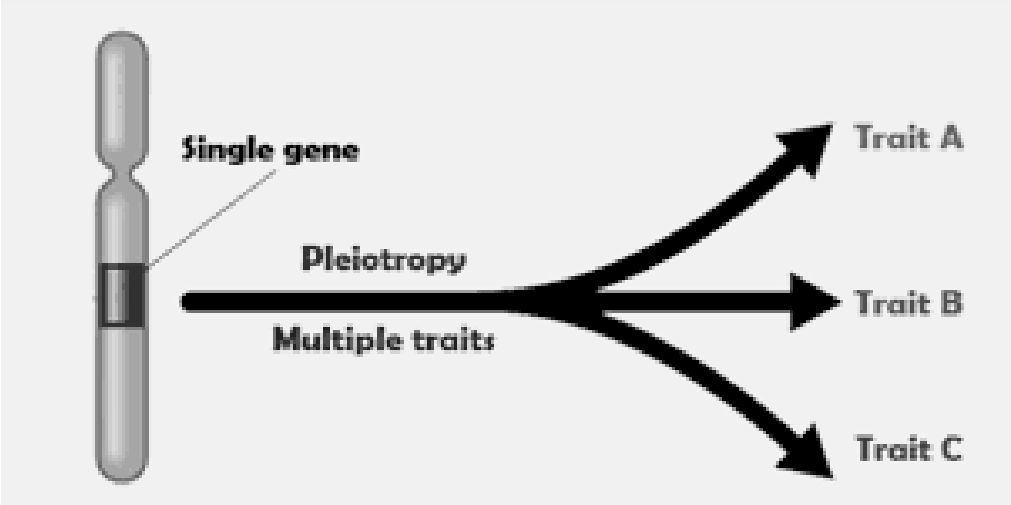

A
B
C
D






Lóbulo frontal
Surco central
Lóbulo parietal
Surco parietooccipital
Polo frontal
Polo occipital
Lóbulo occipital
Fosa lat. del cerebro
Polo temporal
Lóbulo temporal
Surco lat.
Cisura preoccipital





LETTERS

nature genetics

GRIN2A mutations in acquired epileptic aphasia and related childhood focal epilepsies and encephalopathies with speech and language dysfunction

Gaetan Lesca[1–4,24], Gabrielle Rudolf[4–6,24], Nadine Bruneau[4,7–9], Natalia Lozovaya[7–10], Audrey Labalme[1,4], Nadia Boutry-Kryza[3,4,11], Manal Salmi[4,7–9], Timur Tsintsadze[4,7–9], Laura Addis[12], Jacques Motte[4,13], Sukhvir Wright[14], Vera Tsintsadze[7–9], Anne Michel[15], Diane Doummar[16], Karine Lascelles[17], Lisa Strug[18,19], Patrick Waters[14], Julitta de Bellescize[4,20], Pascal Vrielynck[21], Anne de Saint Martin[4,22], Dorothee Ville[4,23], Philippe Ryvlin[3,4,20], Alexis Arzimanoglou[3,4,20], Edouard Hirsch[4–6], Angela Vincent[14], Deb Pal[12], Nail Burnashev[4,7–9], Damien Sanlaville[1–4] & Pierre Szepetowski[4,7–9]

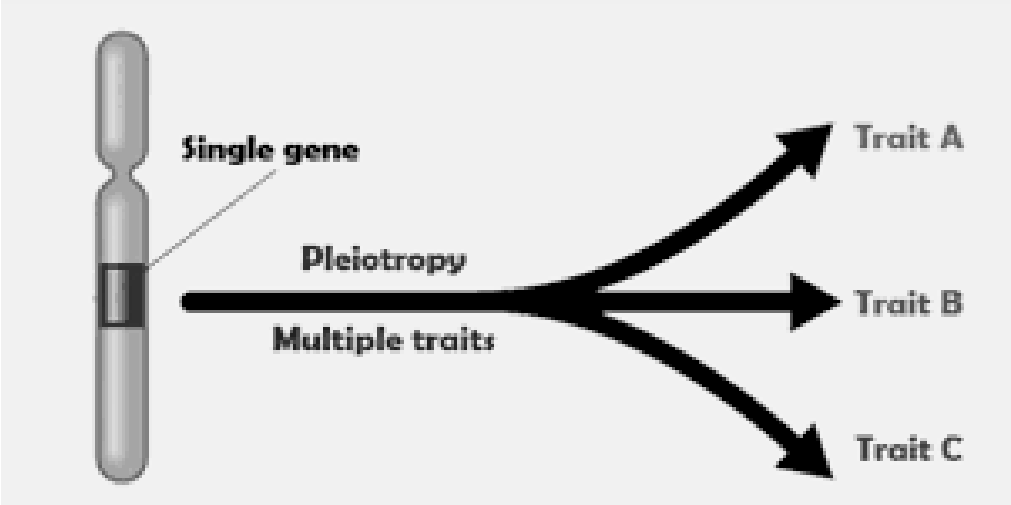
European Journal of Human Genetics (2014) 22, 171–178

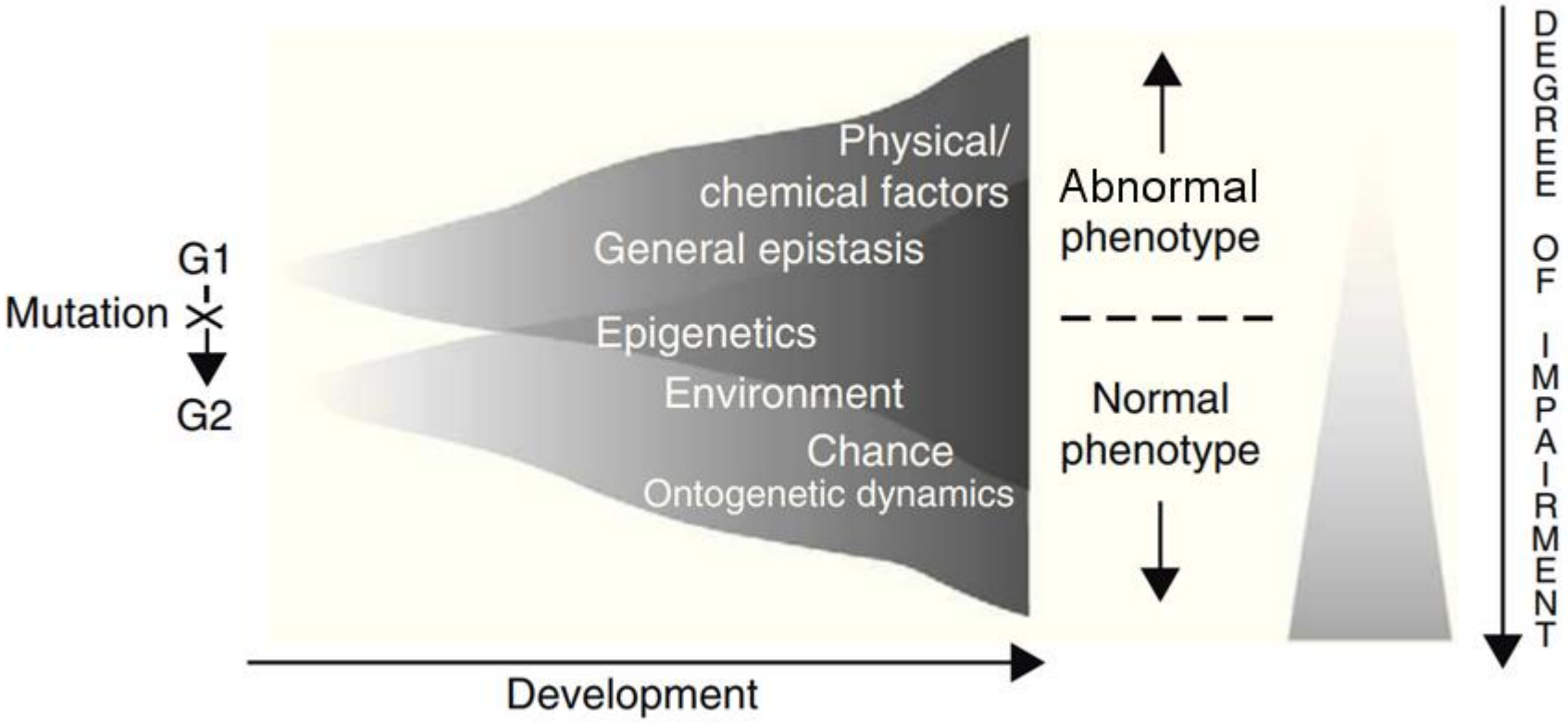
www.nature.com/ejhg

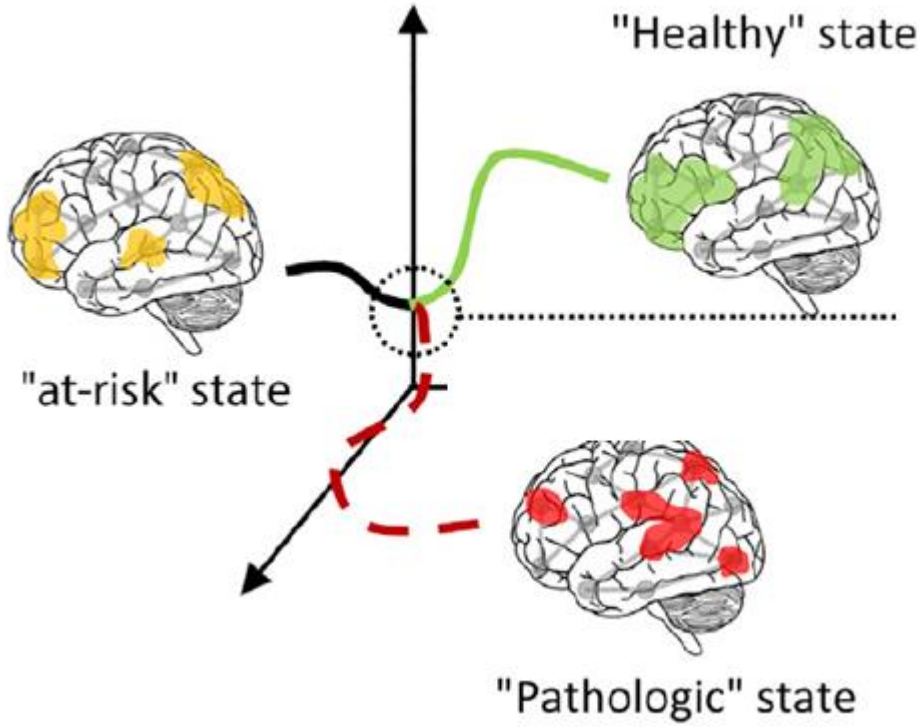
REVIEW

Shining a light on *CNTNAP2*: complex functions to complex disorders

Pedro Rodenas-Cuadrado[1], Joses Ho[1,2] and Sonja C Vernes*[,1,3]


Single gene
Pleiotropy
Multiple traits
Trait A
Trait B
Trait C


G1
Mutation
G2
Physical/
chemical factors
General epistasis
Epigenetics
Environment
Chance
Ontogenetic dynamics
Abnormal
phenotype
Normal
phenotype
Development
DEGREE OF IMPAIRMENT



CellPress

Neuron
Review

From Maps to Multi-dimensional Network Mechanisms of Mental Disorders

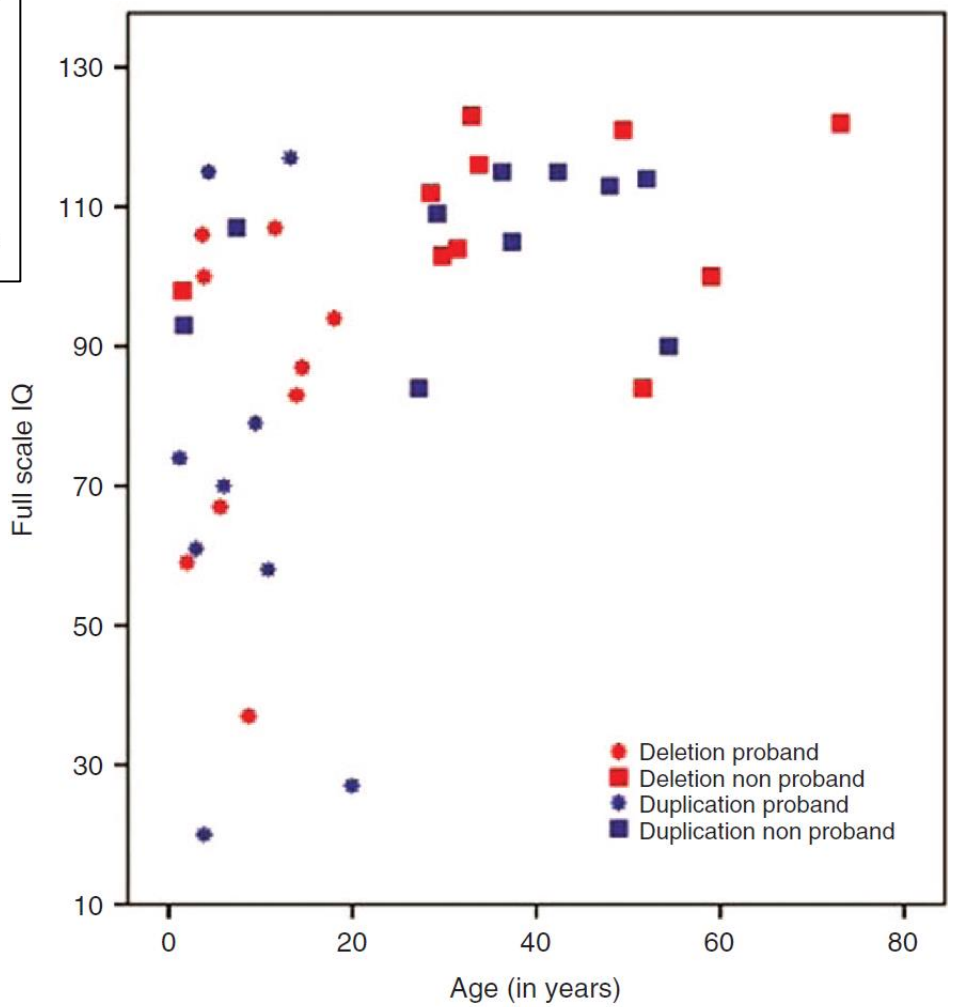
Urs Braun,[1] Axel Schaefer,[1] Richard F. Betzel,[2] Heike Tost,[1] Andreas Meyer-Lindenberg,[1] and Danielle S. Bassett[2,3,*]
[1]Central Institute of Mental Health, Medical Faculty Mannheim/University of Heidelberg, 68159 Mannheim, Germany
[2]Department of Bioengineering, University of Pennsylvania, Philadelphia, PA 19104, USA
[3]Department of Electrical and Systems Engineering, University of Pennsylvania, Philadelphia, PA 19104, USA
*Correspondence: dsb@seas.upenn.edu
https://doi.org/10.1016/j.neuron.2017.11.007

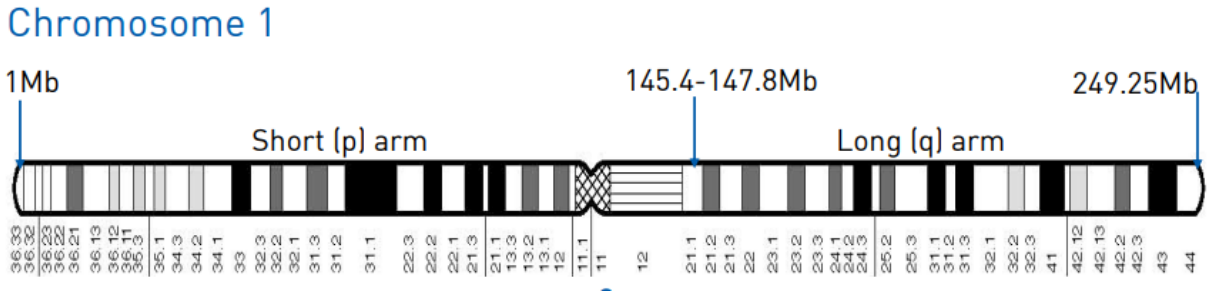
KEIO/1

ORIGINAL RESEARCH ARTICLE | Genetics inMedicine

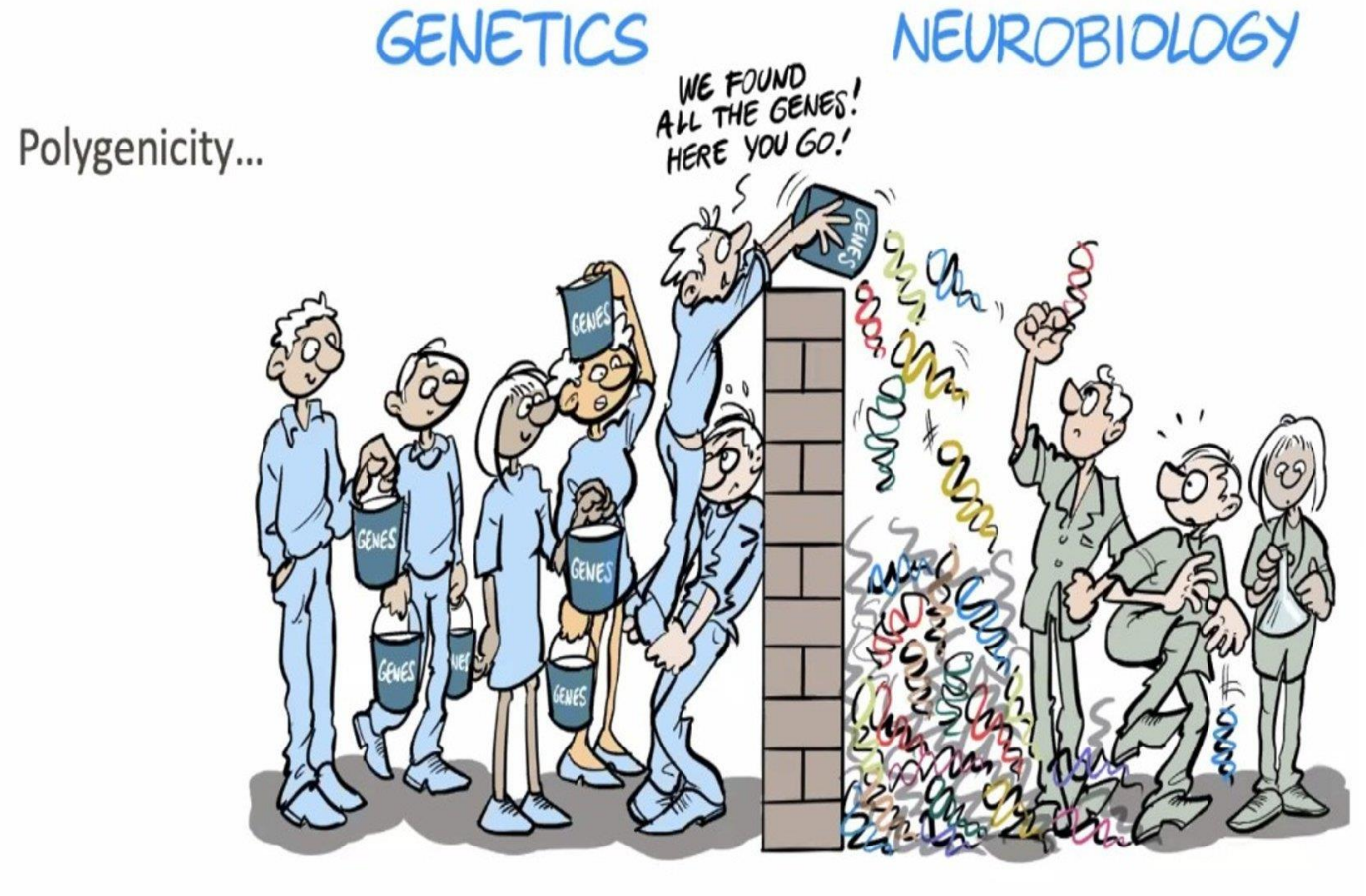
Clinical phenotype of the recurrent 1q21.1 copy-number variant

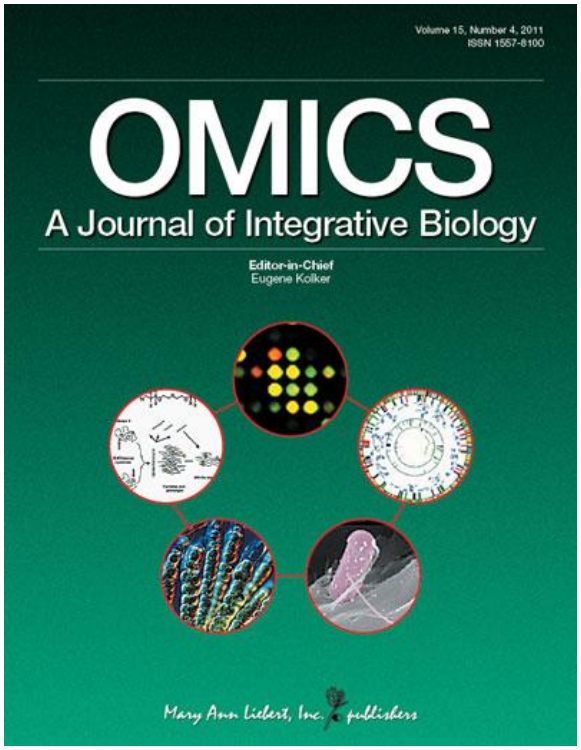
Raphael Bernier, PhD[1], Kyle J. Steinman, MD[2], Beau Reilly, PhD[3], Arianne Stevens Wallace, PhD[1], Elliott H. Sherr, MD[4], Nicholas Pojman, BS[4], Heather C. Mefford, MD, PhD[5], Jennifer Gerdts, PhD[1], Rachel Earl, BS[1], Ellen Hanson, PhD[6,7], Robin P. Goin-Kochel, PhD[8], Leandra Berry, PhD[8], Stephen Kanne, PhD[9], LeeAnne Green Snyder, PhD[6,10], Sarah Spence, MD[11], Melissa B. Ramocki, MD[12], David W. Evans, PhD[13], John E. Spiro, PhD[10], Christa L. Martin, PhD[13,14], David H. Ledbetter, PhD[13,14] and Wendy K. Chung, MD[15,16]; on behalf of the Simons VIP consortium.

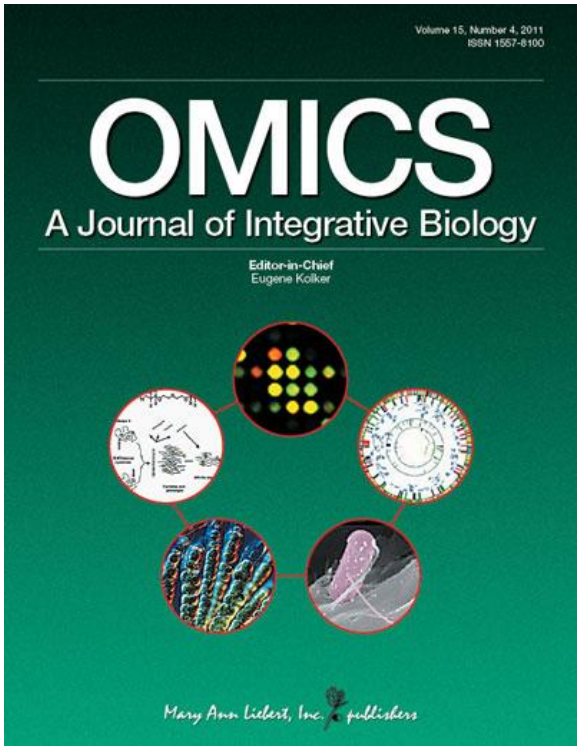




Polygenicity…




Volume 15, Number 4, 2011
ISSN 1557-8100
OMICS
A Journal of Integrative Biology
Editor-in-Chief
Eugene Kolker
Mary Ann Liebert, Inc. publishers



Language	Phenome
Cognitive functions	Cognome
Neural synchronization	Dynome
Neural wiring	Connectome
Neural populations and brain areas	Cytome
Codes for neural development and assembly	Toponome
Signalling pathways	Organome
Protein interactions	Interactome
Proteins	Proteome
RNAs	Transcriptome
Epigenetic modifications	Epigenome
Genes	Genome

KEIO/1

Neurobiology of Stress 12 (2020) 100223

Contents lists available at ScienceDirect

Neurobiology of Stress

journal homepage: www.elsevier.com/locate/ynstr

Dyslexia as an adaptation to cortico-limbic stress system reactivity

John R. Kershner

University of Toronto, Dept of Applied Psychology University of Toronto, Toronto, ON M5S 1A1, Canada

brain sciences

MDPI

Opinion

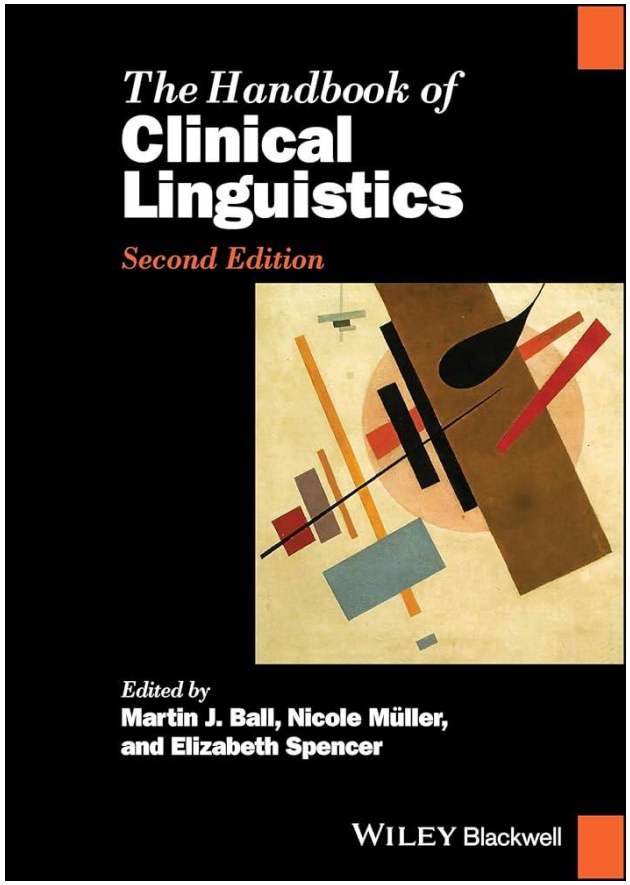
Developmental Dyslexia: Environment Matters

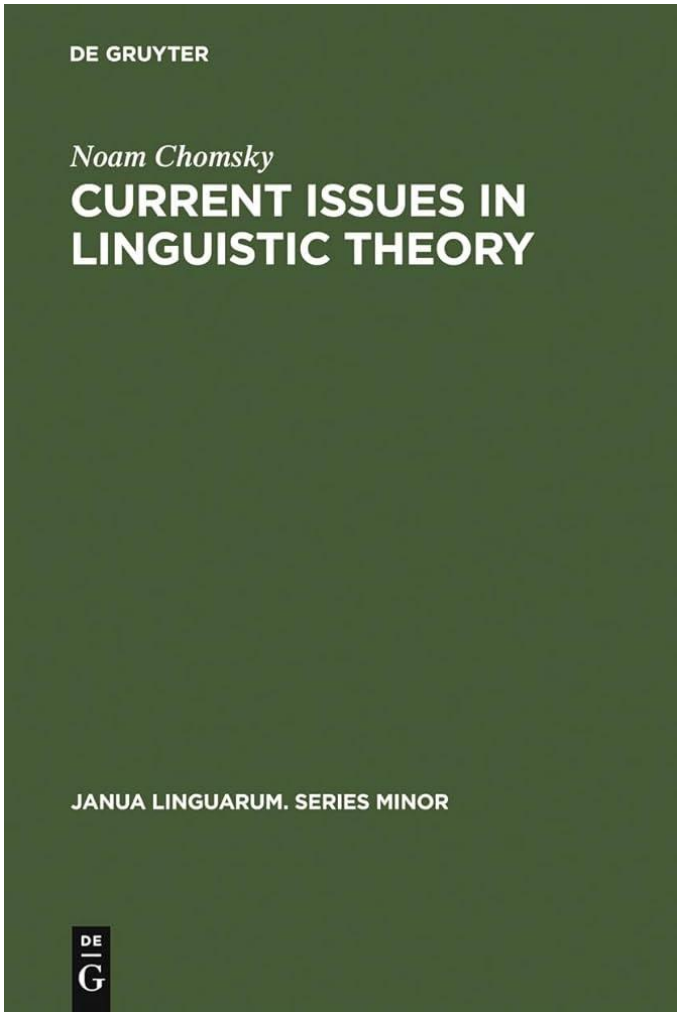
Daniela Theodoridou [1], Pavlos Christodoulides [2], Victoria Zakopoulou [3] and Maria Syrrou [1,*]

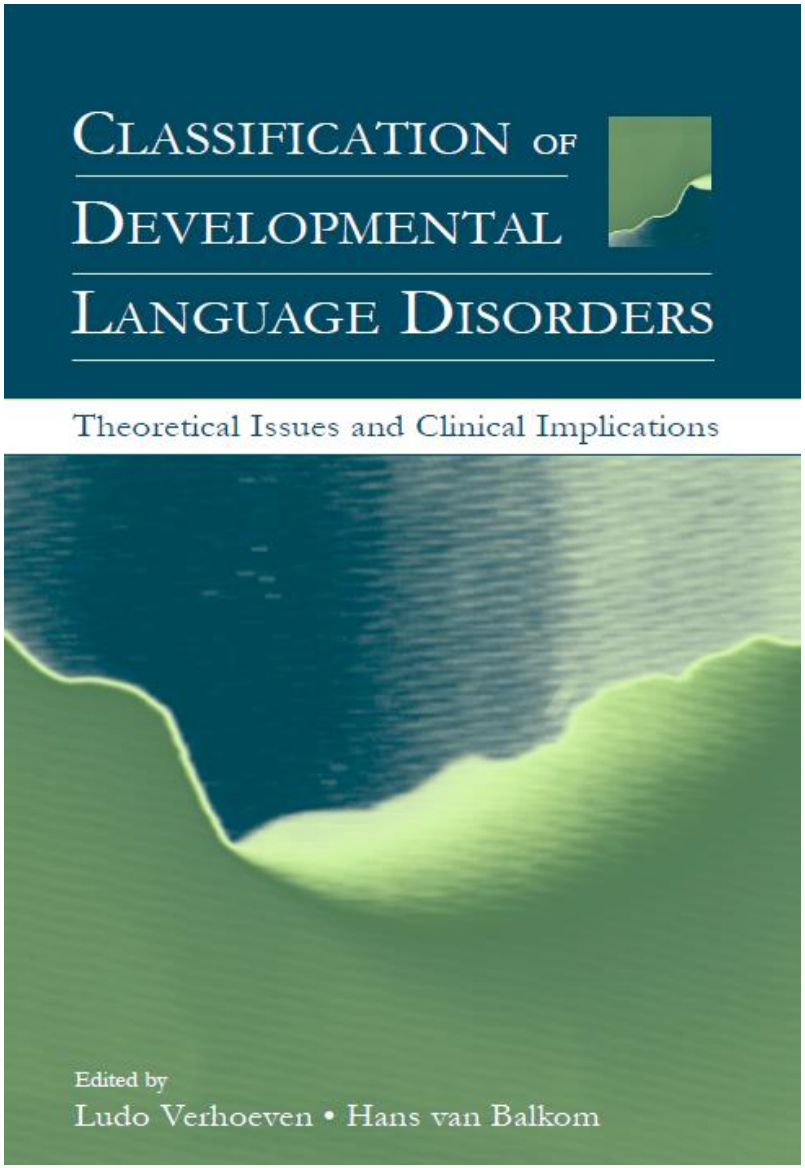
1 Laboratory of Biology, Faculty of Medicine, School of Health Sciences, University of Ioannina, 45110 Ioannina, Greece; danielatheodoridou@gmail.com
2 Laboratory of Physiology, Faculty of Medicine, School of Health Sciences, University of Ioannina, 45110 Ioannina, Greece; pchristo@uoi.gr
3 Department of Speech and Language Therapy, School of Health Sciences, University of Ioannina, 45110 Ioannina, Greece; vzakop@uoi.gr
* Correspondence: msyrrou@uoi.gr; Tel.: +30-2651007612

HOW I HAVE STRUCTURED MY TALK

1. Introduction
2. Clinical linguistics: struggling with symptoms and causes
3. Bridging genes to language (and vice versa)
4. Putting the focus on brain oscillations
5. Conclusions and future prospects


The Handbook of
Clinical
Linguistics
Second Edition
Edited by
Martin J. Ball, Nicole Müller,
and Elizabeth Spencer
WILEY Blackwell


DE GRUYTER
Noam Chomsky
CURRENT ISSUES IN
LINGUISTIC THEORY
JANUA LINGUARUM. SERIES MINOR
DE
G



If a linguistic analysis is correct, it identifies computations/representations that are computed [in real time] in the minds/brains of speakers

"To understand any developmental syndrome, it is essential to distinguish between the behavioral phenotype (based on scores from standardized tests of overt behavior) and the cognitive phenotype (based on in-depth analyses of the mental processes underlying the overt behavior)"

Karmiloff-Smith (2008: 693)

42 Research into Williams Syndrome: The State of the Art

ANNETTE KARMILOFF-SMITH

8 Developmental Language Disorders: Nosologic Considerations

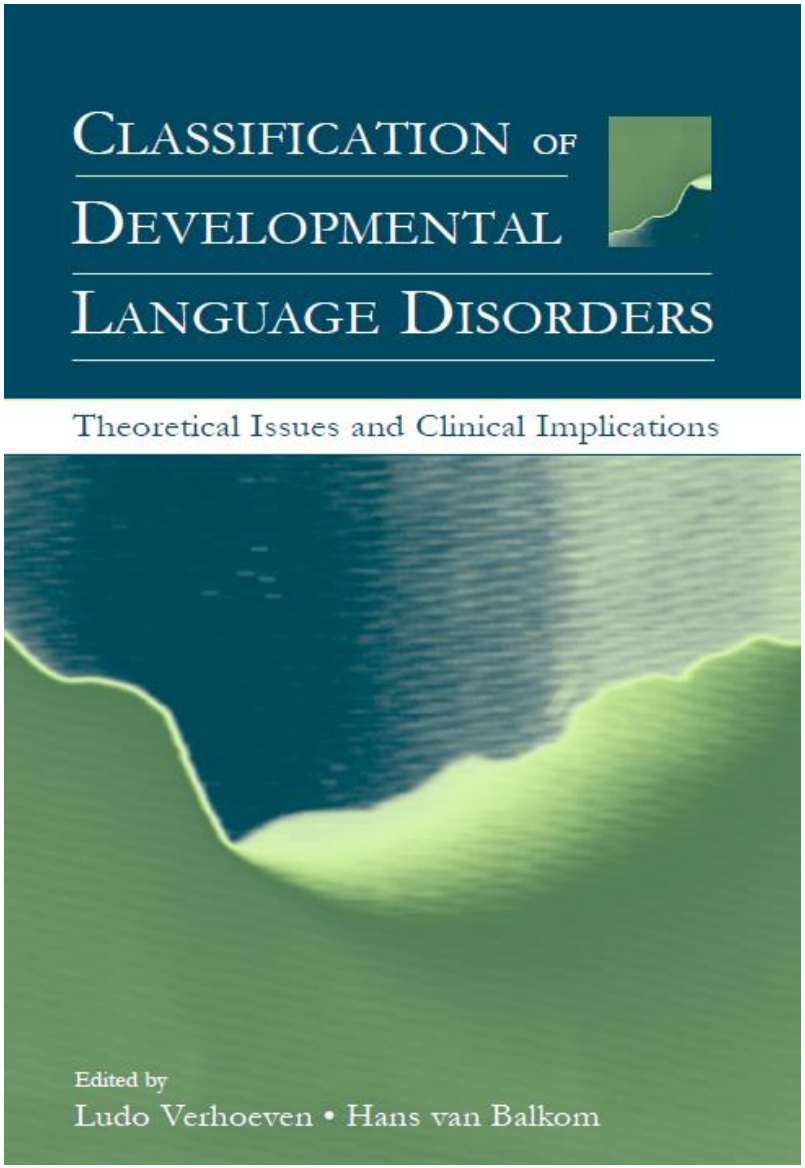
ISABELLE RAPIN
DORIS A. ALLEN

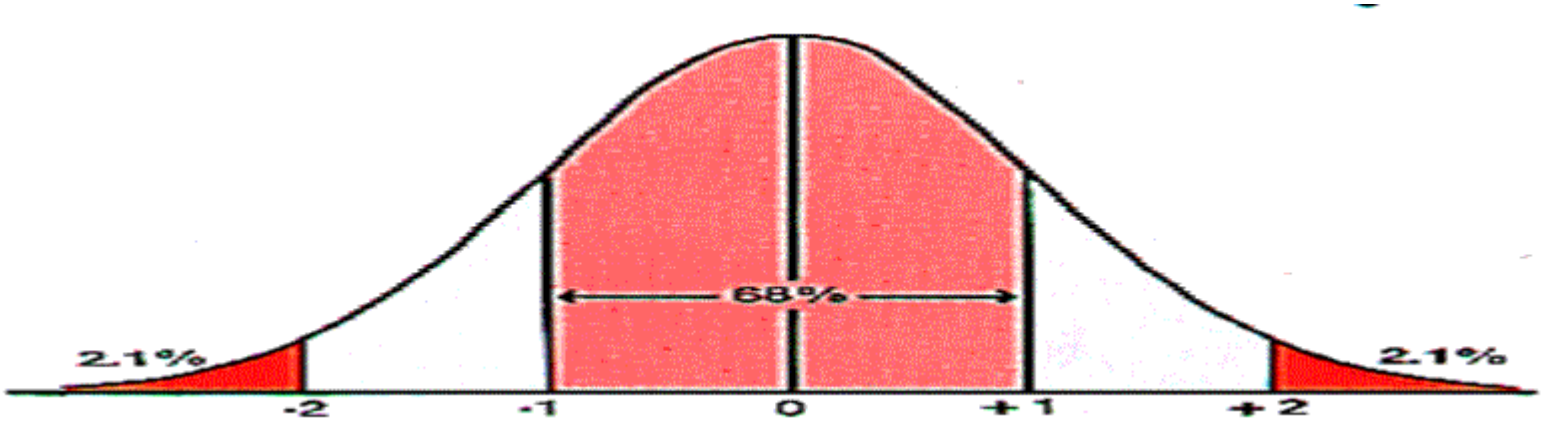
Preschool children who fail to develop effective speech at the expected age and whose behavior is often aberrant present a serious challenge to parents, educators, speech and language pathologists, and physicians. In fact, their problem is so poorly understood, even today, that there is no general agreement on a classification of disorders presenting with these symptoms. We have no firm evidence of their cause, we cannot predict a likely outcome with assurance, and we have few hard data on the effectiveness of our intervention programs.

This chapter is a preliminary attempt by a child neurologist and a developmental psycholinguist to bring some conceptual order to the problem of children with *developmental language disability* (DLD). This attempt is clinically based and does not have the rigor of systematically collected experimental data. It also lacks the underpinnings that could have been provided if neuropsychologic tests, EEGs, and computerized transaxial tomography (CT) scans had been available for all the children. But, as pointed out by Benton (1978), clinical and experimental methods are complementary, and clinical observation is often a necessary first step to later, more focused experimental studies.

NEUROPSYCHOLOGY OF
LANGUAGE, READING, AND SPELLING

ISBN 0-12-408680-8





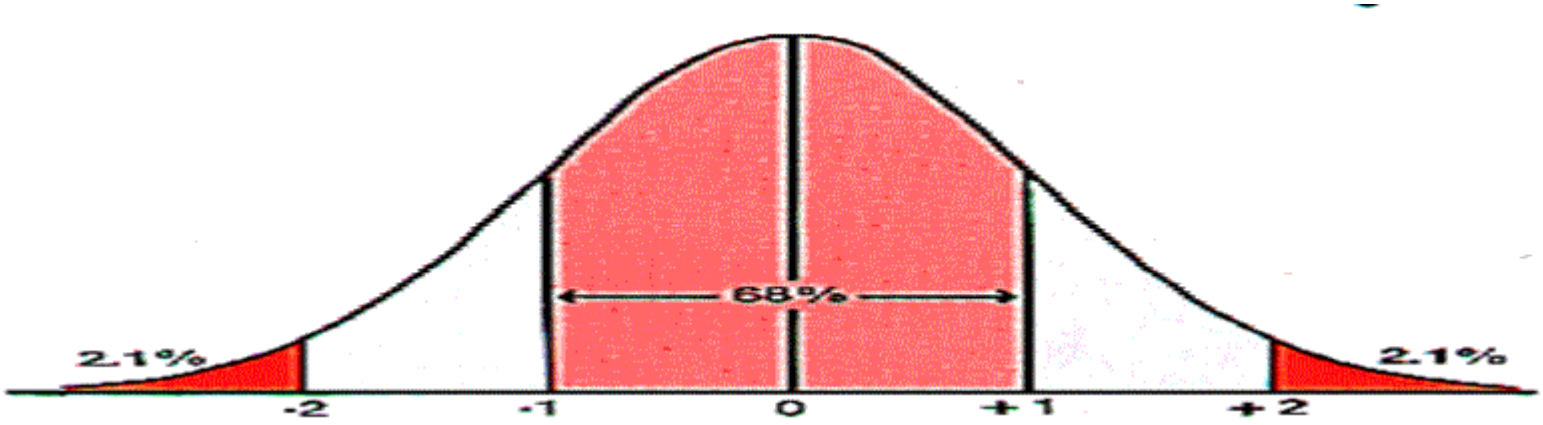
aps
ASSOCIATION FOR PSYCHOLOGICAL SCIENCE

Rethinking the Diagnosis of Mental Disorders: Data-Driven Psychological Dimensions, Not Categories, as a Framework for Mental-Health Research, Treatment, and Training

Current Directions in Psychological Science
2021, Vol. 30(2) 151–158

DOI: 10.1177/0963721421990353
www.psychologicalscience.org/CDPS
SAGE

Christopher C. Conway[1], Robert F. Krueger[2], and the HiTOP Consortium Executive Board*
[1]Department of Psychology, Fordham University, and [2]Department of Psychology, University of Minnesota
*The members of the HiTOP Consortium Executive Board are listed in the Transparency section at the end of the article.


68%
2.1%
2.1%
-2
-1
0
+1
+2


Adobe Stock | #510336870

Lizarazu Phd Thesis: Speech-brain synchronization: a possible cause for developmental dyslexia

Thesis · March 2017

CITATIONS
3

READS
5,357

1 author:

Mikel Lizarazu
Basque Center on Cognition, Brain and Language
43 PUBLICATIONS **787** CITATIONS

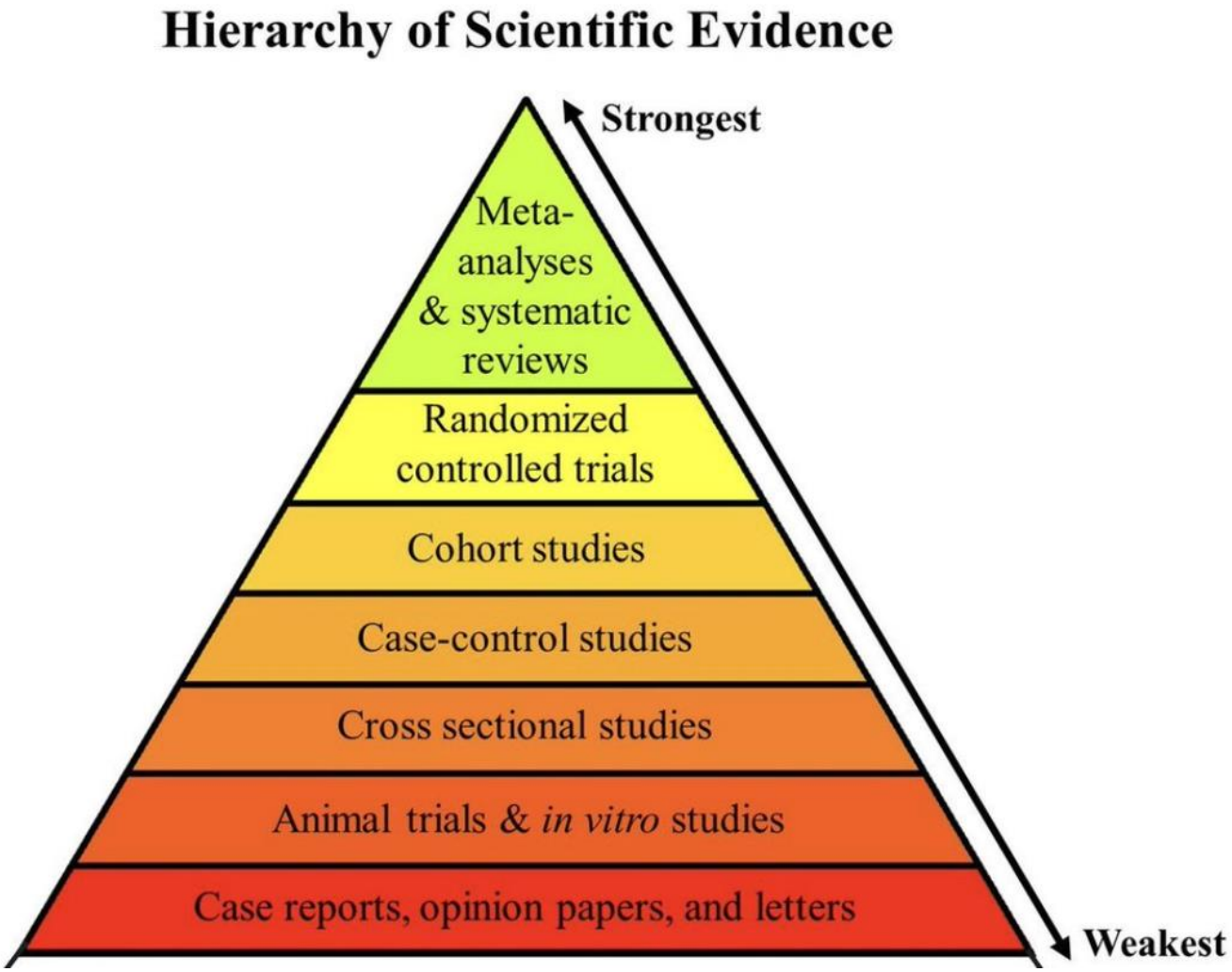
SEE PROFILE

Cogn Neuropsychol. 2012 February ; 29(1-2): 34–55. doi:10.1080/02643294.2012.710600.

The *maps problem* and the *mapping problem*: Two challenges for a cognitive neuroscience of speech and language

David Poeppel
Department of Psychology, NYU, 6 Washington Place, New York, NY 10003, 212 992 7489, david.poeppel@nyu.edu

Distill language (and linguistic theory) into a set of computational and representational primitives!

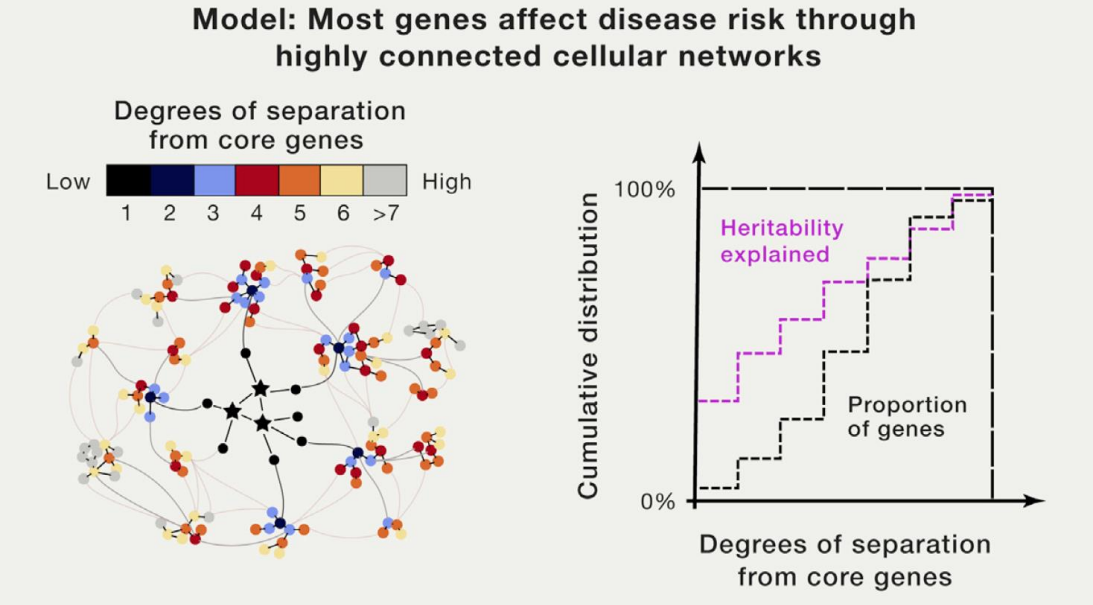

Hierarchy of Scientific Evidence
Strongest
Meta-analyses & systematic reviews
Randomized controlled trials
Cohort studies
Case-control studies
Cross sectional studies
Animal trials & *in vitro* studies
Case reports, opinion papers, and letters
Weakest

Leading Edge
Perspective

Cell

An Expanded View of Complex Traits: From Polygenic to Omnigenic

Evan A. Boyle,[1,*] Yang I. Li,[1,*] and Jonathan K. Pritchard[1,2,3,*]
[1]Department of Genetics
[2]Department of Biology
[3]Howard Hughes Medical Institute
Stanford University, Stanford, CA 94305, USA
*Correspondence: eaboyle@stanford.edu (E.A.B.), yangili@stanford.edu (Y.I.L.), pritch@stanford.edu (J.K.P.)
http://dx.doi.org/10.1016/j.cell.2017.05.038

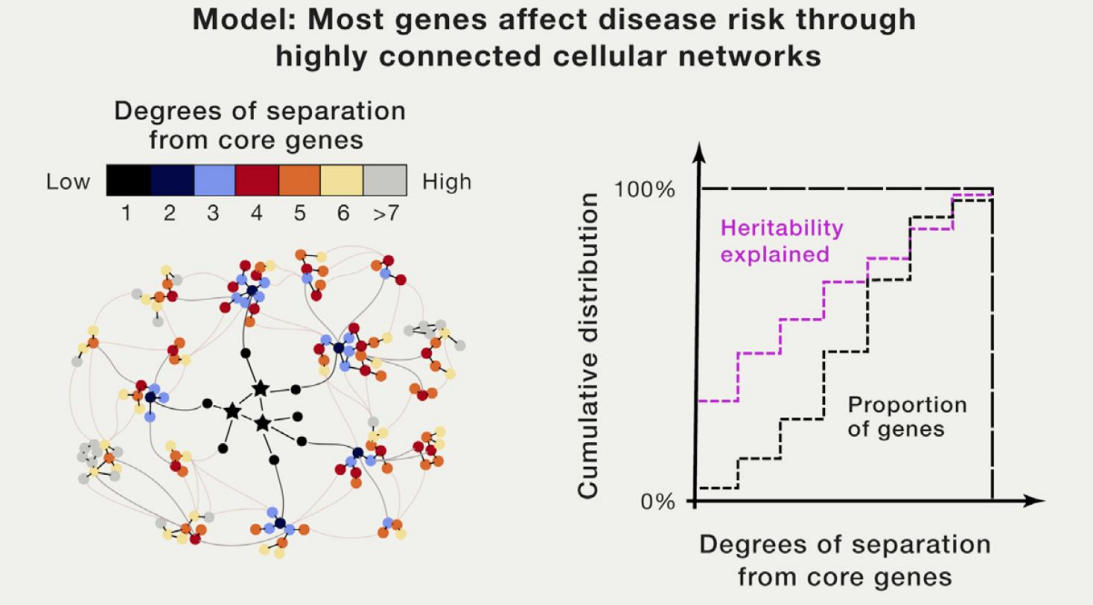


Leading Edge
Perspective

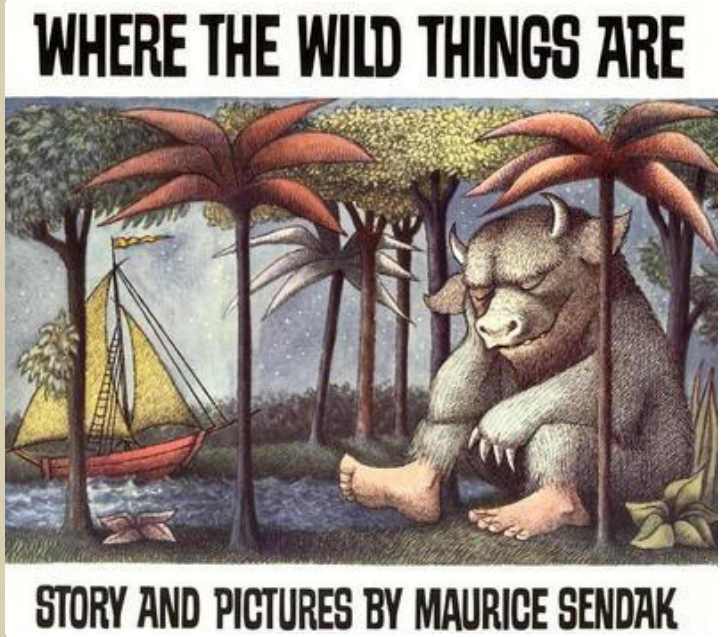
Cell

An Expanded View of Complex Traits: From Polygenic to Omnigenic

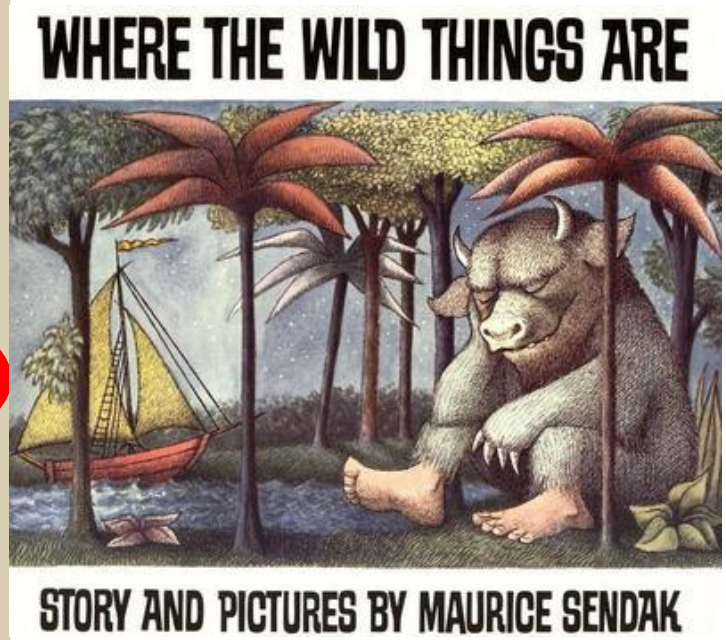
Evan A. Boyle,[1,*] Yang I. Li,[1,*] and Jonathan K. Pritchard[1,2,3,*]
[1]Department of Genetics
[2]Department of Biology
[3]Howard Hughes Medical Institute
Stanford University, Stanford, CA 94305, USA
*Correspondence: eaboyle@stanford.edu (E.A.B.), yangili@stanford.edu (Y.I.L.), pritch@stanford.edu (J.K.P.)
http://dx.doi.org/10.1016/j.cell.2017.05.038



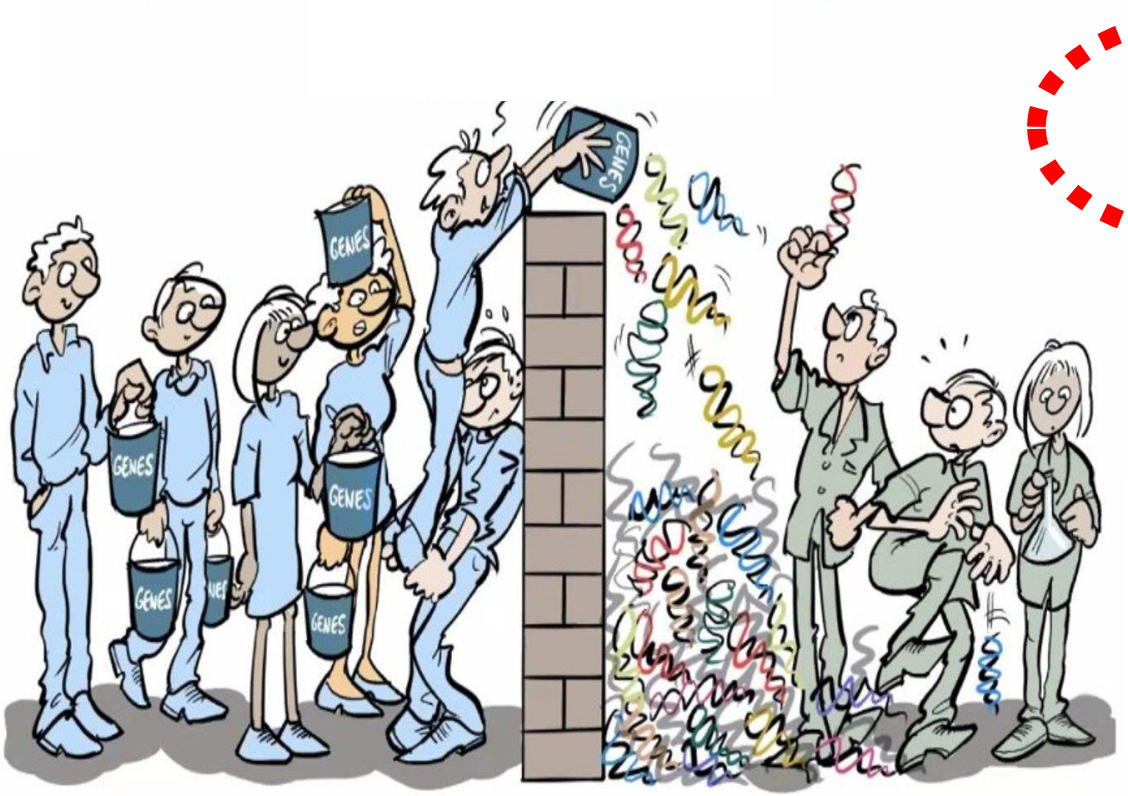
Language	Phenome
Cognitive functions	Cognome
Neural synchronization	Dynome
Neural wiring	Connectome
Neural populations and brain areas	Cytome
Codes for neural development and assembly	Toponome
Signalling pathways	Organome
Protein interactions	Interactome
Proteins	Proteome
RNAs	Transcriptome
Epigenetic modifications	Epigenome
Genes	Genome



Language	Phenome
Cognitive functions	Cognome
Neural synchronization	Dyn…
Neural wiring	Connectome
Neural populations and brain areas	Cytome
Codes for neural development and assembly	Toponome
Signalling pathways	Organome
Protein interactions	Interactome
Proteins	Proteome
RNAs	Transcriptome
Epigenetic modifications	Epigenome
Genes	Genome

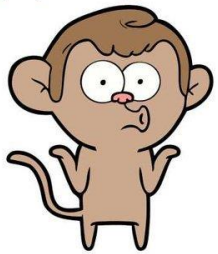


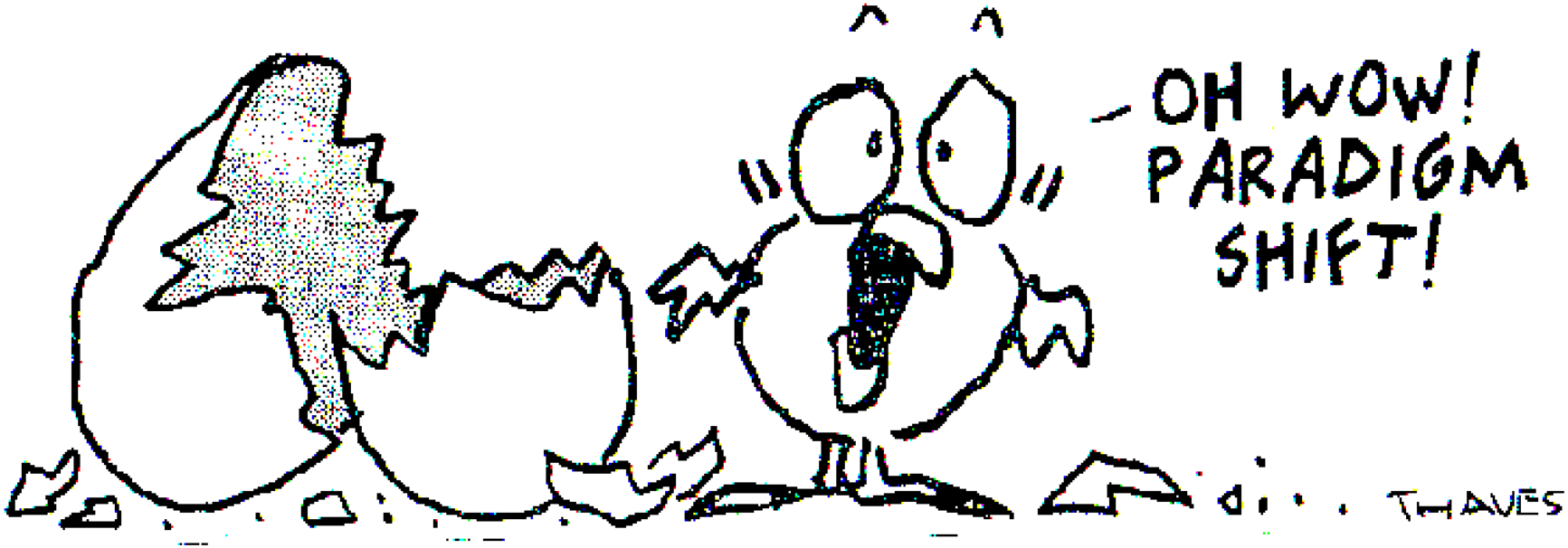
Molecular biology Clinical linguistics

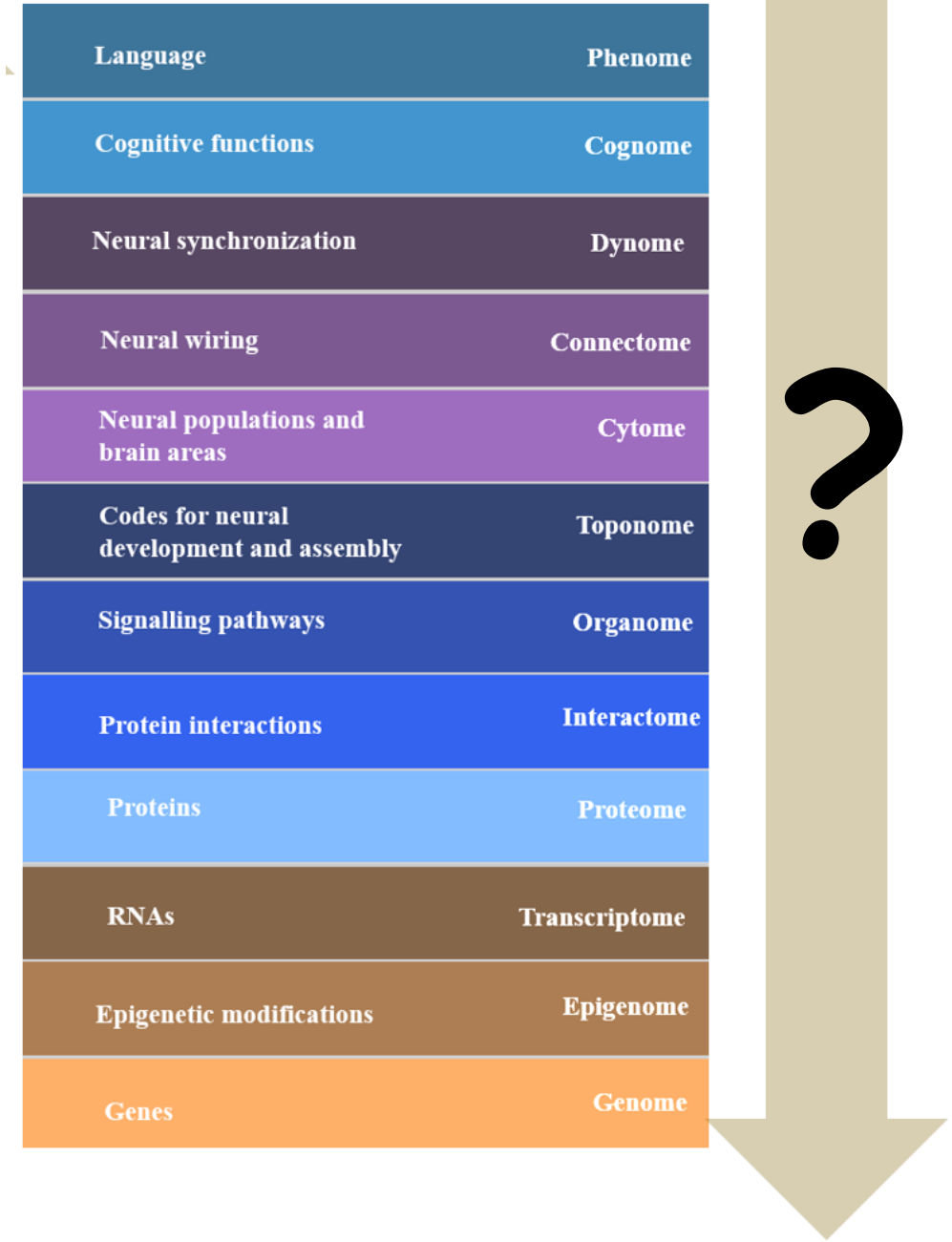


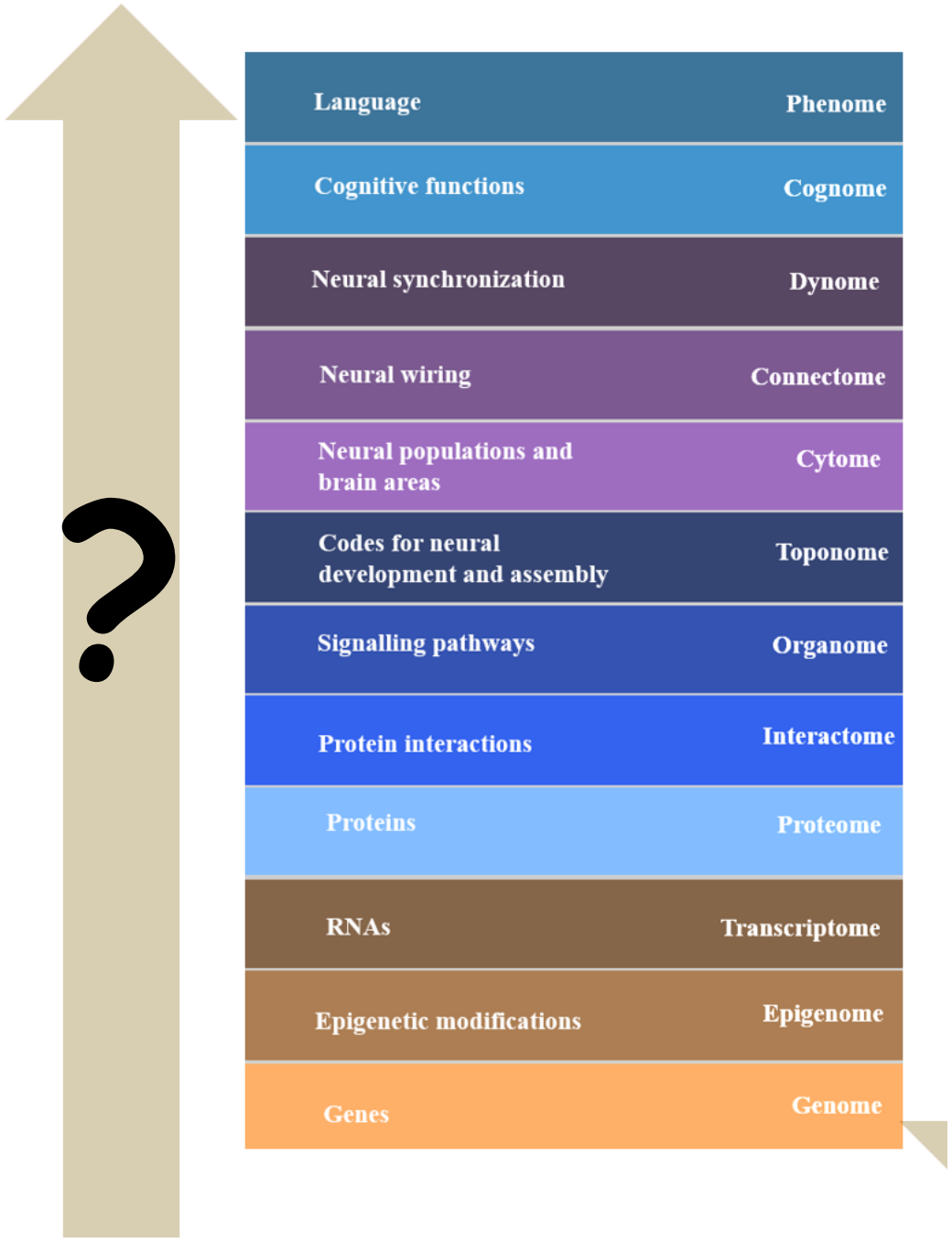
Functional experiments

basic and clinical psychologists

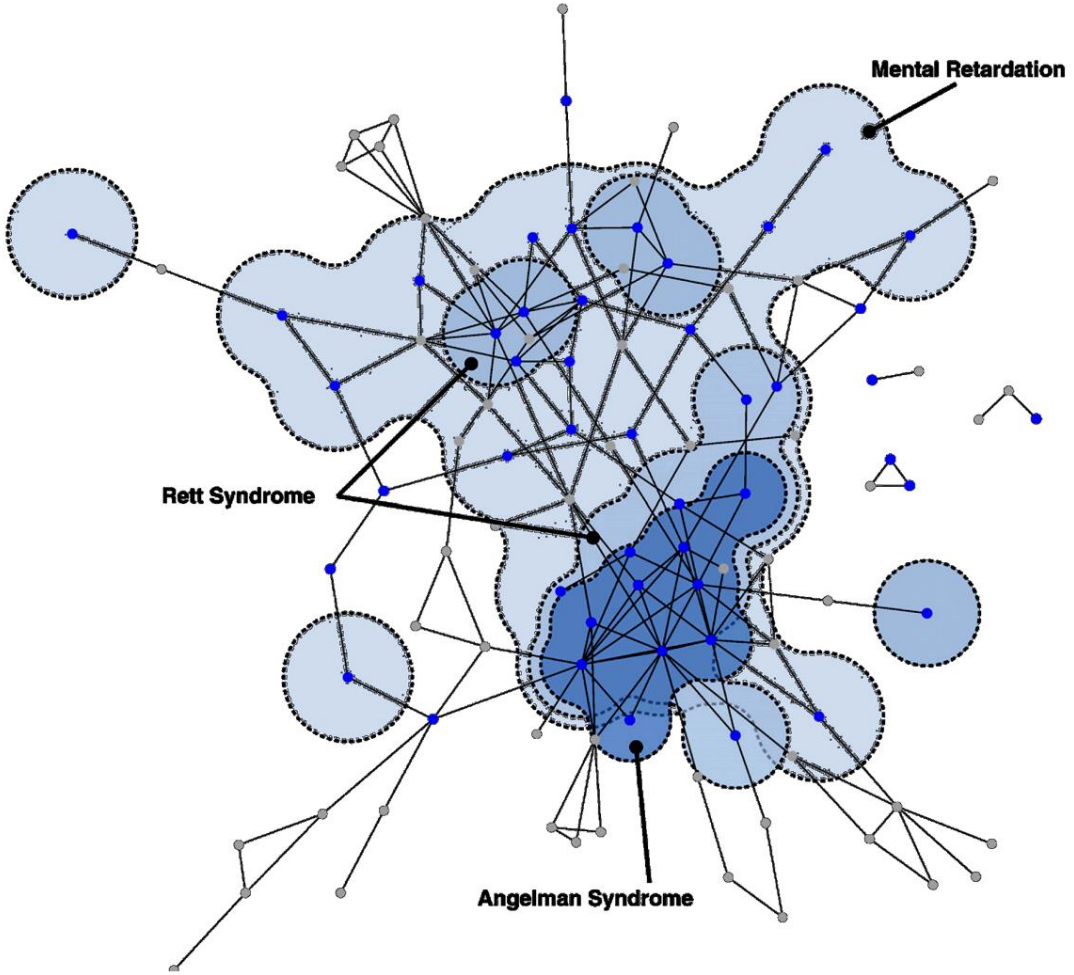



OH WOW!
PARADIGM
SHIFT!
THAVES

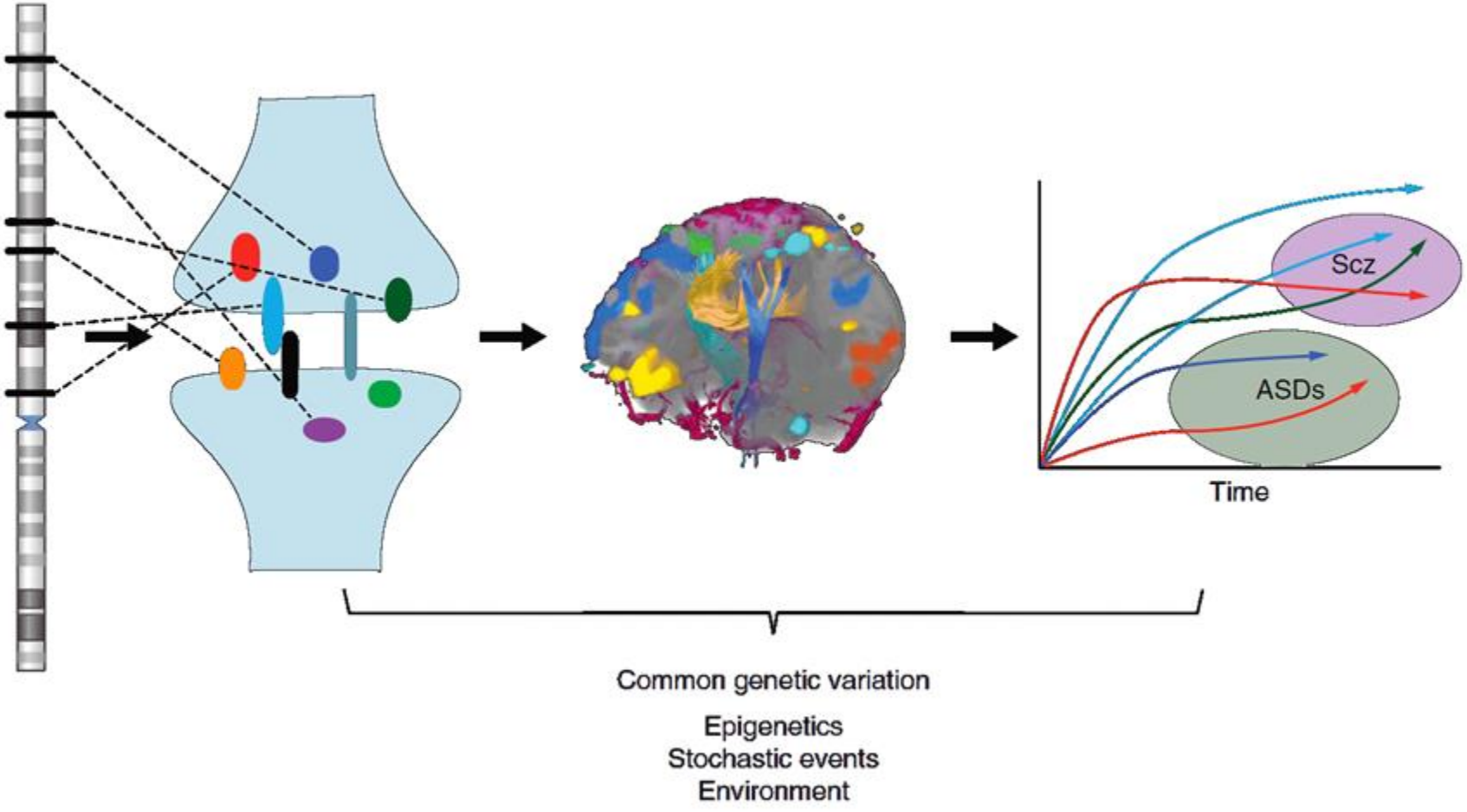

Language
Phenome
Cognitive functions
Cognome
Neural synchronization
Dynome
Neural wiring
Connectome
Neural populations and brain areas
Cytome
Codes for neural development and assembly
Toponome
Signalling pathways
Organome
Protein interactions
Interactome
Proteins
Proteome
RNAs
Transcriptome
Epigenetic modifications
Epigenome
Genes
Genome
?


?
Language
Phenome
Cognitive functions
Cognome
Neural synchronization
Dynome
Neural wiring
Connectome
Neural populations and brain areas
Cytome
Codes for neural development and assembly
Toponome
Signalling pathways
Organome
Protein interactions
Interactome
Proteins
Proteome
RNAs
Transcriptome
Epigenetic modifications
Epigenome
Genes
Genome

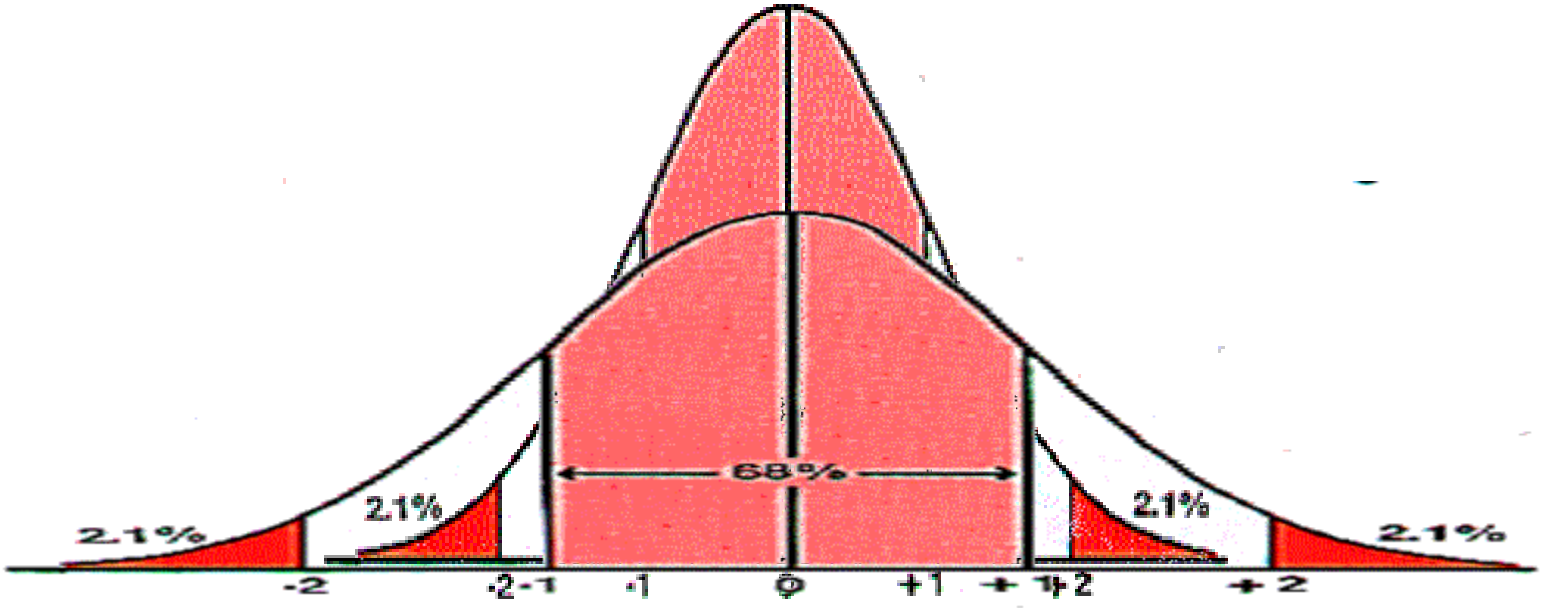
1. There are less disorders than causes of disorders



1. There are less disorders than causes of disorders

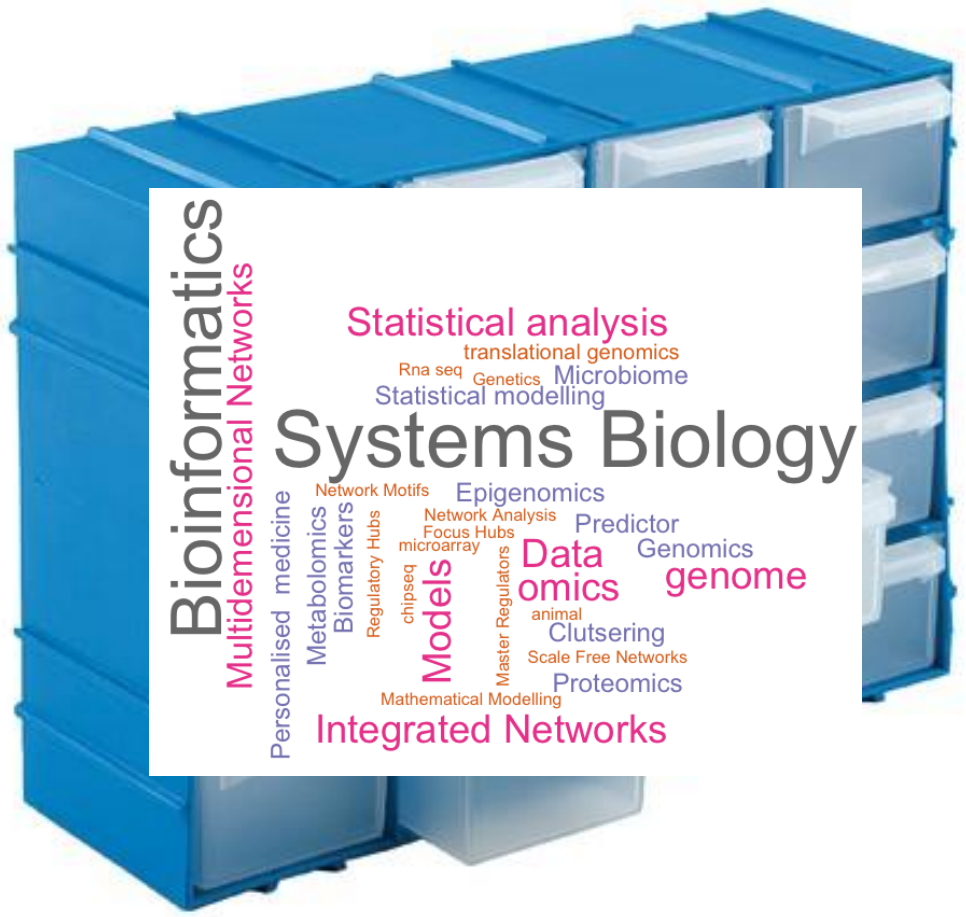


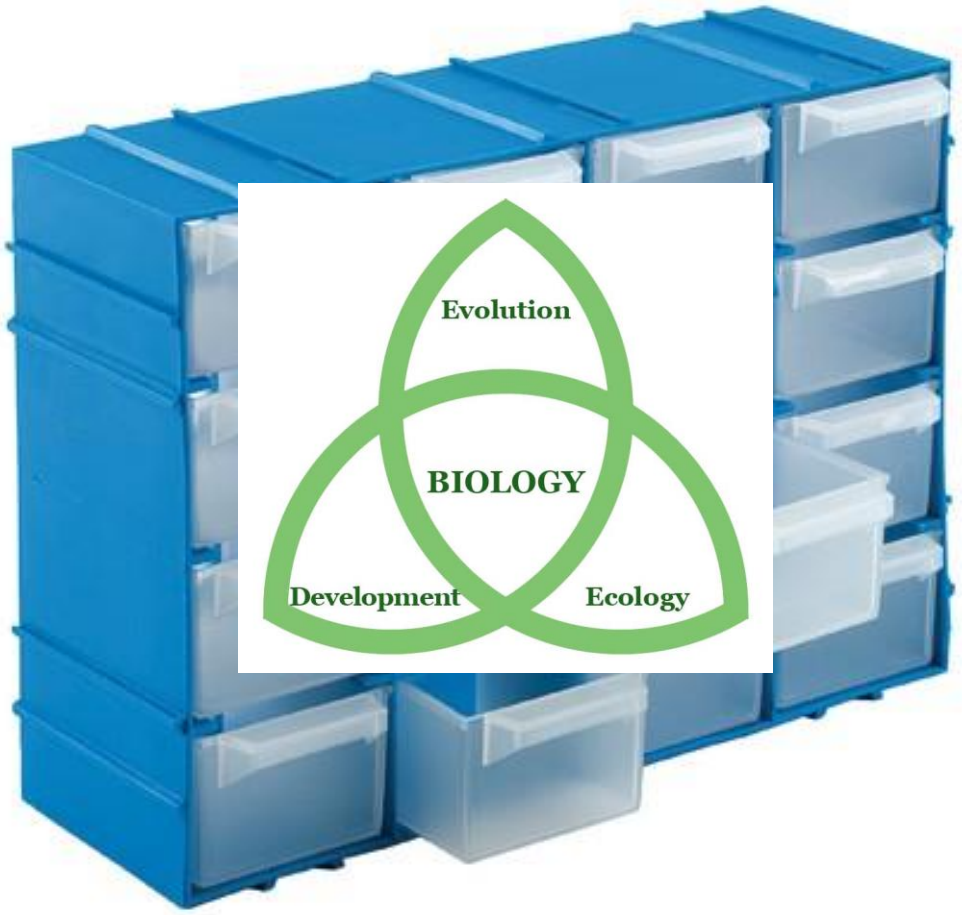
2. The variability exhibited by neurodiverse people is greater than the variability observed in neurotypical subjects



3. Breakdowns and compensations never occur randomly
   - some aspects of language are particularly vulnerable
   - some aspects of language are particularly resistant to damage

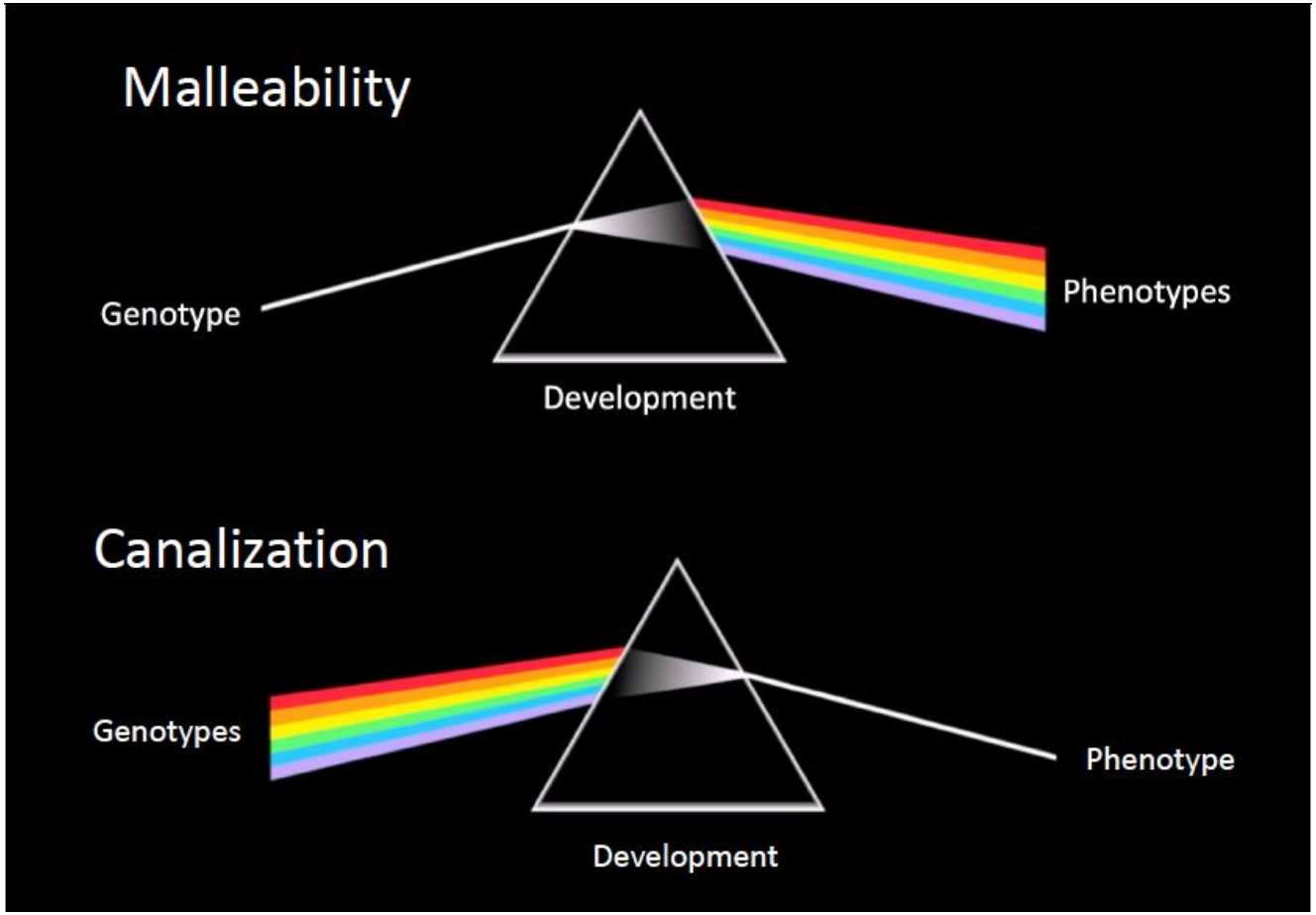
KEIO/1


Bioinformatics
Multidemensional Networks
Personalised medicine
Metabolomics
Biomarkers
Regulatory Hubs
chipseq
Models
Master Regulators
Statistical analysis
translational genomics
Rna seq
Genetics
Microbiome
Statistical modelling
Systems Biology
Network Motifs
Epigenomics
Network Analysis
Focus Hubs
microarray
Predictor
Data
Genomics
omics
genome
animal
Clutsering
Scale Free Networks
Proteomics
Mathematical Modelling
Integrated Networks


Evolution
BIOLOGY
Development
Ecology

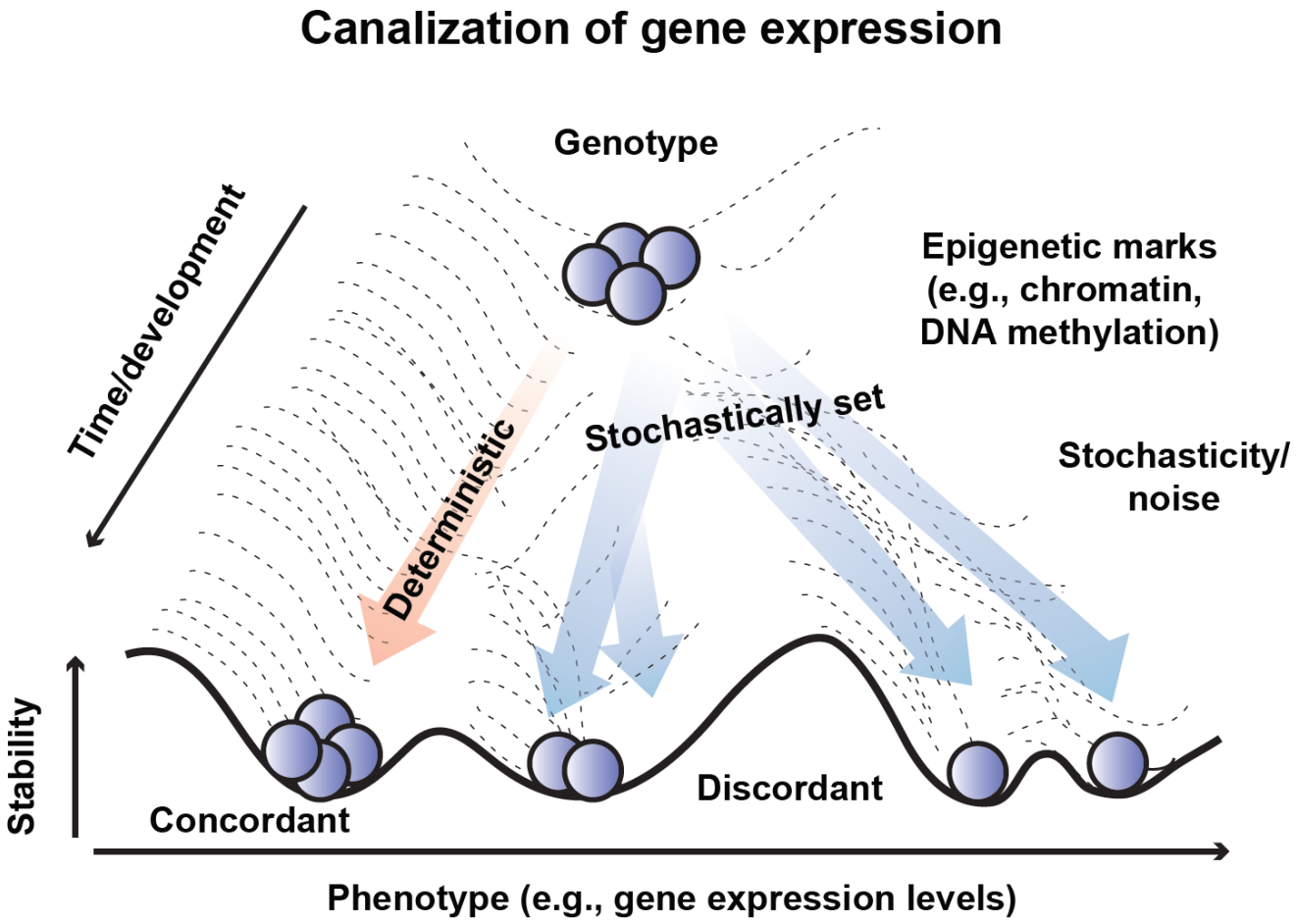
1. There are less disorders than causes of disorders

2. The variability exhibited by neurodiverse people is greater than the variability observed in neurotypical subjects



1. There are less disorders than causes of disorders

2. The variability exhibited by neurodiverse people is greater than the variability observed in neurotypical subjects



1. There are less disorders than causes of disorders

2. The variability exhibited by neurodiverse people is greater than the variability observed in neurotypical subjects

3. Breakdowns and compensations never occur randomly

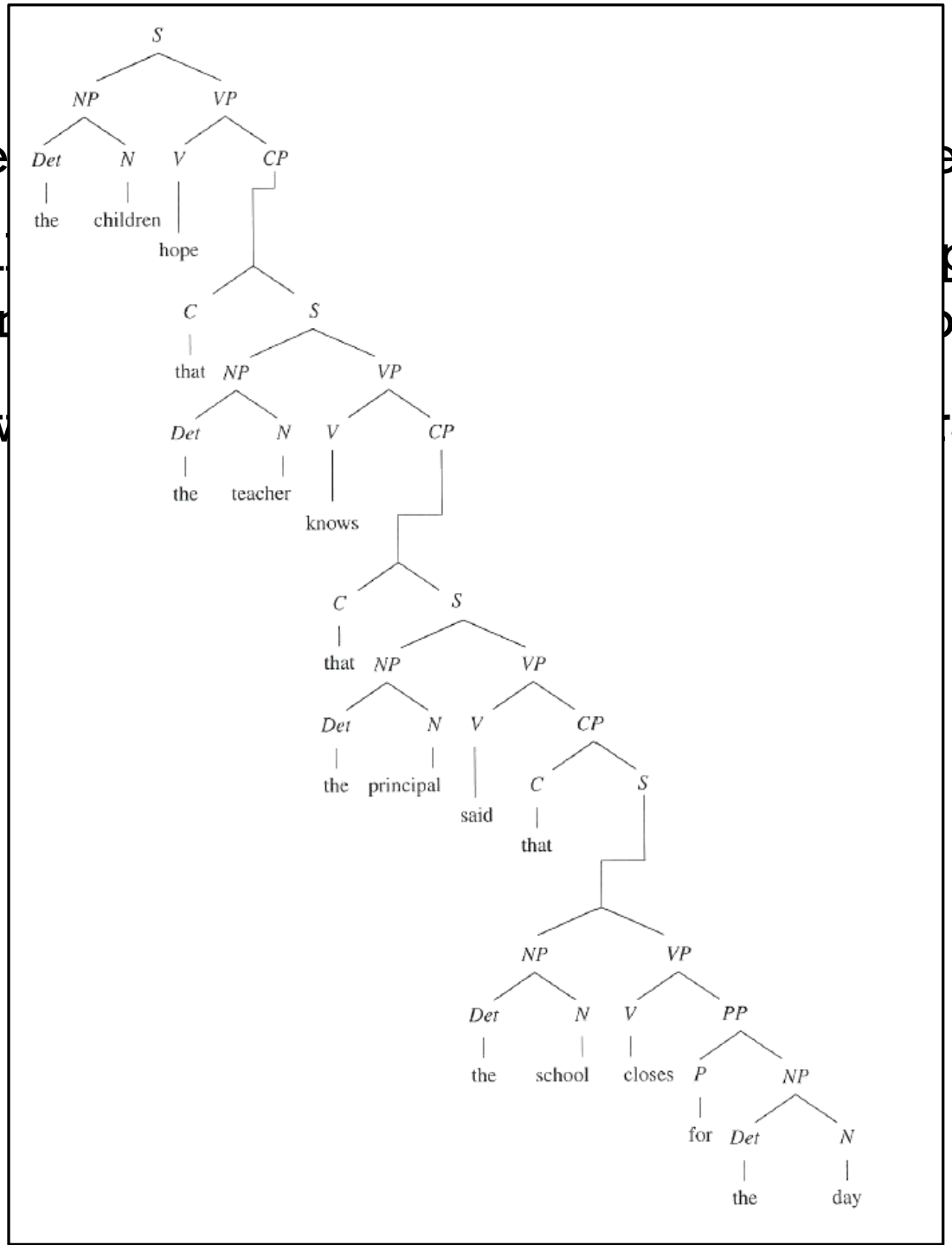
Robust

Evolvable

1. There are ers

2. The varia ple is
greater thar bical subjects

3. Breakdov randomly



Archival Report

Biological Psychiatry

Genetic Markers of Human Evolution Are Enriched in Schizophrenia

Saurabh Srinivasan, Francesco Bettella, Morten Mattingsdal, Yunpeng Wang, Aree Witoelar, Andrew J. Schork, Wesley K. Thompson, Verena Zuber, The Schizophrenia Working Group of the Psychiatric Genomics Consortium, The International Headache Genetics Consortium, Bendik S. Winsvold, John-Anker Zwart, David A. Collier, Rahul S. Desikan, Ingrid Melle, Thomas Werge, Anders M. Dale, Srdjan Djurovic, and Ole A. Andreassen

PLOS | GENETICS

RESEARCH ARTICLE

Widespread signatures of positive selection in common risk alleles associated to autism spectrum disorder

Renato Polimanti[1,2]*, **Joel Gelernter**[1,2,3,4]

1 Department of Psychiatry, Yale School of Medicine, West Haven, Connecticut, United States of America, **2** VA CT Healthcare Center, West Haven, Connecticut, United States of America, **3** Departments of Genetics, Yale School of Medicine, New Haven, Connecticut, United States of America, **4** Department of Neuroscience, Yale University School of Medicine, New Haven, Connecticut, United States of America

ELSEVIER

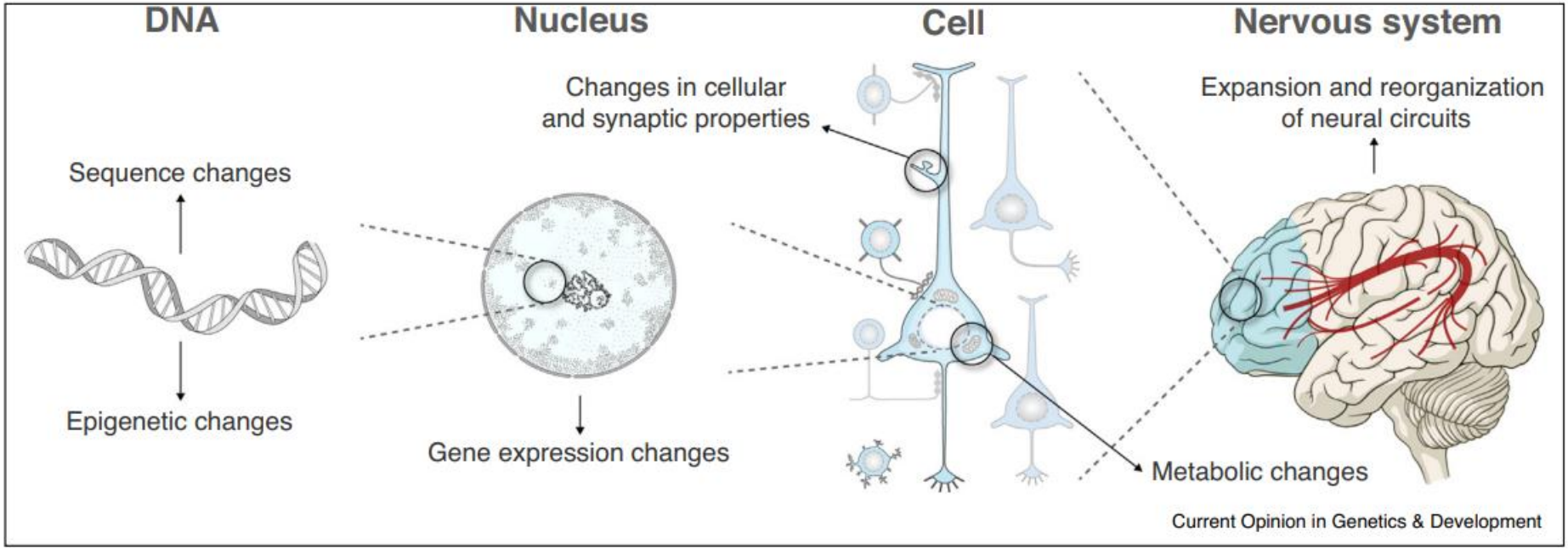
Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Genetics & Development

The evolution of the human brain and disease susceptibility

Kartik Pattabiraman[1,2,5], Sydney Keaton Muchnik[1,3,5] and Nenad Sestan[1,2,3,4]

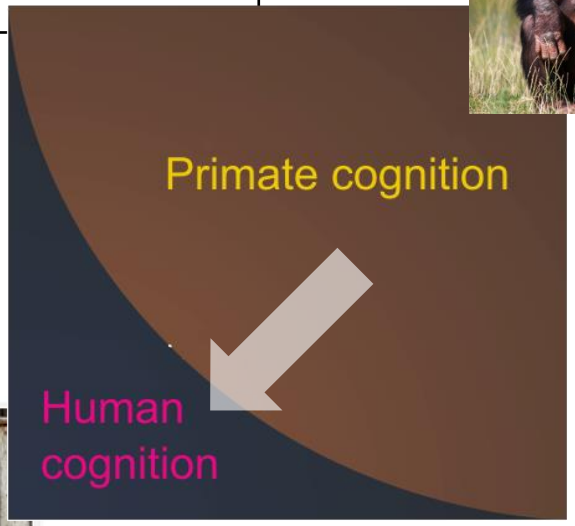


PERSPECTIVES

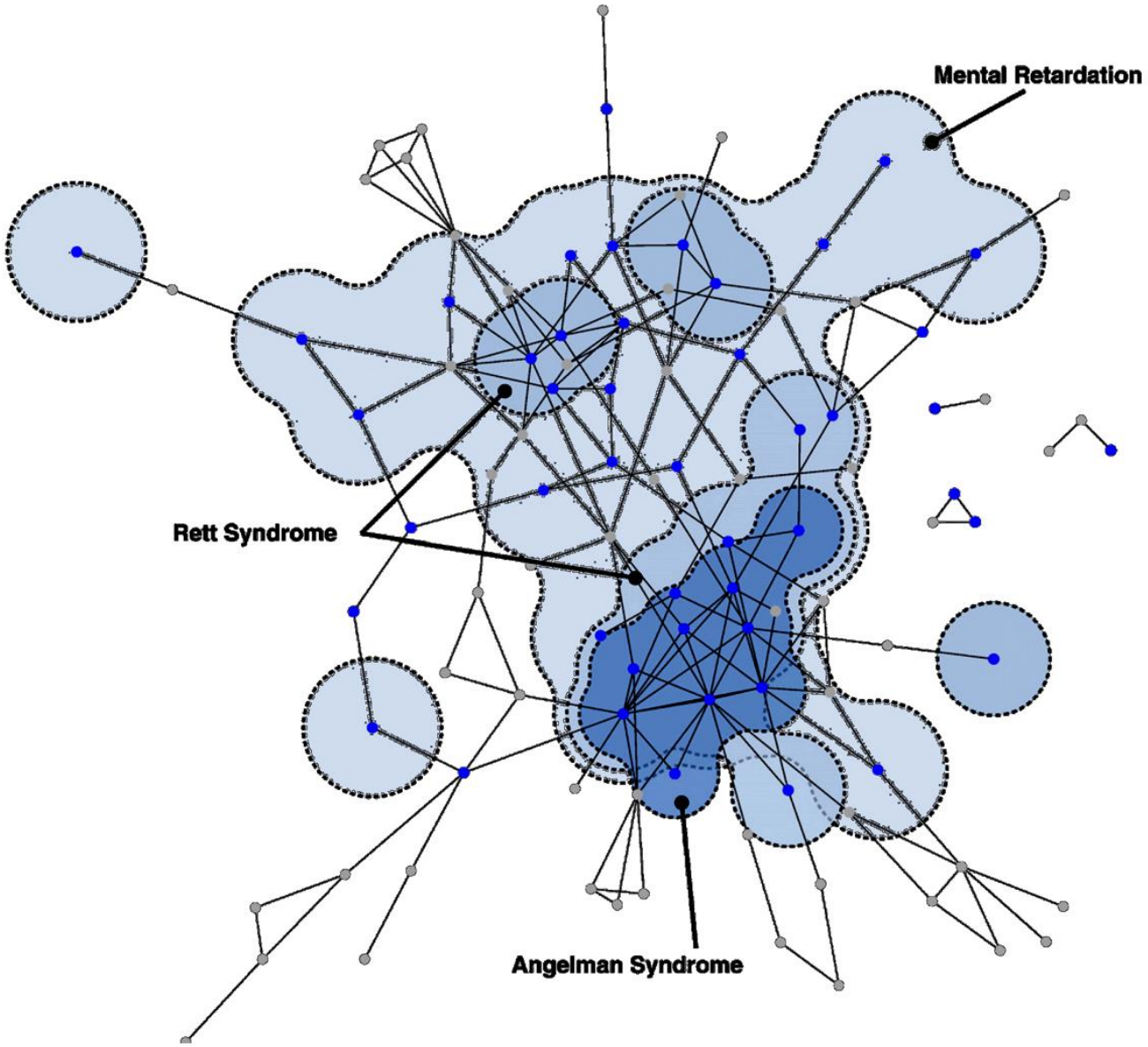
OPINION

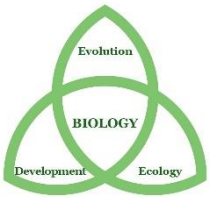
Decanalization and the origin of complex disease

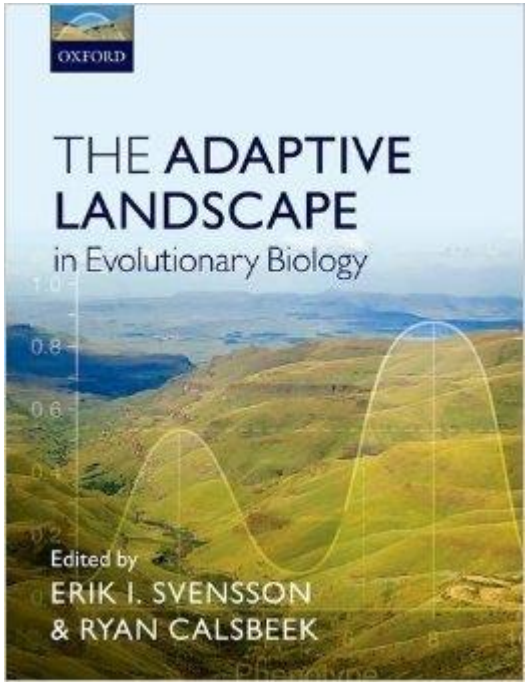
Greg Gibson

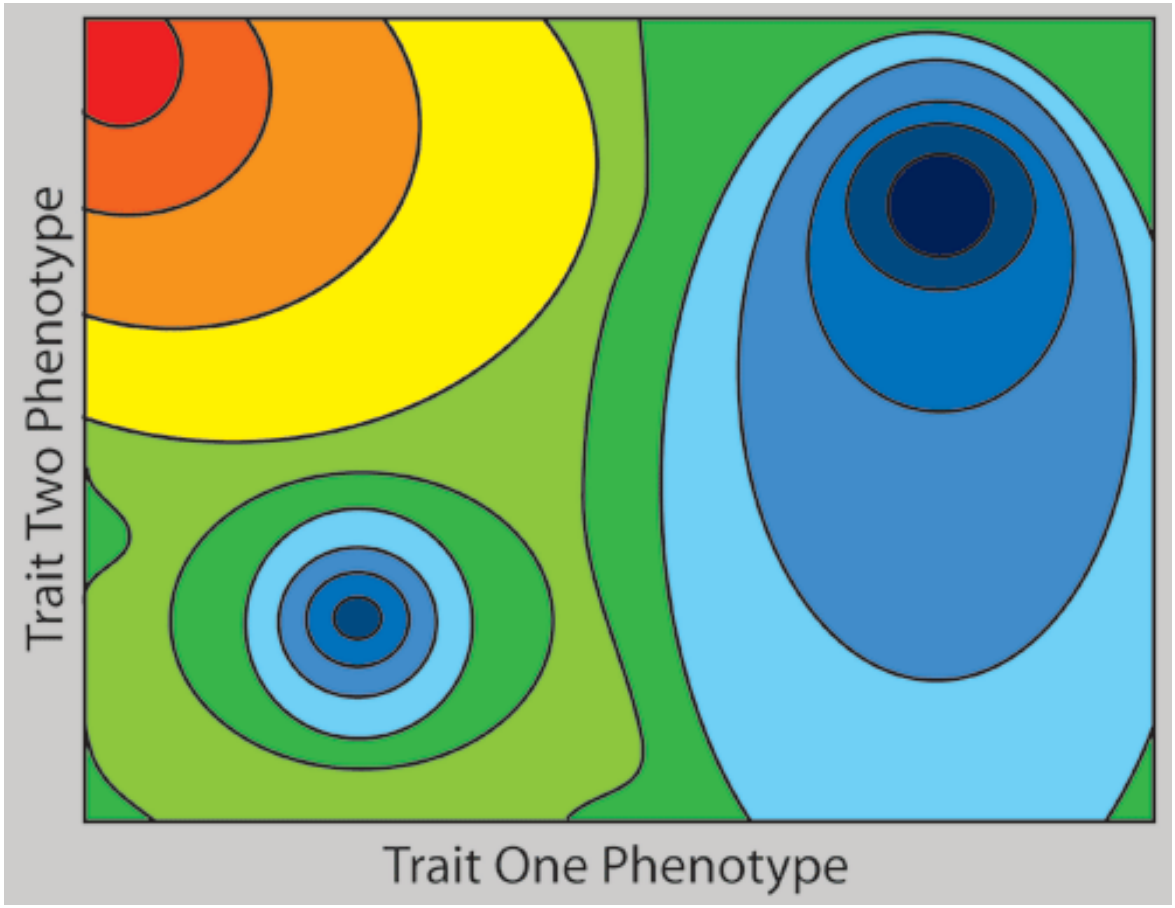


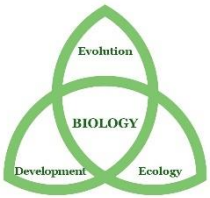
- demography
- DNA
- culture

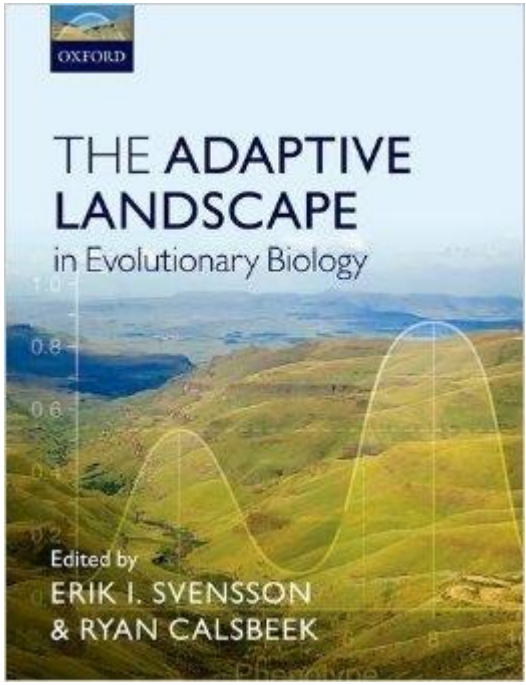

Mental Retardation
Rett Syndrome
Angelman Syndrome

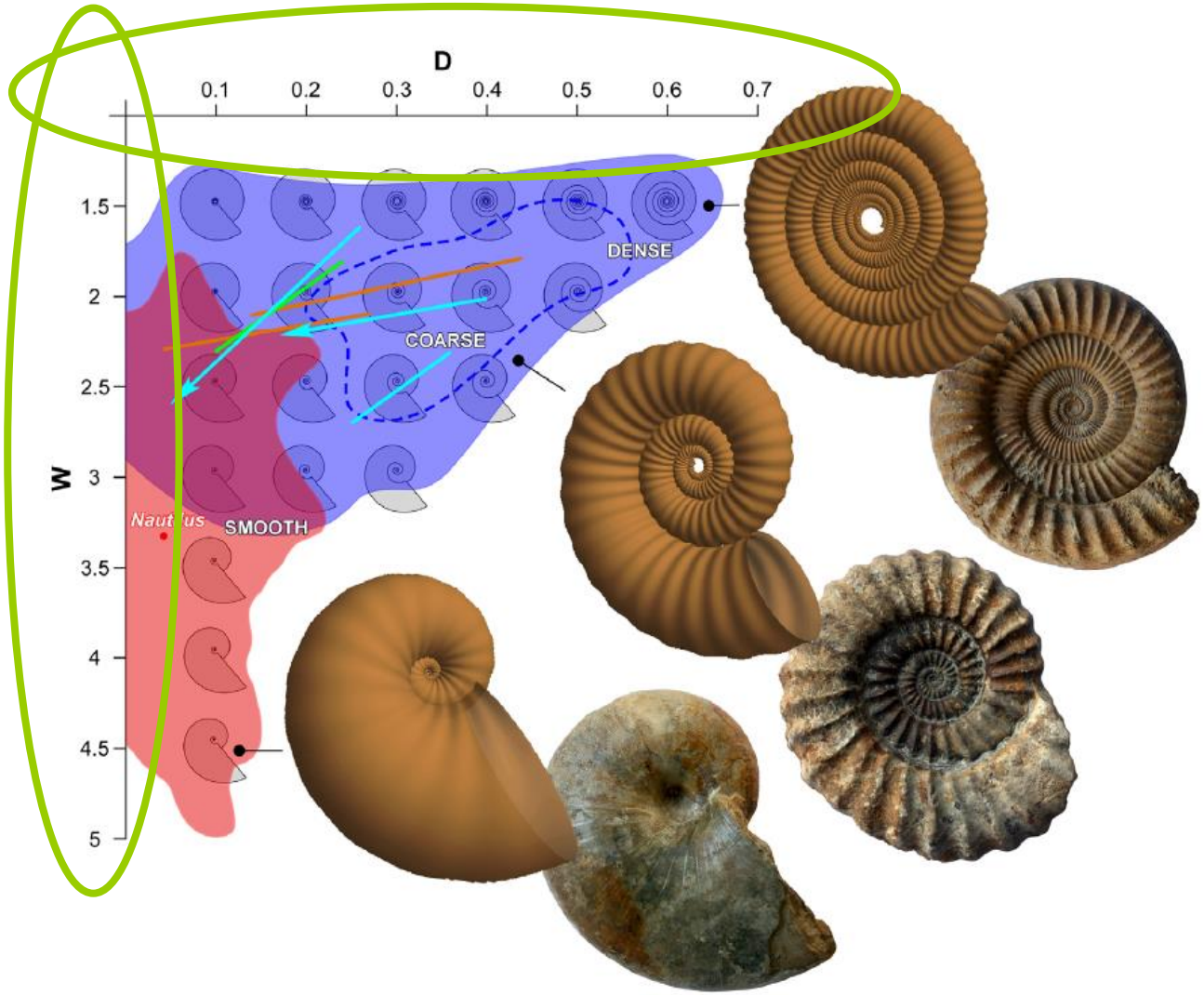

Evolution
BIOLOGY
Development
Ecology

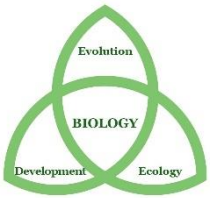

OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

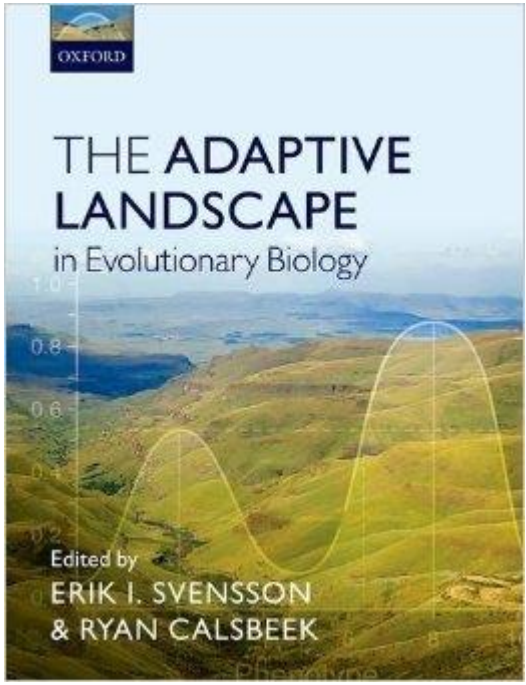

Trait Two Phenotype
Trait One Phenotype

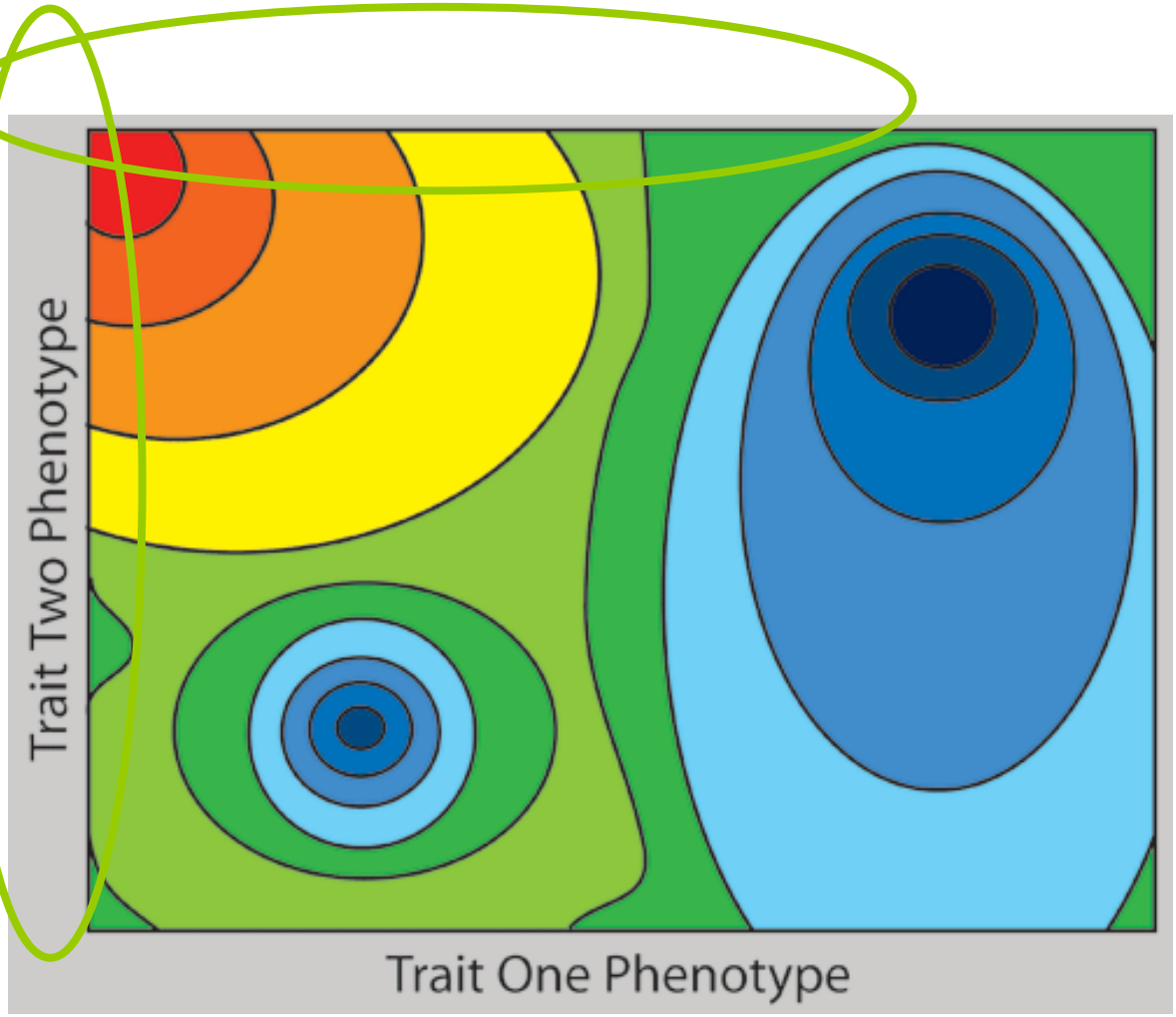

Evolution
BIOLOGY
Development
Ecology

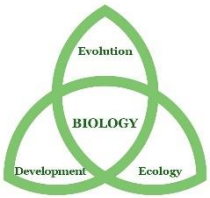

OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

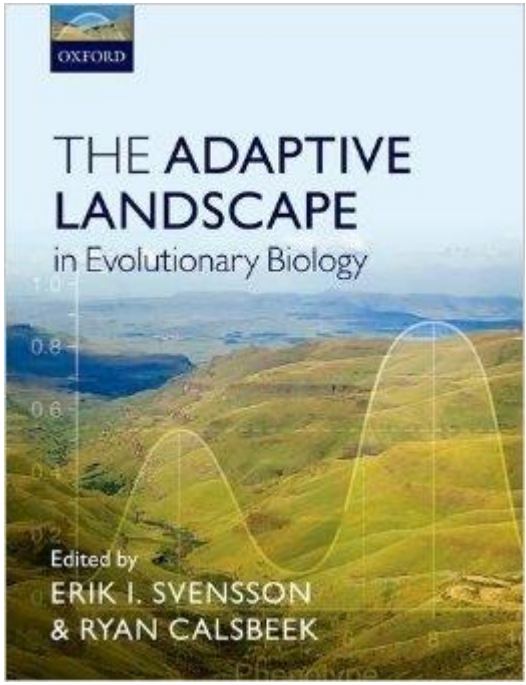

D
0.1
0.2
0.3
0.4
0.5
0.6
0.7
W
1.5
2
2.5
3
3.5
4
4.5
5
DENSE
COARSE
SMOOTH
Nautilus

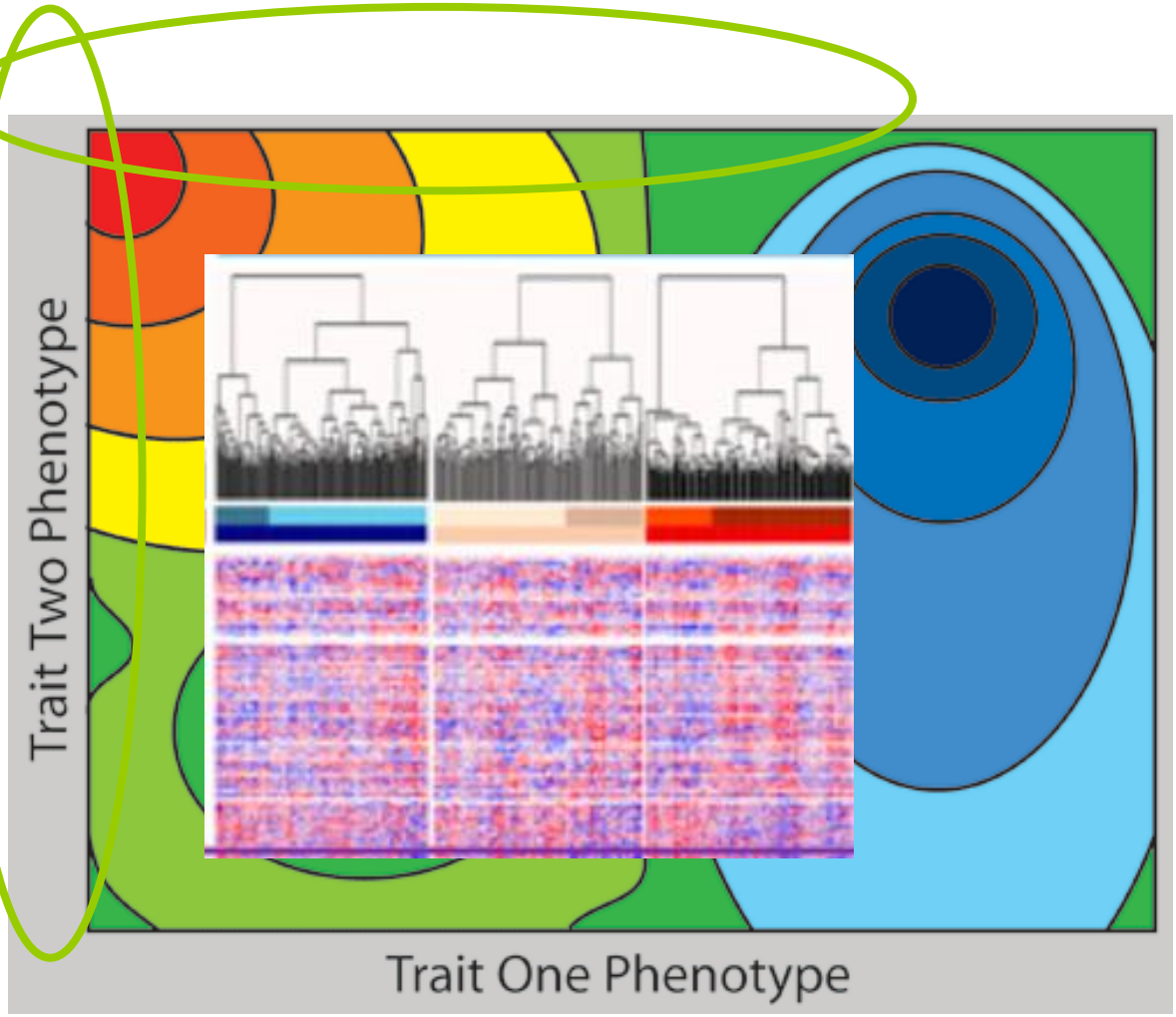

Evolution
BIOLOGY
Development
Ecology

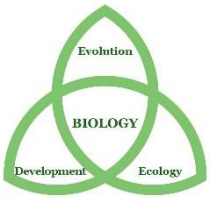

OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

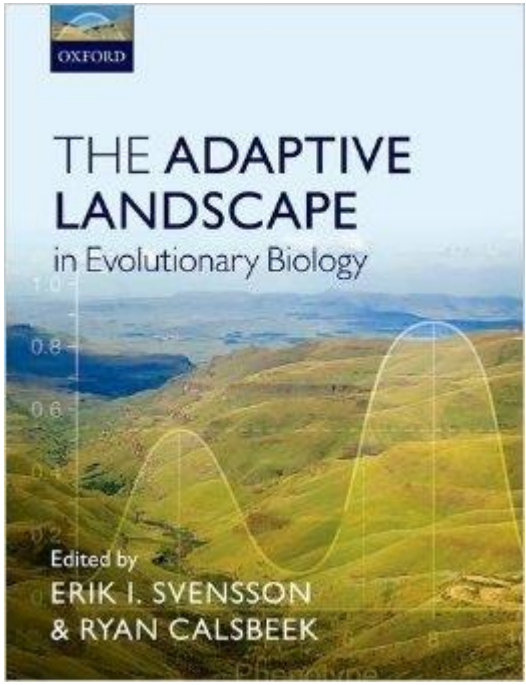

Trait Two Phenotype
Trait One Phenotype

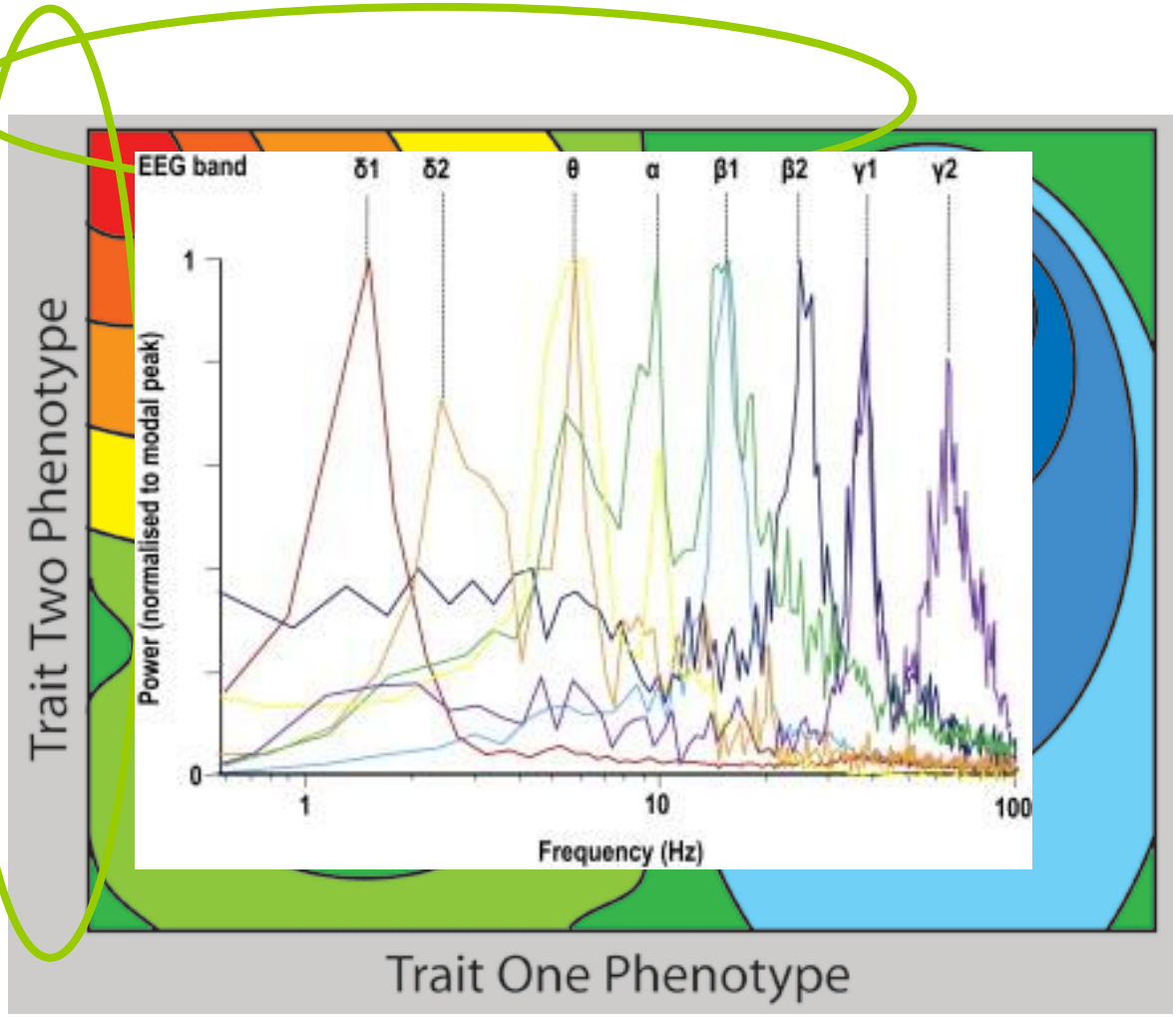

Evolution
BIOLOGY
Development
Ecology

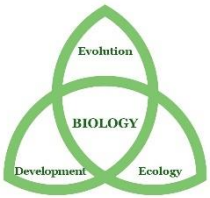

OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

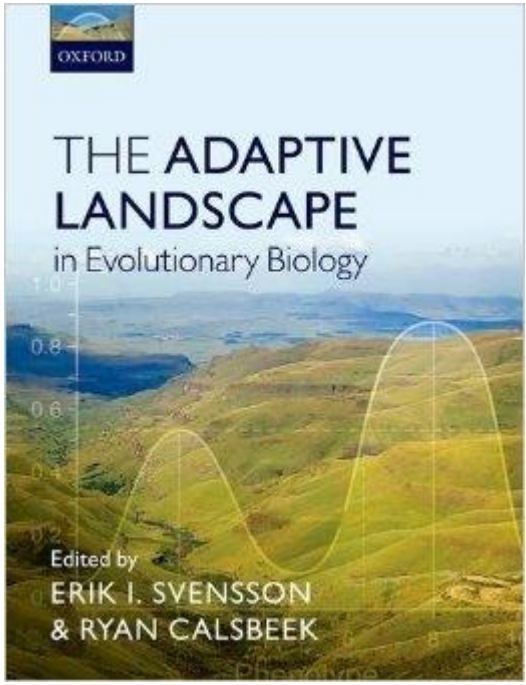

Trait Two Phenotype
Trait One Phenotype


Evolution
BIOLOGY
Development
Ecology


OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

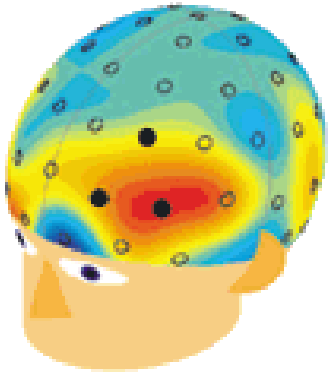

Trait Two Phenotype
EEG band
δ1
δ2
θ
α
β1
β2
γ1
γ2
Power (normalised to modal peak)
1
0
1
10
100
Frequency (Hz)
Trait One Phenotype


Evolution
BIOLOGY
Development
Ecology

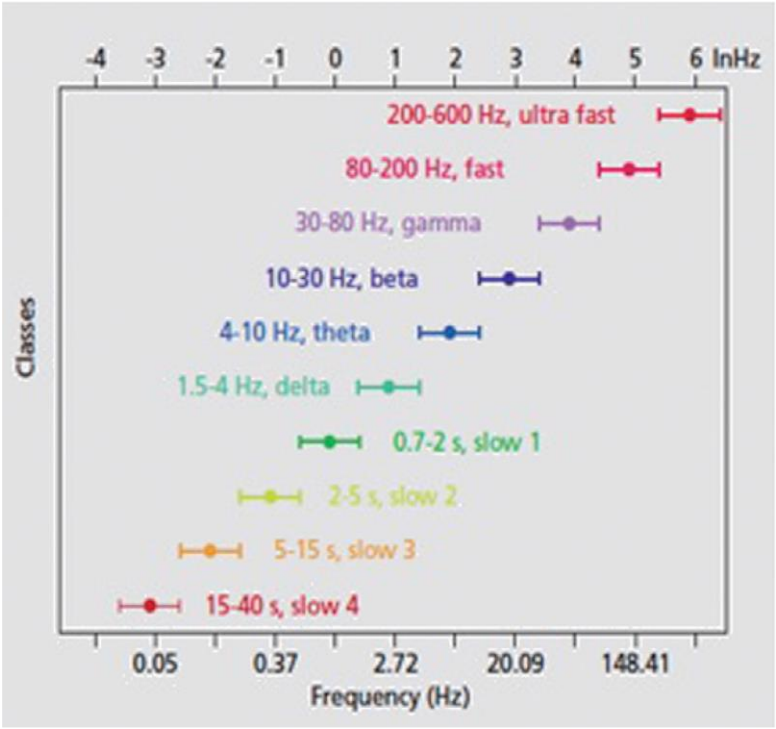

OXFORD
THE ADAPTIVE LANDSCAPE
in Evolutionary Biology
Edited by
ERIK I. SVENSSON
& RYAN CALSBEEK

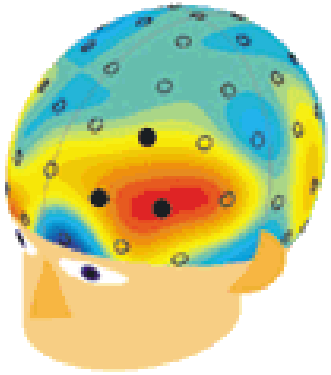
HOW I HAVE STRUCTURED MY TALK

1. Introduction
2. Clinical linguistics: struggling with symptoms and causes
3. Bridging genes to language (and vice versa)
4. Putting the focus on brain oscillations
5. Conclusions and future prospects



- Brain rhythms are primitive components of brain function



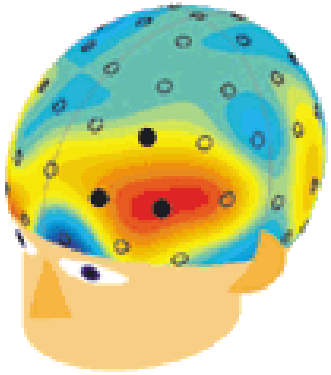


- Brain rhythms are primitive components of brain function
- (Ideally) They allow to understand (not just to map) the brain specialization for language

Cogn Neuropsychol. 2012 February ; 29(1-2): 34–55. doi:10.1080/02643294.2012.710600.

The *maps problem* and the *mapping problem*: Two challenges for a cognitive neuroscience of speech and language

David Poeppel
Department of Psychology, NYU, 6 Washington Place, New York, NY 10003, 212 992 7489, david.poeppel@nyu.edu



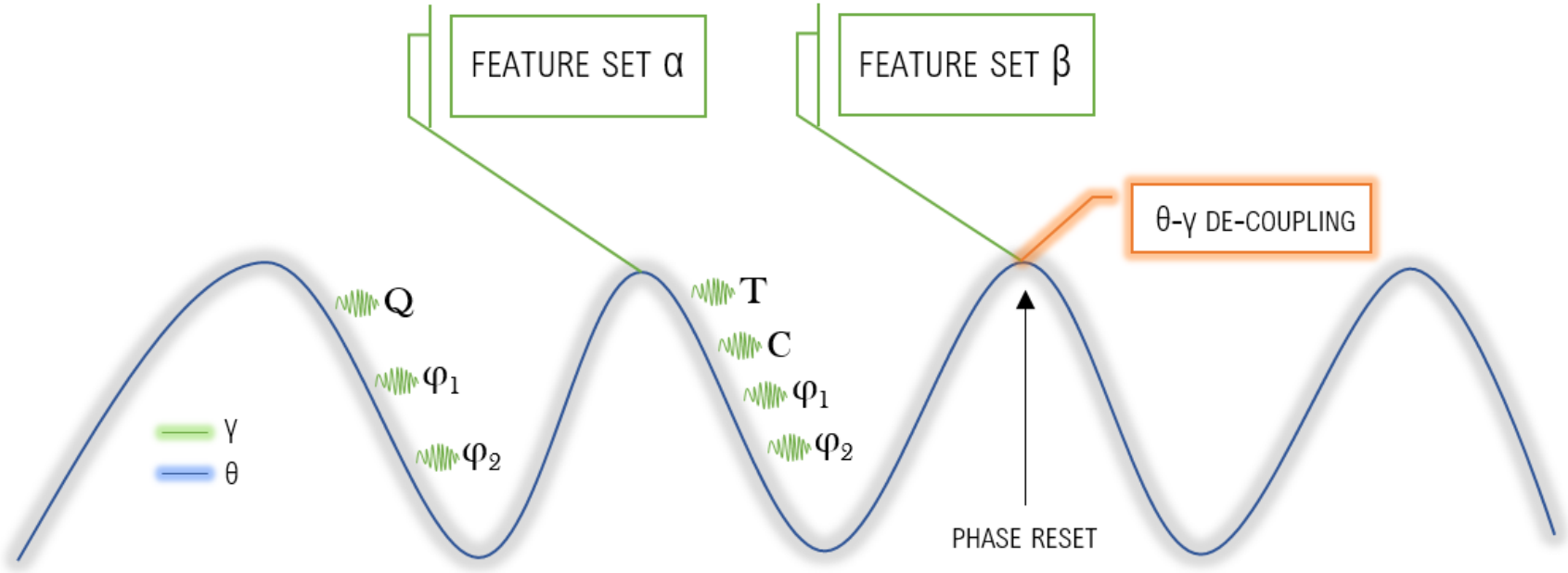
frontiers in Psychology

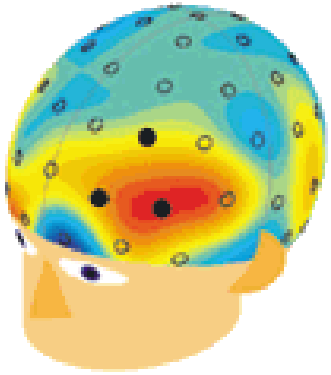
HYPOTHESIS AND THEORY
published: 13 October 2015
doi: 10.3389/fpsyg.2015.01515

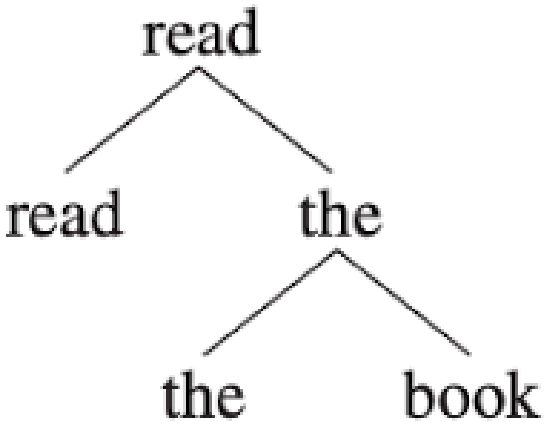
The brain dynamics of linguistic computation

Elliot Murphy *

Division of Psychology and Language Sciences, University College London, London, UK





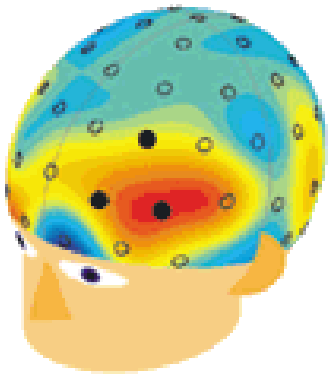


PERSPECTIVE

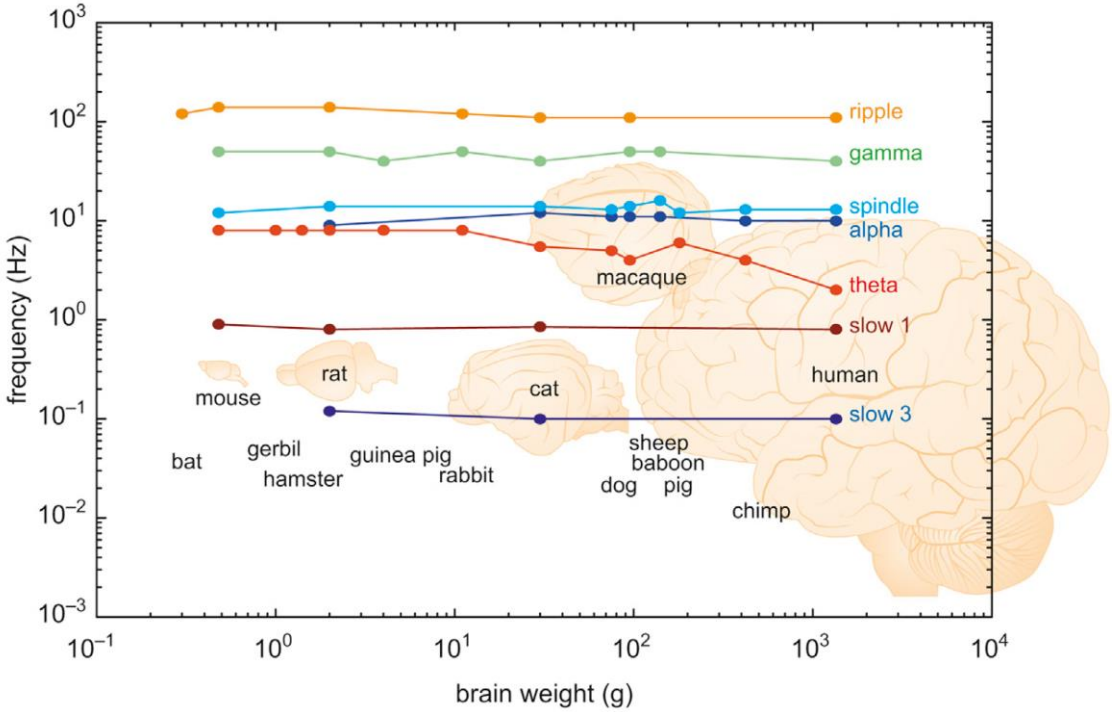
nature neuroscience

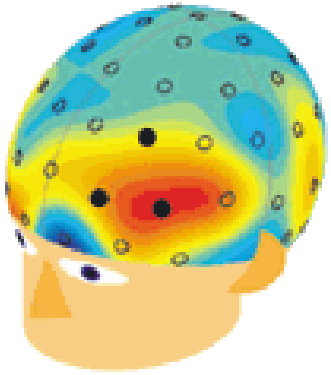
Cortical oscillations and speech processing: emerging computational principles and operations

Anne-Lise Giraud[1] & David Poeppel[2]



- Brain rhythms are primitive components of brain function
- (Ideally) They allow to understand (not just to map) the brain specialization for language
- They connect to:
  - Aspects of human biology conserved across species





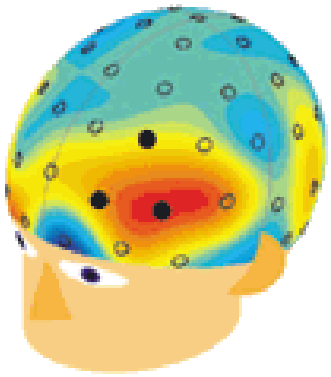
- Brain rhythms are primitive components of brain function
- (Ideally) They allow to understand (not just to map) the brain specialization for language
- They connect to:
  - Aspects of human biology conserved across species

DE GRUYTER MOUTON Theoretical Linguistics 2016; 42(1-2): 117–137

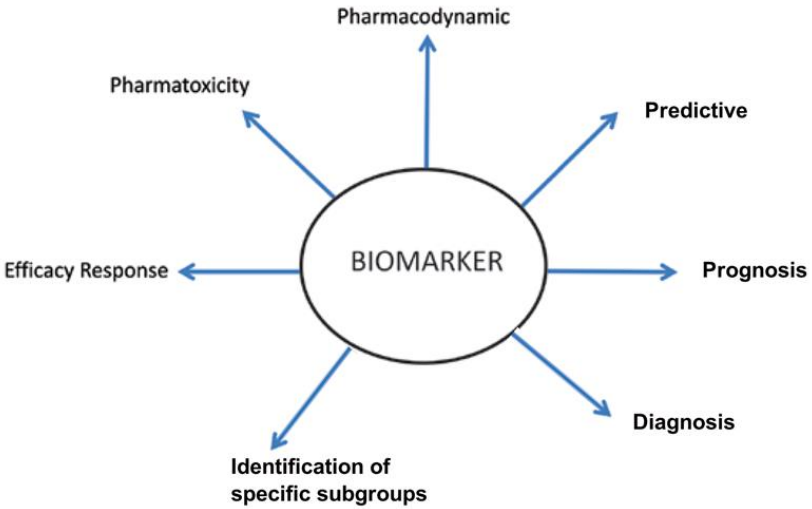
Elliot Murphy*

Evolutionary monkey oscillomics: Generating linking hypotheses from preserved brain rhythms

DOI 10.1515/tl-2016-0005



- Brain rhythms are primitive components of brain function
- (Ideally) They allow to understand (not just to map) the brain specialization for language
- They connect to :
  - Aspects of human biology conserved across species
  - Aspects of human biology known to vary within the species



YALE JOURNAL OF BIOLOGY AND MEDICINE 88 (2015), pp.25-36.
FOCUS: AUTISM SPECTRUM DISORDERS
YJBM

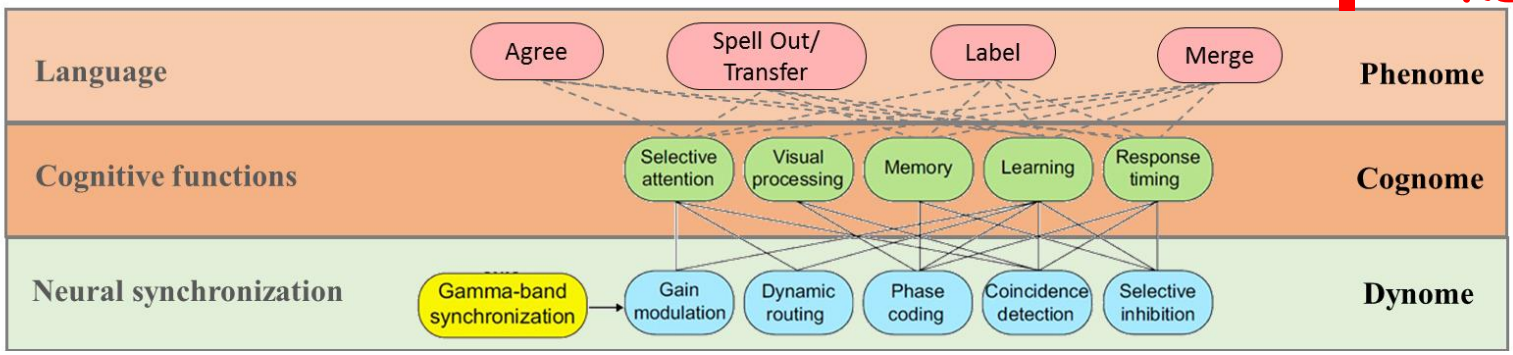
Prospective MEG Biomarkers in ASD: Pre-Clinical Evidence and Clinical Promise of Electrophysiological Signatures

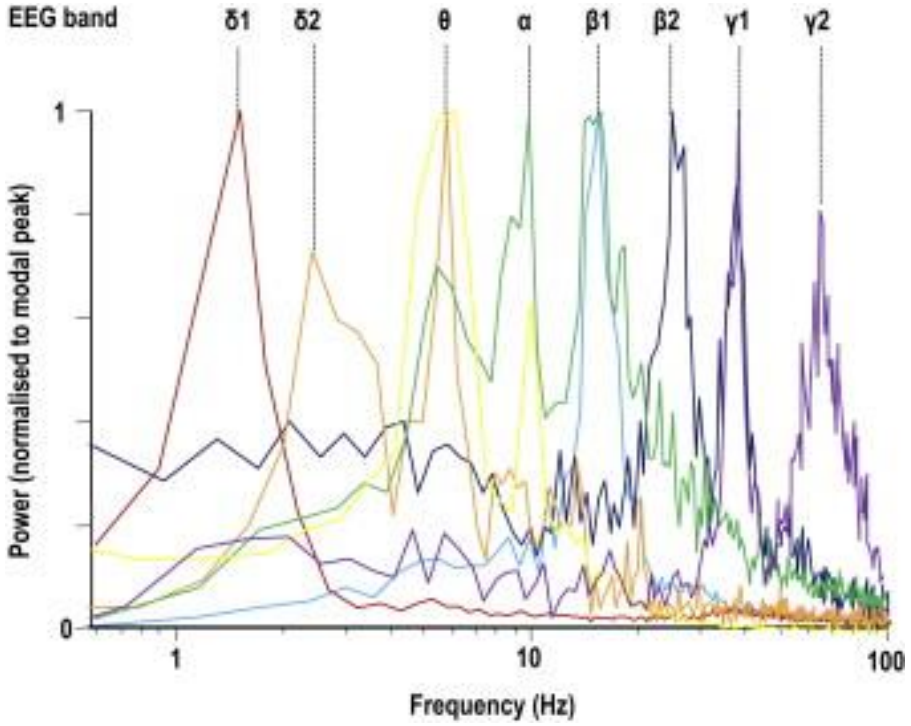
Russell G. Port[a,b,c], Ayesha R. Anwar[b], Matthew Ku[b], Gregory C. Carlson[a,c], Steven J. Siegel[a,c,d], and Timothy P.L. Roberts[b,c*]

[a]Department of Psychiatry, Perelman School of Medicine, University of Pennsylvania, [b]Lurie Family Foundations MEG Imaging Center, Department of Radiology, The Children's Hospital of Philadelphia, [c]Neurosciences Graduate Group, University of Pennsylvania, and [d]Bioengineering Graduate Group, University of Pennsylvania, Philadelphia, Pennsylvania

Table 1. Electrophysiological signatures of ASD and their potential for biomarker use.

Biomarker	Simple to Implement	Sensitive	Specific	Scale	Responsive to Treatment	Biological Basis	Predictive
M50/100 Latency Delay	✓	✓	✓	✓	✓ (pre-clinical)	✓ (hypothesized)	untested
MMF/N	✓	✓	to RDoC domain but not clinical label	✓	untested	less clear	untested
Gamma Band Dysfunction	✓	✓	to RDoC domains, but not clinical label	✓	✓	✓	✓

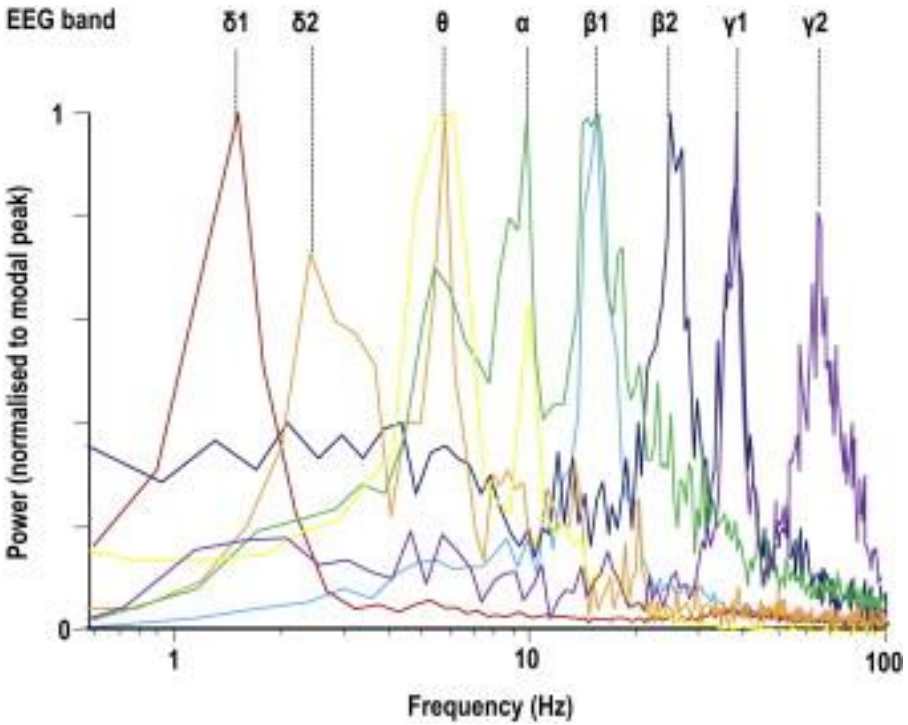

Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome


EEG band
δ1
δ2
θ
α
β1
β2
γ1
γ2
Power (normalised to modal peak)
1
0
1
10
100
Frequency (Hz)

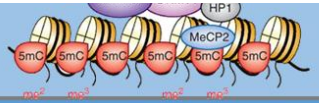
Neural synchronization

Gamma-band synchronization → Gain modulation | Dynamic routing | Phase coding | Coincidence detection | Selective inhibition

Dynome



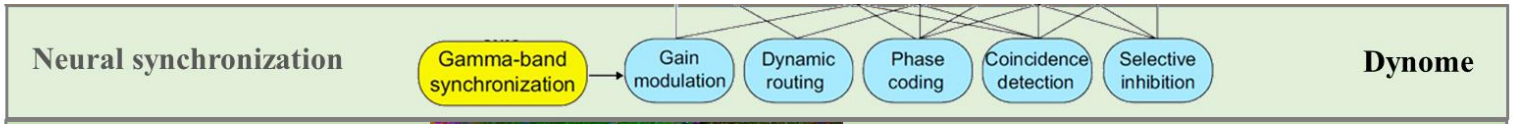
Epigenetic modifications



Epigenome

Genes *CLOCK CNTNAP2 CNTNNB1 DISC1 DLX1 DLX5 DLX6 EGFR FMR1 FOXP1 MECP2 MET NCAM1 NRG1 NRG3 POU3F2 RELN ROBO2 GFB1*

Genome



Behavioural Brain Research 329 (2017) 84–95

Contents lists available at ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Review

An oscillopathic approach to developmental dyslexia: From genes to speech processing

Miguel Jiménez-Bravo[a], Victoria Marrero[a], Antonio Benítez-Burraco[b,*]

[a] Department of Philology, UNED, Madrid, Spain
[b] Department of Philology, University of Huelva, Huelva, Spain

frontiers in Human Neuroscience

HYPOTHESIS AND THEORY
published: 23 August 2016
doi: 10.3389/fnhum.2016.00422

Bridging the Gap between Genes and Language Deficits in Schizophrenia: An Oscillopathic Approach

Elliot Murphy[1] and Antonio Benítez-Burraco[2]*

[1] Division of Psychology and Language Sciences, University College London, London, UK, [2] Department of Philology, University of Huelva, Huelva, Spain

Journal of Neurolinguistics 67 (2023) 101139

Contents lists available at ScienceDirect

Journal of Neurolinguistics

journal homepage: www.elsevier.com/locate/jneuroling

Language deficits in *GRIN2A* mutations and Landau–Kleffner syndrome as neural dysrhythmias

Antonio Benítez-Burraco[a,*], Koji Hoshi[b], Elliot Murphy[c,d]

[a] Department of Spanish, Linguistics, And Theory of Literature (Linguistics), Faculty of Philology, University of Seville, Seville, Spain
[b] Faculty of Economics, Keio University, Tokyo, Japan
[c] Vivian L. Smith Department of Neurosurgery, McGovern Medical School, University of Texas Health Science Center at Houston, TX, USA
[d] Texas Institute for Restorative Neurotechnologies, University of Texas Health Science Center at Houston, TX, USA

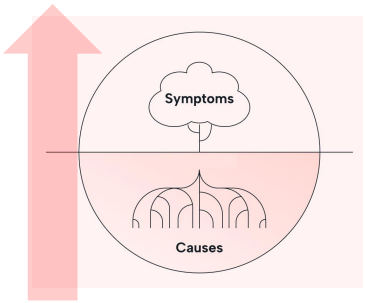
n Neuroscience

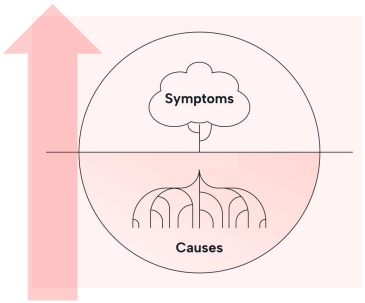
HYPOTHESIS AND THEORY
published: 18 March 2016
doi: 10.3389/fnhum.2016.00120

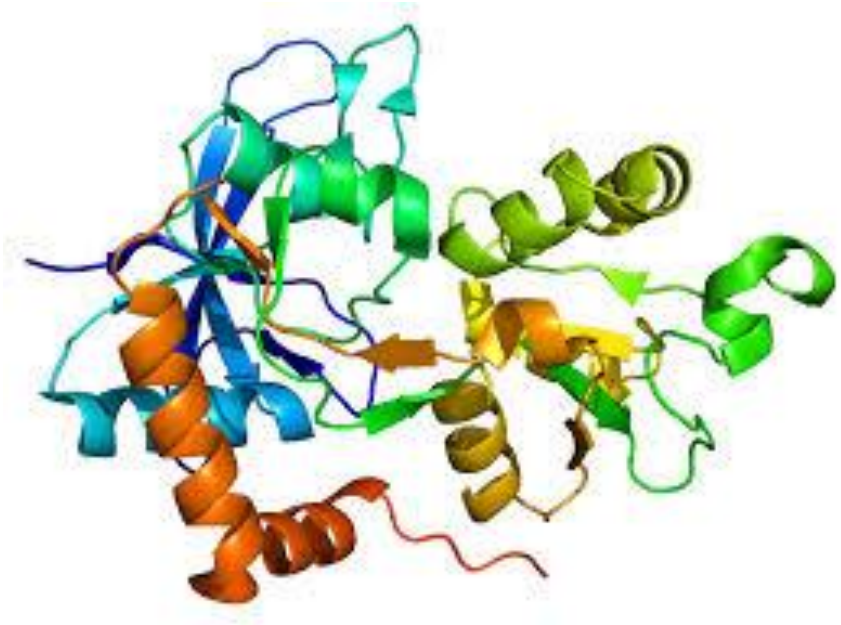
The Oscillopathic Nature of Language Deficits in Autism: From Genes to Language Evolution

*Antonio Benítez-Burraco[1] and Elliot Murphy[2]**

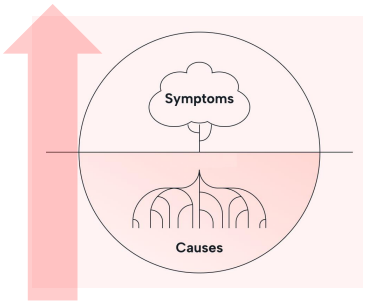
[1] Department of Spanish Philology and its Didactis, University of Huelva, Huelva, Spain, [2] Division of Psychology and Language Sciences, University College London, London, UK

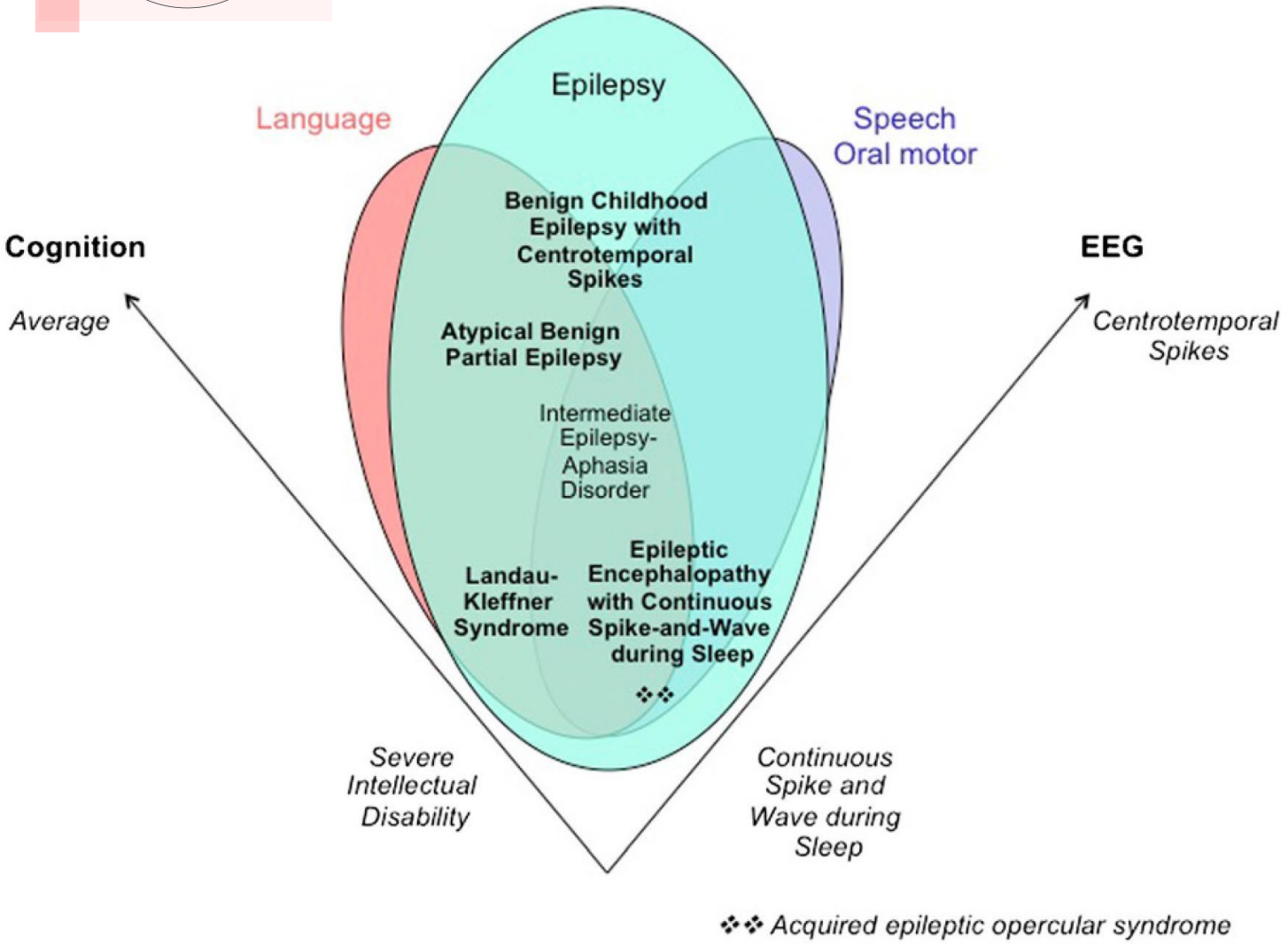

Symptoms
Causes

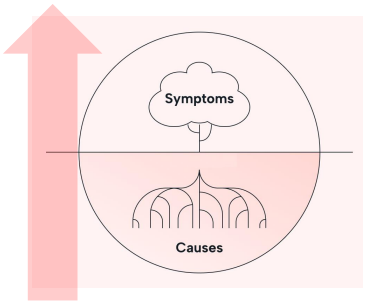




GRIN2A


Symptoms
Causes

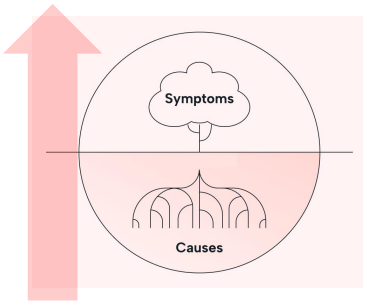

Epilepsy
Language
Speech
Oral motor
Benign Childhood Epilepsy with Centrotemporal Spikes
Atypical Benign Partial Epilepsy
Intermediate Epilepsy-Aphasia Disorder
Landau-Kleffner Syndrome
Epileptic Encephalopathy with Continuous Spike-and-Wave during Sleep
❖❖
Cognition
Average
Severe Intellectual Disability
EEG
Centrotemporal Spikes
Continuous Spike and Wave during Sleep
❖❖ Acquired epileptic opercular syndrome



Language

Speech
Oral motor

- dysarthria
- dyspraxia
- dysphasia
- aphasia / language regression
- phonological problems
- moderate-to-severe receptive language impairment



Journal of Neurolinguistics 67 (2023) 101139

Contents lists available at ScienceDirect

Journal of Neurolinguistics

journal homepage: www.elsevier.com/locate/jneuroling

Language deficits in *GRIN2A* mutations and Landau–Kleffner syndrome as neural dysrhythmias

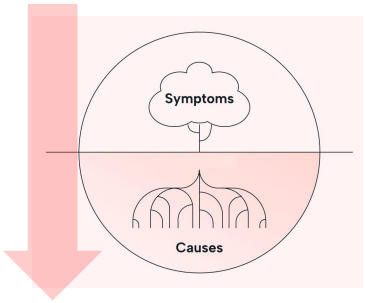
Antonio Benítez-Burraco [a,*], Koji Hoshi [b], Elliot Murphy [c,d]

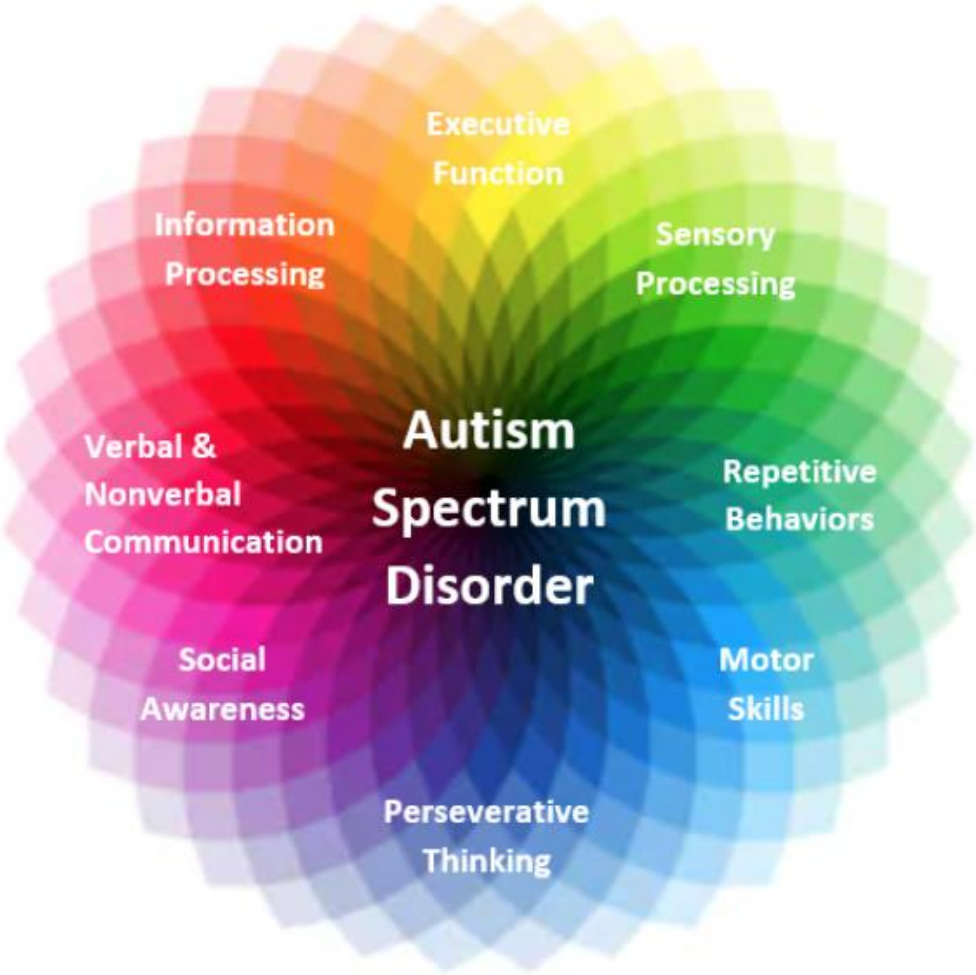
[a] Department of Spanish, Linguistics, And Theory of Literature (Linguistics), Faculty of Philology, University of Seville, Seville, Spain
[b] Faculty of Economics, Keio University, Tokyo, Japan
[c] Vivian L. Smith Department of Neurosurgery, McGovern Medical School, University of Texas Health Science Center at Houston, TX, USA
[d] Texas Institute for Restorative Neurotechnologies, University of Texas Health Science Center at Houston, TX, USA

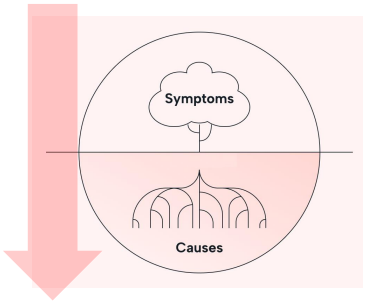
❽ **Phenome**	Errors in articulation, spelling, and reading, and problems with pitch and prosody
❼ **Cognome**	Anomalous processing of fast-rate phonemic and syllabic information
❻ **Dynome**	Abnormal γ activity in the auditory cortex
❺ **Cytome** ❹ **Toponome/ Organome**	Reduced number of functional NMDA receptors in fast-firing interneurons of the auditory cortex
❸ **Proteome**	Reduced amount of GRIN2A in the auditory cortex
❷ **Transcriptome**	Reduced expression of *GRIN2A* in the auditory cortex
❶ **Genome**	Mutations in *GRIN2A*

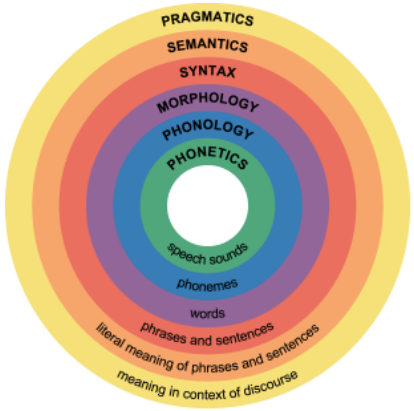
Motivated layers of biological complexity relevant for language

Expected gene-oscillations-language associations for *GRIN2A*


Symptoms
Causes


Executive Function
Information Processing
Sensory Processing
Autism Spectrum Disorder
Verbal & Nonverbal Communication
Repetitive Behaviors
Social Awareness
Motor Skills
Perseverative Thinking



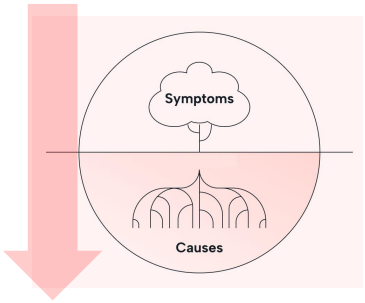


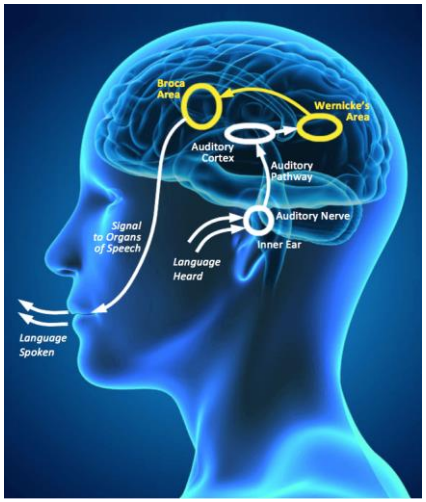
Problems with structural aspects of language

- phonological problems
- morphosyntactic problems
  - clitics
  - relative clauses
  - wh-questions
  - passives
  - embedded clauses

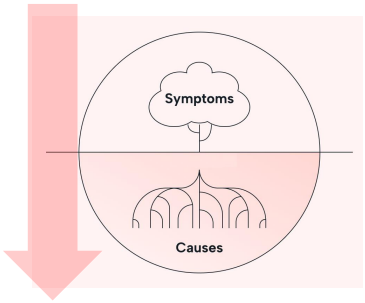
Problems with pragmatics

- difficulties for understanding non-literal language
- atypical conversational exchanges

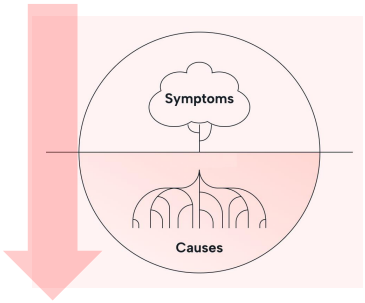




- impaired semantic knowledge and sematic integration during sentence processing
- greater reliance on auditory/phonological cues + reduced reliance on semantics during word learning
- deficit in phonological processing

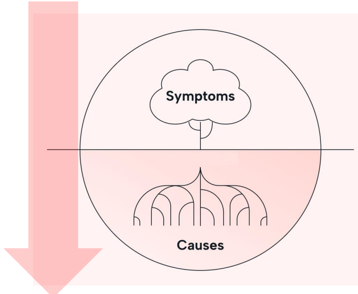


- increased variability
- delays, asynchronies, and/or deviances

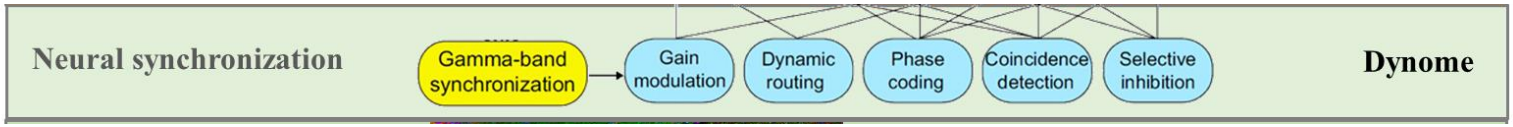


- altered Mental Time Travel abilities
- impaired capacity for cross-modal thinking
- altered Theory of Mind
- increased reliance on rules

KEIO/1


Symptoms
Causes


autism
genetic
abnormalities
daily activities
developmental
restricted behavior
independent
interest
hand flapping
disorder
affected
superior skills
family
factors
repetitive behavior
self-injury
perception
general population
ritualistic behavior
severity
dressing ritual
help
compulsive behavior
relative
health
evidence
attention
symptoms
repetitive
behavior
brain disorders
nervous system
repetitive movement
head rolling
unchanging
menu
communication
activity
focus
limited
occurrence
prodigious
unvarying pattern
Compulsive behavior
hand biting
complex
movements
emotional
childhood
health care
clinical
physically
educational
eye poking
body rocking
resistance to change
systems level
associated
preoccupation
pattern
sensory symptoms
social interaction
impaired toy change
interactions between



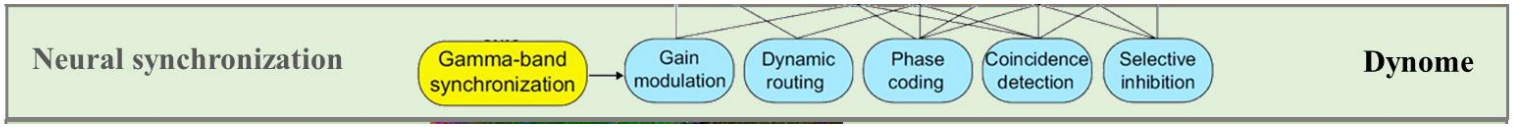
frontiers in Human Neuroscience

HYPOTHESIS AND THEORY
published: 18 March 2016
doi: 10.3389/fnhum.2016.00120

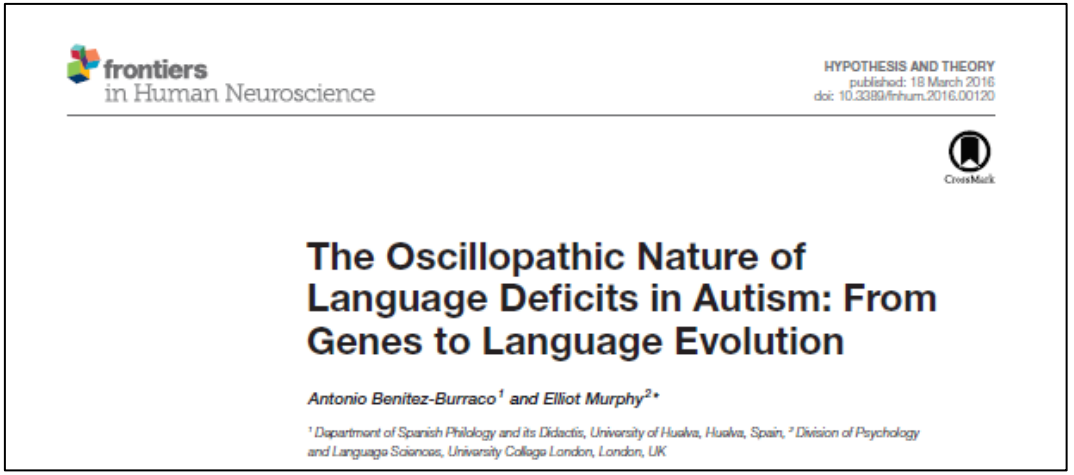
The Oscillopathic Nature of Language Deficits in Autism: From Genes to Language Evolution

Antonio Benítez-Burraco[1] *and Elliot Murphy*[2]*

[1] Department of Spanish Philology and its Didactics, University of Huelva, Huelva, Spain, [2] Division of Psychology and Language Sciences, University College London, London, UK



Frequency band	Role in the present model of language computation	Observed and predicted differences in Autism Spectrum Disorder
Delta (~0.5-4Hz)	Involved in phrasal processing and possibly labeling.	Increased in eyes-closed resting state exam; predicted to be disrupted in processing phrases involving raising and passives.
Theta (~4-10Hz)	Hippocampal source; embeds γ to generate cyclic transfer of syntactic objects; involved more generally in memory retrieval.	Reduced coherence in children; does not synergistically engage with γ during speech; predicted to be disrupted in certain memory retrieval processes.
Alpha (~8-12Hz)	Synchronizes distant cortical regions; embeds γ generated cross-cortically to yield inter-modular set-formation; involved in lexical decision making.	Reduced cross-cortically; reduced resting-state α-γ phase amplitude coupling; increased in resting state; predicted to be disrupted during certain lexicalisations.
Beta (~10-30Hz)	When γ is slowed to β and coupled with α via a basal ganglia-thalamic-cortical loop, syntactic objects are labeled; holds objects in memory.	Reduced cross-frequency coupling with γ; predicted to be disrupted in the maintenance of syntactic objects in raising, passives and *wh*-questions.
Gamma (~30-100Hz)	Generates syntactic objects before β holds them in memory; central role in a number of linguistic operations; involved in lexical processing.	Over-connectivity gives rise to increased γ; reduced in rSTG and lIFG during picture naming; predicted to be disrupted quite generally in linguistic cognition.

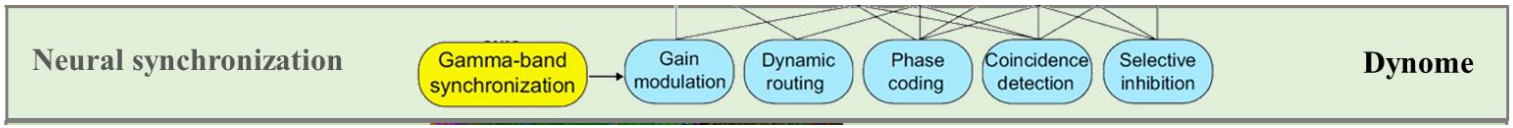

frontiers in Human Neuroscience

HYPOTHESIS AND THEORY
published: 18 March 2016
doi: 10.3389/fnhum.2016.00120

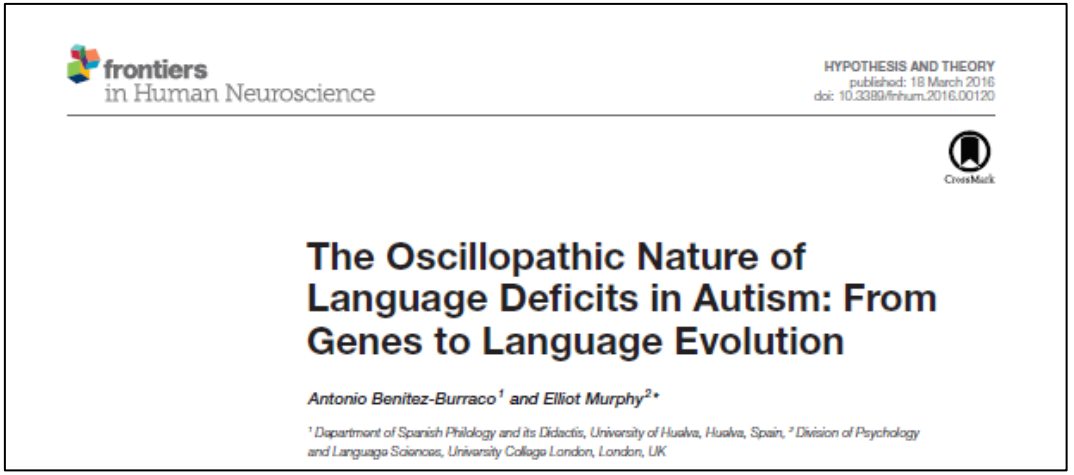
The Oscillopathic Nature of Language Deficits in Autism: From Genes to Language Evolution

Antonio Benítez-Burraco[1] *and Elliot Murphy*[2]*

[1] Department of Spanish Philology and its Didactics, University of Huelva, Huelva, Spain, [2] Division of Psychology and Language Sciences, University College London, London, UK



Frequency band	Role in the present model of language computation	Observed and predicted differences in Autism Spectrum Disorder
Delta (~0.5-4Hz)	Involved in phrasal processing and possibly labeling.	Increased in eyes-closed resting state exam; predicted to be disrupted in processing phrases involving raising and passives.
Theta (~4-10Hz)	Hippocampal source; embeds γ to generate cyclic transfer of syntactic objects; involved more generally in memory retrieval.	Reduced coherence in children; does not synergistically engage with γ during speech; predicted to be disrupted in certain memory retrieval processes.
Alpha (~8-12Hz)	Synchronizes distant cortical regions; embeds γ generated cross-cortically to yield inter-modular set-formation; involved in lexical decision making.	Reduced cross-cortically; reduced resting-state α-γ phase amplitude coupling; increased in resting state; predicted to be disrupted during certain lexicalisations.
Beta (~10-30Hz)	When γ is slowed to β and coupled with α via a basal ganglia-thalamic-cortical loop, syntactic objects are labeled; holds objects in memory.	Reduced cross-frequency coupling with γ; predicted to be disrupted in the maintenance of syntactic objects in raising, passives and *wh*-questions.
Gamma (~30-100Hz)	Generates syntactic objects before β holds them in memory; central role in a number of linguistic operations; involved in lexical processing.	Over-connectivity gives rise to increased γ; reduced in rSTG and lIFG during picture naming; predicted to be disrupted quite generally in linguistic cognition.

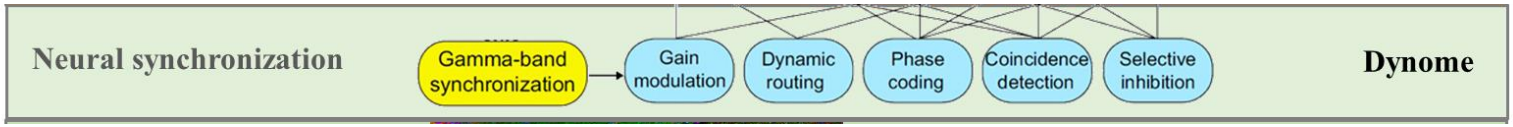

frontiers in Human Neuroscience

HYPOTHESIS AND THEORY
published: 18 March 2016
doi: 10.3389/fnhum.2016.00120

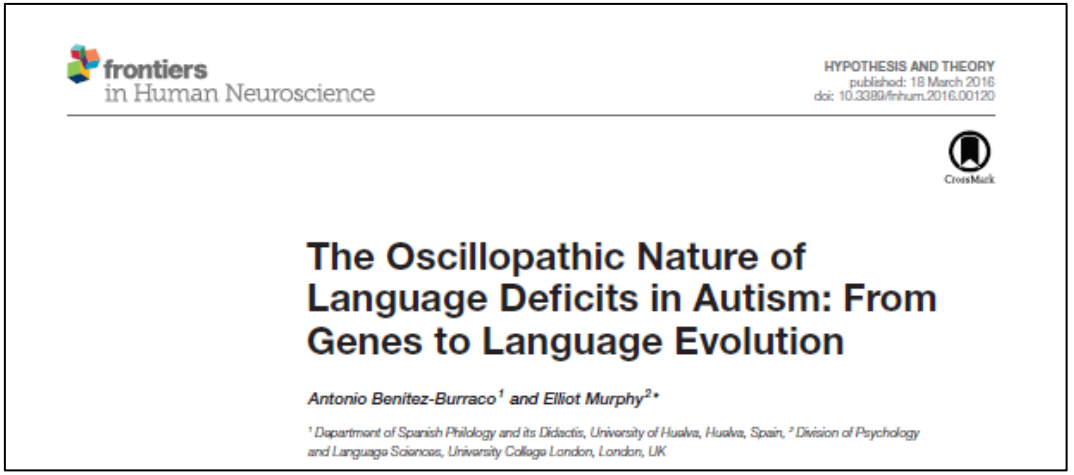
The Oscillopathic Nature of Language Deficits in Autism: From Genes to Language Evolution

Antonio Benítez-Burraco[1] *and Elliot Murphy*[2]*

[1] Department of Spanish Philology and its Didactics, University of Huelva, Huelva, Spain, [2] Division of Psychology and Language Sciences, University College London, London, UK



Frequency band	Role in the present model of language computation	Observed and predicted differences in Autism Spectrum Disorder
Delta (~0.5-4Hz)	Involved in phrasal processing and possibly labeling.	Increased in eyes-closed resting state exam; predicted to be disrupted in processing phrases involving raising and passives.
Theta (~4-10Hz)	Hippocampal source; embeds γ to generate cyclic transfer of syntactic objects; involved more generally in memory retrieval.	Reduced coherence in children; does not synergistically engage with γ during speech; predicted to be disrupted in certain memory retrieval processes.
Alpha (~8-12Hz)	Synchronizes distant cortical regions; embeds γ generated cross-cortically to yield inter-modular set-formation; involved in lexical decision making.	Reduced cross-cortically; reduced resting-state α-γ phase amplitude coupling; increased in resting state; predicted to be disrupted during certain lexicalisations.
Beta (~10-30Hz)	When γ is slowed to β and coupled with α via a basal ganglia-thalamic-cortical loop, syntactic objects are labeled; holds objects in memory.	Reduced cross-frequency coupling with γ; predicted to be disrupted in the maintenance of syntactic objects in raising, passives and *wh*-questions.
Gamma (~30-100Hz)	Generates syntactic objects before β holds them in memory; central role in a number of linguistic operations; involved in lexical processing.	Over-connectivity gives rise to increased γ; reduced in rSTG and lIFG during picture naming; predicted to be disrupted quite generally in linguistic cognition.


frontiers in Human Neuroscience

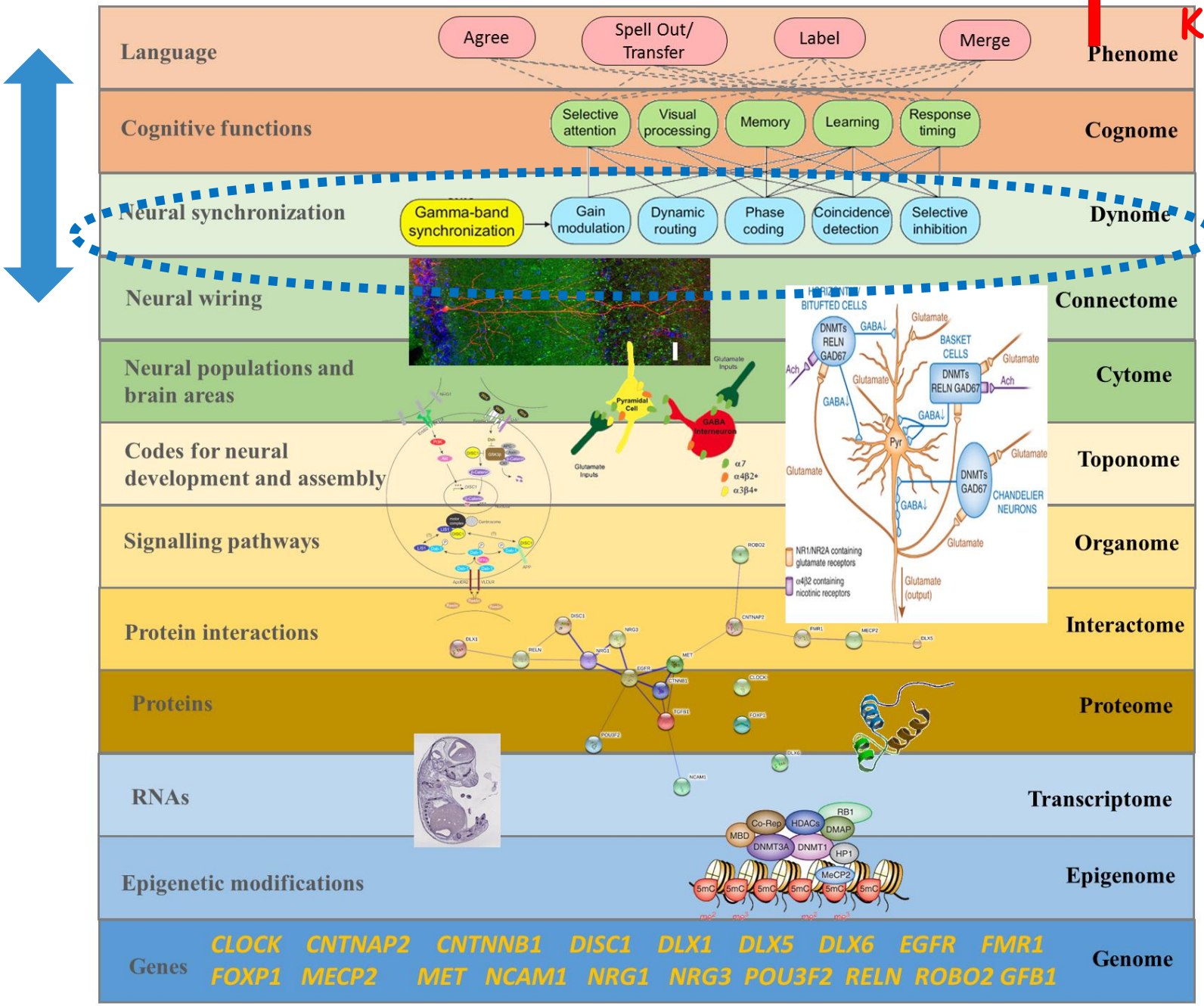
HYPOTHESIS AND THEORY
published: 18 March 2016
doi: 10.3389/fnhum.2016.00120

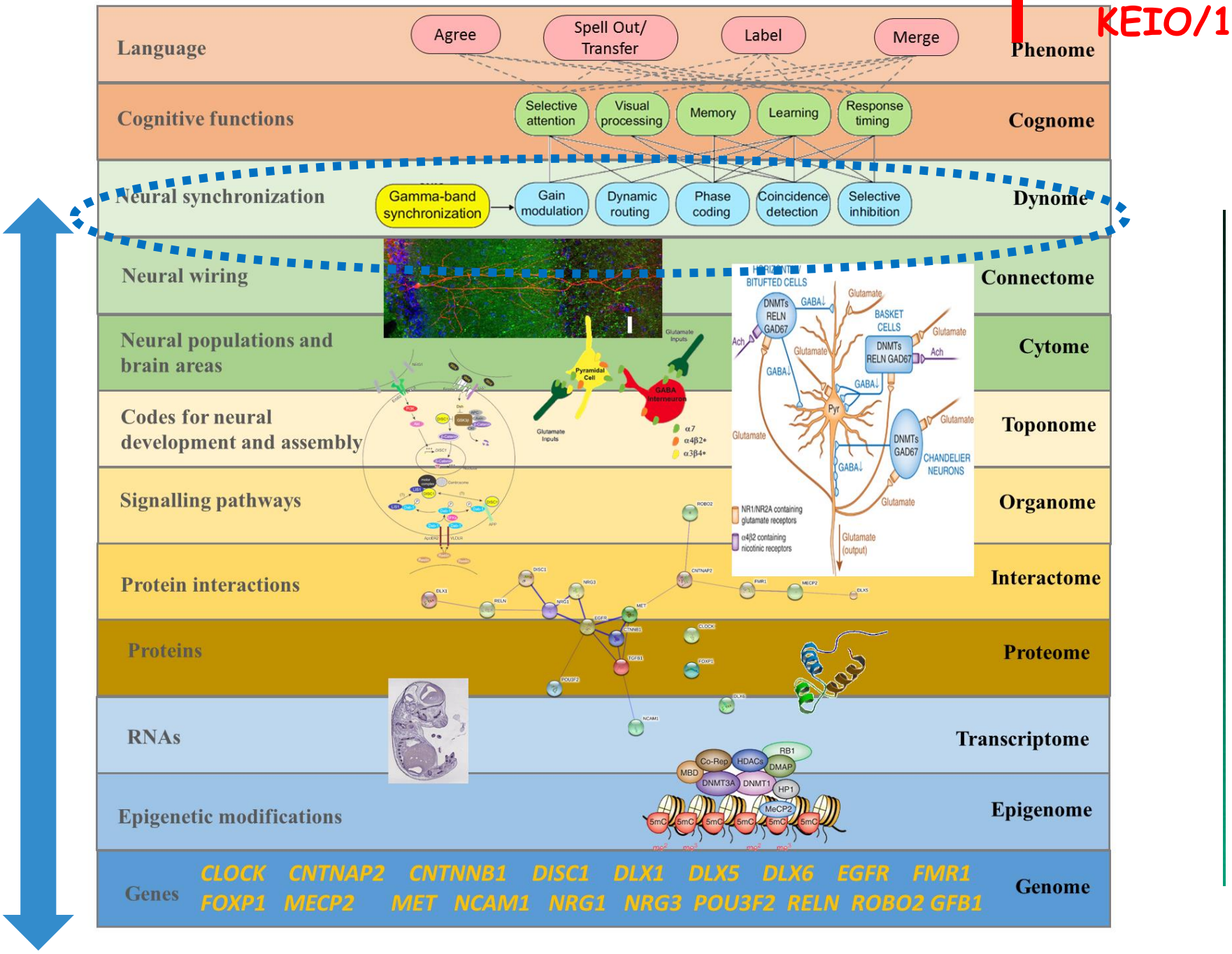
The Oscillopathic Nature of Language Deficits in Autism: From Genes to Language Evolution

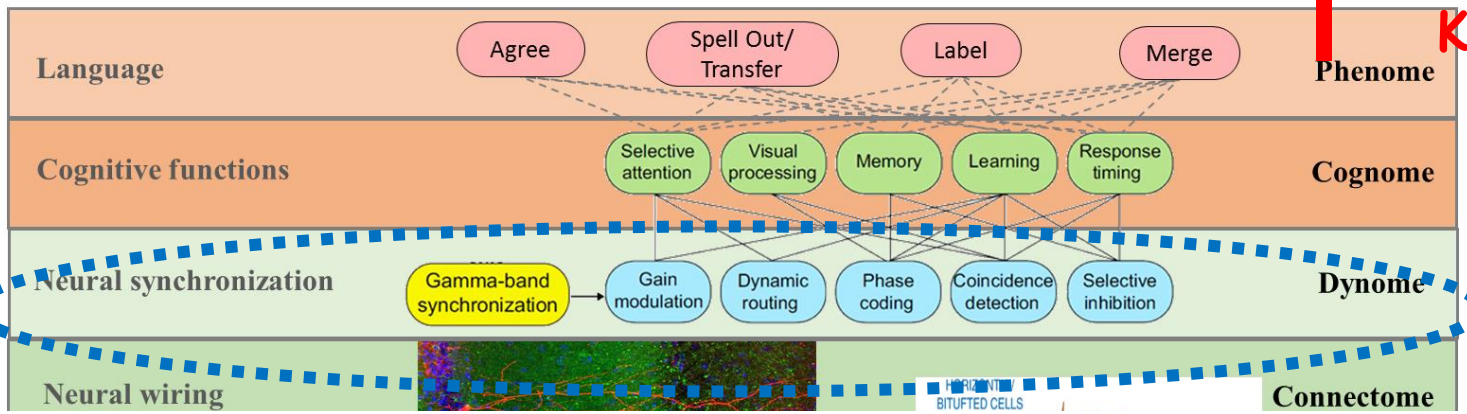
Antonio Benítez-Burraco[1] and Elliot Murphy[2]*

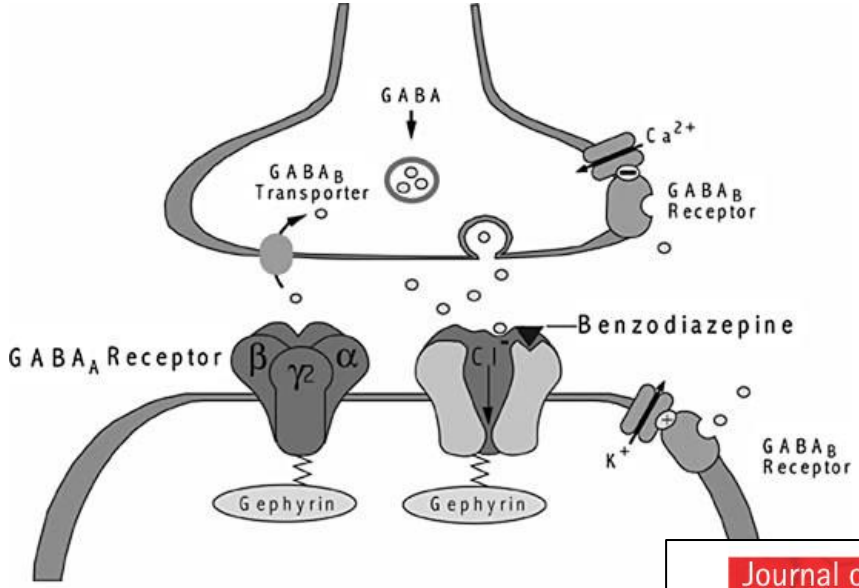
[1] Department of Spanish Philology and its Didactics, University of Huelva, Huelva, Spain, [2] Division of Psychology and Language Sciences, University College London, London, UK

KEIO/1


Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome
Neural wiring
Connectome
Neural populations and brain areas
Pyramidal Cell
GABA interneuron
Glutamate Inputs
α7
α4β2*
α3β4*
Cytome
Codes for neural development and assembly
Toponome
Signalling pathways
Organome
BITUFTED CELLS
DNMTs RELN GAD67
GABA↓
Glutamate
BASKET CELLS
Ach
Pyr
DNMTs GAD67
CHANDELIER NEURONS
NR1/NR2A containing glutamate receptors
α4β2 containing nicotinic receptors
Glutamate (output)
Protein interactions
Interactome
Proteins
Proteome
RNAs
Transcriptome
Epigenetic modifications
Co-Rep
HDACs
RB1
DMAP
MBD
DNMT3A
DNMT1
HP1
MeCP2
5mC
Epigenome
Genes
CLOCK CNTNAP2 CNTNNB1 DISC1 DLX1 DLX5 DLX6 EGFR FMR1
FOXP1 MECP2 MET NCAM1 NRG1 NRG3 POU3F2 RELN ROBO2 GFB1
Genome


Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome
Neural wiring
Connectome
Neural populations and brain areas
Pyramidal Cell
GABA interneuron
Glutamate Inputs
Cytome
Codes for neural development and assembly
Toponome
Signalling pathways
Organome
BITUFTED CELLS
DNMTs RELN GAD67
BASKET CELLS
CHANDELIER NEURONS
Pyr
Glutamate
GABA
Ach
NR1/NR2A containing glutamate receptors
α4β2 containing nicotinic receptors
Protein interactions
Interactome
Proteins
Proteome
RNAs
Transcriptome
Co-Rep
HDACs
RB1
DMAP
MBD
DNMT3A
DNMT1
HP1
MeCP2
5mC
Epigenetic modifications
Epigenome
Genes
CLOCK CNTNAP2 CNTNNB1 DISC1 DLX1 DLX5 DLX6 EGFR FMR1
FOXP1 MECP2 MET NCAM1 NRG1 NRG3 POU3F2 RELN ROBO2 GFB1
Genome





Journal of Neurochemistry
JNC
JOURNAL OF NEUROCHEMISTRY | 2010 | 114 | 1414–1423
doi: 10.1111/j.1471-4159.2010.06858.x

Decreased $GABA_B$ receptors in the cingulate cortex and fusiform gyrus in Autism

Adrian L. Oblak,* Terrell T. Gibbs† and Gene J. Blatt*

*Department of Anatomy and Neurobiology, Laboratory of Autism Neuroscience Research, Boston University School of Medicine, Boston, MA, USA

†Department of Pharmacology and Experimental Therapeutics, Boston University School of Medicine, Boston, MA, USA

Neuroscience & Biobehavioral Reviews

Available online 27 July 2016

In Press, Corrected Proof — Note to users

Review article

Language deficits in schizophrenia and autism as related oscillatory connectomopathies: An evolutionary account

Elliot Murphy[a], Antonio Benítez-Burraco[b]

6

Category fluency

Semantic processing

5

Working memory

chizophrenia

Autism

8

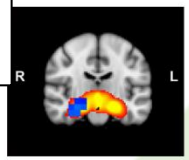
4

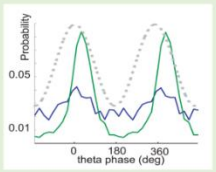
3

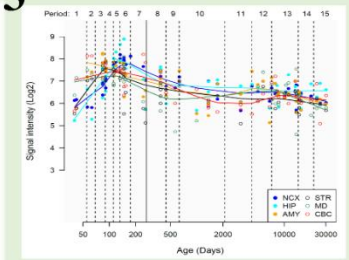
2

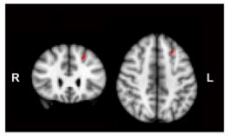
1

b

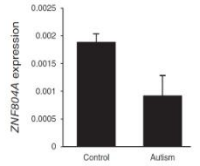




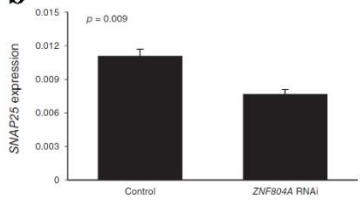




a



b



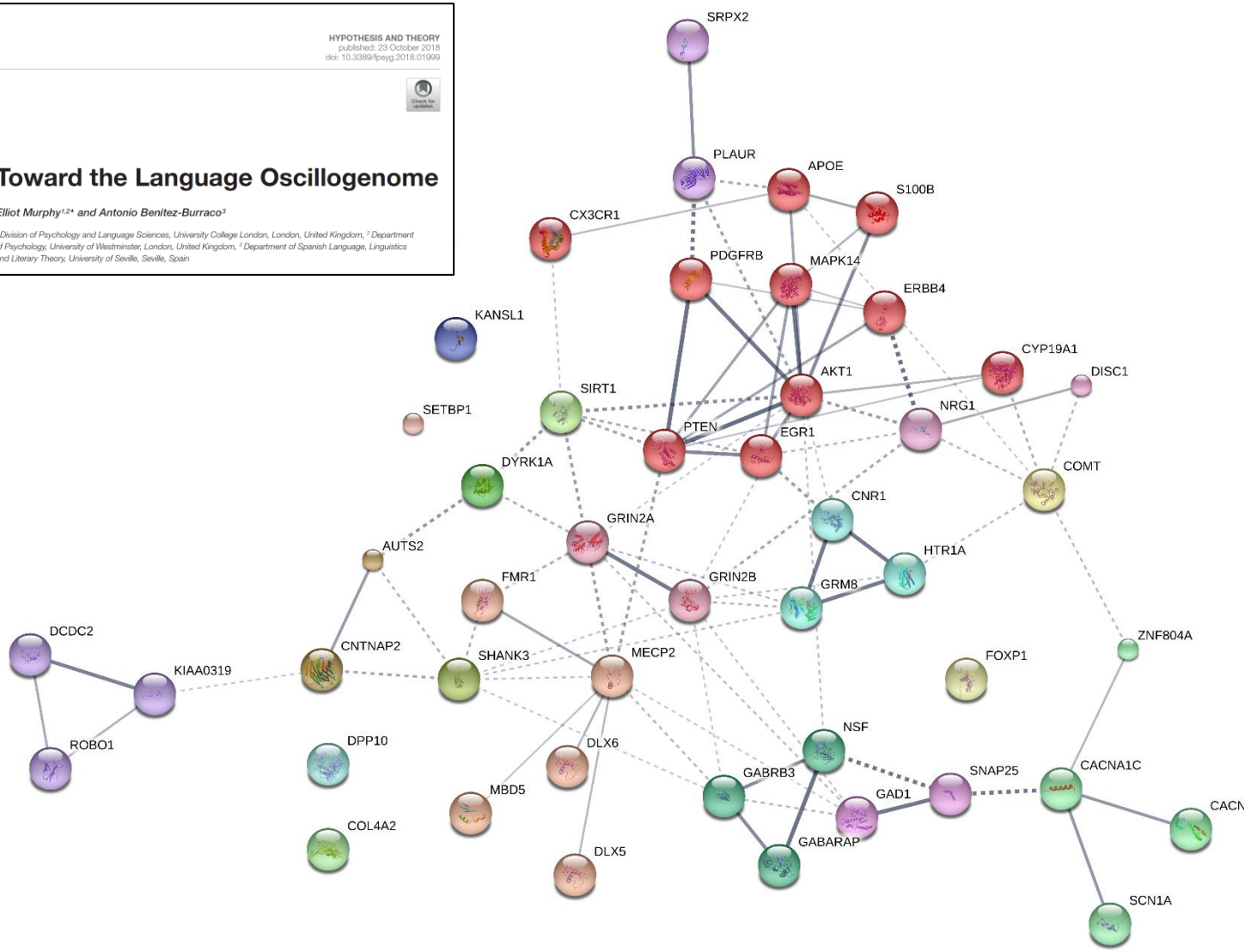
frontiers in Psychology

HYPOTHESIS AND THEORY
published: 23 October 2018
doi: 10.3389/fpsyg.2018.01999

Toward the Language Oscillogenome

Elliot Murphy[1,2]* *and Antonio Benítez-Burraco*[3]

[1] Division of Psychology and Language Sciences, University College London, London, United Kingdom, [2] Department of Psychology, University of Westminster, London, United Kingdom, [3] Department of Spanish Language, Linguistics and Literary Theory, University of Seville, Seville, Spain



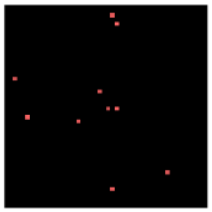
KEIO/1

Upregulation

LO

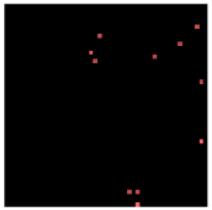
Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	Medial septal nucleus	0.00007973	0.09919	-1.74	4.02
2	Thalamus, sensory-motor cortex related	0.005816	0.2128	-1.95	3.02
3	Agranular insular area, ventral part	0.005816	0.2128	-1.95	3.02
4	island of Calleja major	0.005816	0.2128	-1.94	3.00
5	Agranular insular area, dorsal part, layer 5	0.005816	0.2128	-1.90	2.93
6	intermediate stratum of PalSe	0.0007530	0.2128	-1.90	2.93
7	Bed nuclei of the stria terminalis	0.005816	0.2128	-1.88	2.90
8	intermediate stratum of SePal	0.005816	0.2128	-1.87	2.90
9	superficial stratum of InsCx (cortical plate/marginal zone)	0.005816	0.2128	-1.86	2.89
10	intermediate part of StrSe	0.005816	0.2128	-1.86	2.87

SZ



Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	ventral striatum	0.0001034	0.04531	-1.97	6.10
2	superficial stratum of Str	0.0001034	0.04531	-1.94	6.02
3	Subiculum, dorsal part	0.00003844	0.04531	-1.91	5.90
4	Subiculum, dorsal part, pyramidal layer	0.0002660	0.05301	-1.93	5.66
5	striatal part of olfactory tuberculum	0.0001034	0.04531	-1.77	5.47
6	sublayer 6b of CCx	0.0002660	0.05301	-1.85	5.44
7	mantle zone of isBM	0.0002660	0.05301	-1.83	5.39
8	Midbrain raphe nuclei	0.0006548	0.06834	-1.95	5.24
9	Orbital area, ventrolateral part, layer 5	0.0002660	0.05301	-1.73	5.08
10	corticoid layer of TuStr	0.0006548	0.06834	-1.87	5.02

ASD

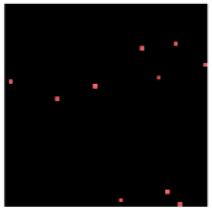


Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	Bed nuclei of the stria terminalis	4.657e-10	3.403e-7	-1.92	28.60
2	Bed nuclei of the stria terminalis, anterior division	1.308e-10	2.867e-7	-1.87	28.24
3	Pallidum, caudal region	4.657e-10	3.403e-7	-1.79	26.63
4	Interanteromedial nucleus of the thalamus	1.763e-8	0.000006440	-1.90	22.67
5	reuniens nucleus, main part	5.569e-8	0.00001221	-1.93	21.89
6	Bed nuclei of the stria terminalis, posterior division, interfascicular nucleus	1.611e-9	8.829e-7	-1.56	21.78
7	lateral posterior nucleus	7.875e-9	0.000003452	-1.64	20.67
8	Nucleus of reunions	1.705e-7	0.00001869	-1.89	20.53
9	intermediate stratum of SePal	1.705e-7	0.00001869	-1.88	20.51
10	septopallidal transition area	1.705e-7	0.00001869	-1.88	20.51

DD

Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	liminal part of alar p1	0.004205	0.2245	-2.22	3.31
2	ventrolateral part of alar p1	0.004205	0.2245	-1.93	2.88
3	Striatum dorsal region	0.04278	0.2347	-1.91	2.77
4	Submedial nucleus of the thalamus	0.04278	0.2347	-1.91	2.77
5	Lateral septal nucleus	0.04278	0.2347	-1.91	2.76
6	r9 part of paragigantocellular nucleus	0.04278	0.2347	-1.91	2.76
7	r10 part of basomedial reticular formation	0.04278	0.2347	-1.90	2.76
8	Hippocampal region	0.004205	0.2245	-1.84	2.75
9	Central medial nucleus of the thalamus	0.04278	0.2347	-1.89	2.75
10	superficial stratum of r7BM	0.04278	0.2347	-1.89	2.73

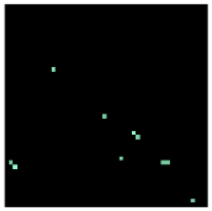
SLI



Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	mantle zone of isBL	0.001634	0.1280	-1.96	4.02
2	mantle zone of m2B	0.001634	0.1280	-1.94	3.99
3	substantia innominata/basal nucleus	0.001634	0.1280	-1.82	3.74
4	liminal alar domain of m1	0.001634	0.1280	-1.79	3.69
5	accumbens nucleus, shell domain	0.001634	0.1280	-1.77	3.64
6	ventrolateral part of alar m1	0.001634	0.1280	-1.76	3.61
7	intermediate stratum of SeStr	0.001634	0.1280	-1.74	3.57
8	m1Lim (VL) part of the periaqueductal gray	0.001634	0.1280	-1.71	3.51
9	periventricular stratum of m1Lim	0.001634	0.1280	-1.68	3.46
10	Dorsal nucleus raphe	0.001634	0.1280	-1.62	3.32

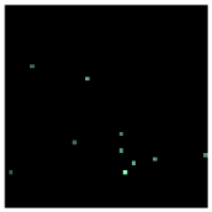
Downregulation

LO



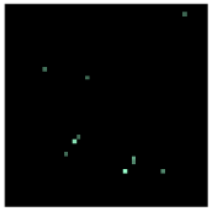
Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	cerebellar nuclei of CbV	0.000006926	0.002135	-2.00	12.32
2	periventricular stratum of cerebellar vermis	0.000006926	0.002135	-1.98	12.17
3	white matter of cerebellar vermis	0.000006926	0.002135	-1.81	11.11
4	medial (fastigial) cerebellar nucleus	0.000006926	0.002135	-1.64	10.09
5	intermediate stratum of cerebellar vermis	0.00007828	0.005678	-1.95	10.08
6	Pyramus (VIII), granular layer	0.00007828	0.005678	-1.88	9.71
7	mantle zone of cerebellar vermis	0.00007828	0.005678	-1.87	9.68
8	cerebellar vermis	0.00007828	0.005678	-1.87	9.67
9	Medulla	0.00007828	0.005678	-1.86	9.64
10	Nodulus (X), molecular layer	0.00007828	0.005678	-1.86	9.61

SZ



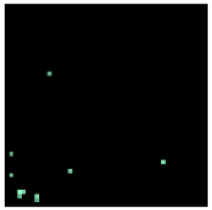
Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	rhombomere 3	0.000001444	0.003166	-1.93	11.13
2	rhombomere 2	0.00001293	0.01417	-1.75	7.46
3	r5 alar plate	0.00003639	0.01595	-1.79	7.39
4	intermediate stratum of r1Ve	0.00003639	0.01595	-1.62	6.71
5	r1 part of superior vestibular nucleus	0.00003639	0.01595	-1.62	6.70
6	Cerebellar nuclei	0.00009818	0.03074	-1.79	6.22
7	mantle zone of r1Ve	0.00009818	0.03074	-1.69	5.89
8	pontine hindbrain	0.0002536	0.06177	-1.71	4.77
9	r1 part of vestibular sensory column	0.0002536	0.06177	-1.56	4.36
10	intermediate (interpositus) cerebellar nucleus	0.001477	0.1245	-1.94	4.03

ASD



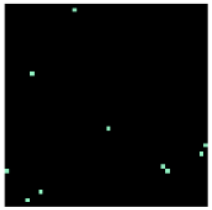
Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	rhombomere 3	3.309e-18	3.626e-15	-1.93	64.25
2	pontine hindbrain	3.309e-18	3.626e-15	-1.73	57.44
3	rhombomere 4	5.446e-13	3.979e-10	-1.91	41.44
4	r6 part of the cochlear column	3.203e-11	1.170e-8	-1.87	34.07
5	rhombomere 2	8.465e-12	4.639e-9	-1.75	33.57
6	mantle zone of r6Co	3.203e-11	1.170e-8	-1.57	28.68
7	r2 part of superior vestibular nucleus	4.931e-9	8.314e-7	-1.95	27.32
8	mantle zone of r5Co	4.931e-9	8.314e-7	-1.92	26.91
9	r5 alar plate	1.463e-9	4.008e-7	-1.78	26.19
10	r3 alar plate	4.931e-9	8.314e-7	-1.84	25.79

DD



Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	Cerebrum	0.2830	0.2922	-4.03	4.96
2	Nodulus (X), molecular layer	0.0002932	0.09780	-1.87	4.34
3	alar plate of evaginated telencephalic vesicle	0.2830	0.2922	-3.42	4.20
4	secondary prosencephalon	0.2830	0.2922	-3.35	4.12
5	pallium	0.2830	0.2922	-3.11	3.83
6	Nodulus (X)	0.0002932	0.09780	-1.60	3.71
7	Cerebral cortex	0.2830	0.2922	-2.83	3.48
8	Lingula (I)	0.004166	0.1852	-1.99	3.35
9	Cortical plate	0.2830	0.2922	-2.72	3.35
10	Uvula (IX)	0.004166	0.1852	-1.93	3.25

SLI



Index	Name	P-value	Adjusted p-value	Z-score	Combined score
1	Retrosplenial area, ventral part, layer 1	0.001614	0.1147	-1.99	4.31
2	layer 3 of PCx	0.001614	0.1147	-1.96	4.24
3	Frontal pole, cerebral cortex	0.001614	0.1147	-1.92	4.16
4	periventricular stratum of r3Co	0.001614	0.1147	-1.91	4.13
5	r2 part of anteroventral cochlear nucleus	0.001614	0.1147	-1.89	4.09
6	Copula pyramidis, molecular layer	0.001614	0.1147	-1.87	4.05
7	superficial stratum of r2Co	0.001614	0.1147	-1.85	4.01
8	Copula pyramidis	0.001614	0.1147	-1.83	3.97
9	r3 part of dorsal cochlear nucleus	0.001614	0.1147	-1.78	3.86
10	forebrain	0.4021	0.4203	-4.40	3.81

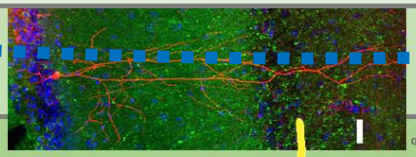
KEIO/1

Language — Agree — Spell Out/ Transfer — Label — Merge — Phenome

Cognitive functions — Selective attention — Visual processing — Memory — Learning — Response timing — Cognome

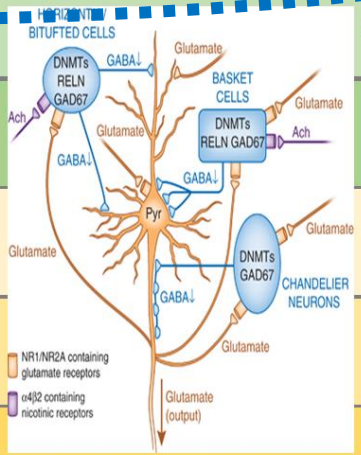
Neural synchronization — Gamma-band synchronization → Gain modulation — Dynamic routing — Phase coding — Coincidence detection — Selective inhibition — Dynome

Neural wiring — Connectome

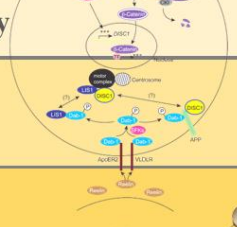


Neural populations and brain areas — Cytome

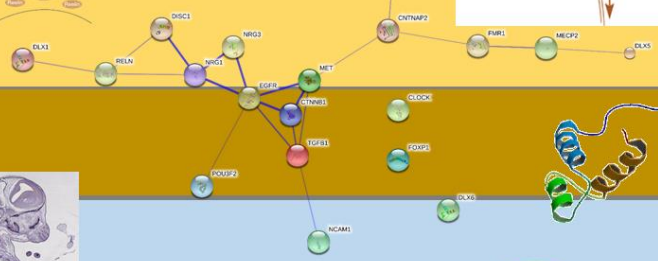
Codes for neural development and assembly — Toponome



Signalling pathways — Organome

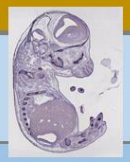


Protein interactions — Interactome

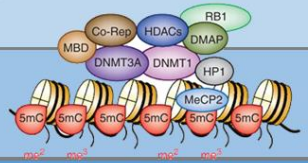


Proteins — Proteome

RNAs — Transcriptome

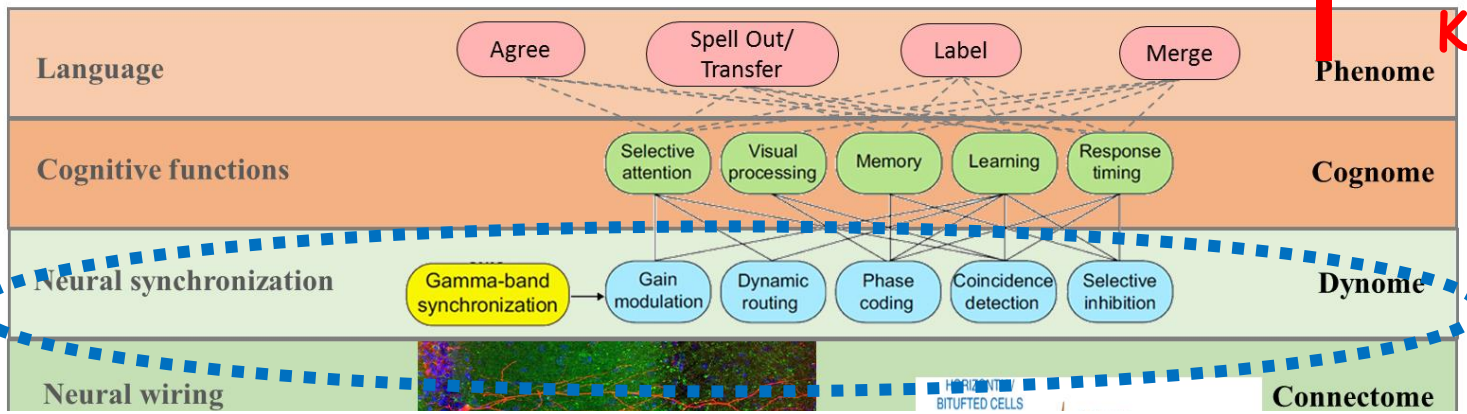


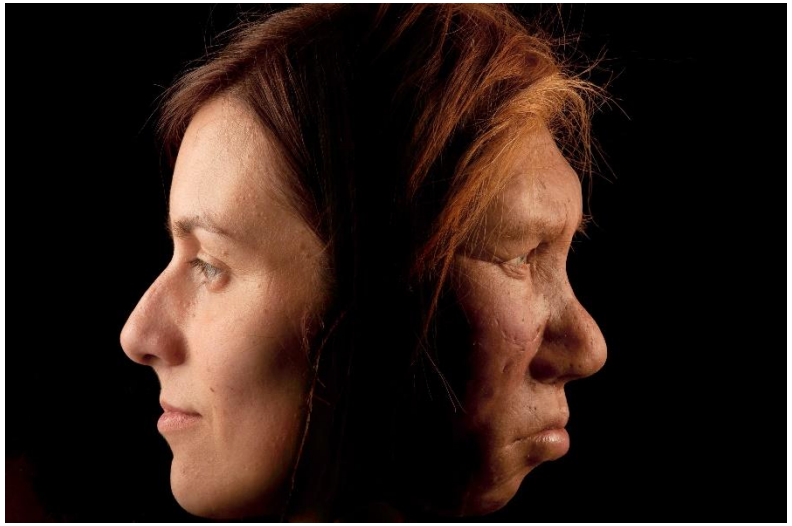
Epigenetic modifications — Epigenome

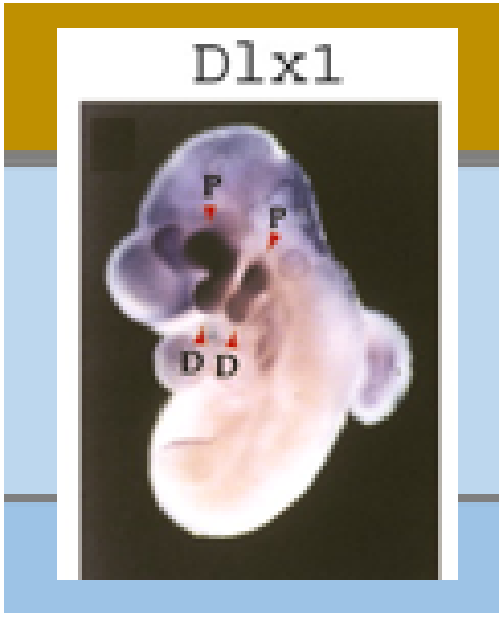


Genes — *CLOCK CNTNAP2 CNTNNB1 DISC1 DLX1 DLX5 DLX6 EGFR FMR1 FOXP1 MECP2 MET NCAM1 NRG1 NRG3 POU3F2 RELN ROBO2 GFB1* — Genome

KEIO/1


Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome
Neural wiring
BITUFTED CELLS
Connectome




Dlx1
P
P
D
D

KEIO/1

Gene	Fixed aa change in AMHs	Positively selected in AMHs	Differentially methylated in AMH skeletal samples	Enriched in AMH DMRs	Oscillomic/oscillopathic features
AUTS2		✓	↑ (body gene)		Epilepsy
CACNA1C			↑ (body gene)		β, γ
CNTNAP2	✓				α
COMT			↑ (downstream the gene)		α
DYRK1A		✓			Inhibition of neural activity
EGR1				✓	Epilepsy
ELP4		✓			High amplitude centrotemporal sharp waves
FMR1	✓				θ, γ
FOXP1		✓	↑ (body gene)		Epileptiform discharges
PDGFRB			↓ (body gene)		γ
ROBO1		✓			Epilepsy
SHANK3			↓ (downstream the gene)		Seizures, abnormal EEG

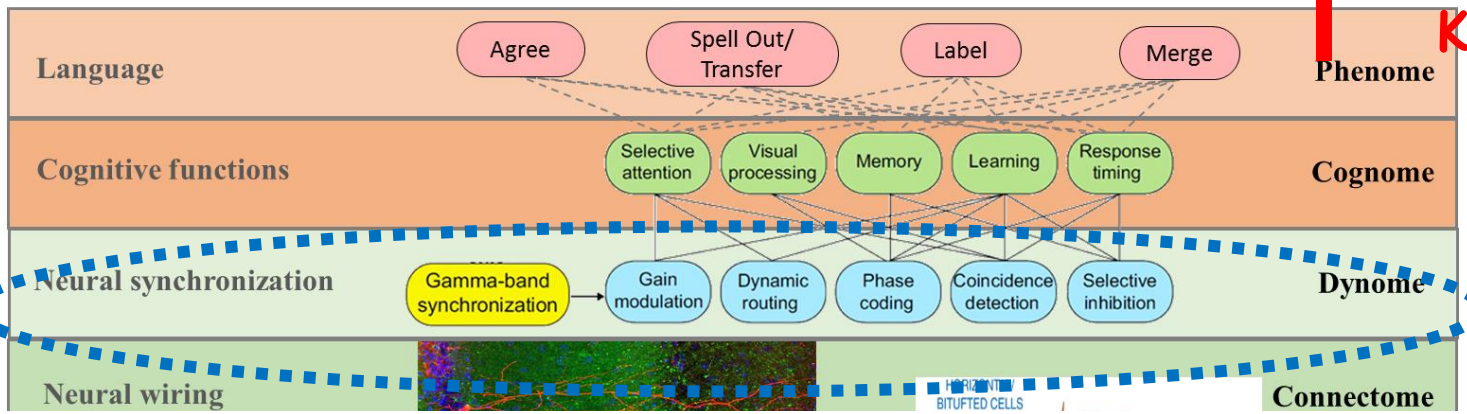
256 memory buffers. However, taken alongside the oscillogenomic evidence presented above – that
257 Neanderthals may have exhibited reduced cross-frequency coupling between θ and γ due to

doi 10.4436/jass.96010

JASs Reports
Journal of Anthropological Sciences
Vol. 96 (2018), pp. 111-124

Paleo-oscillomics: inferring aspects of Neanderthal language abilities from gene regulation of neural oscillations

Elliot Murphy[1,2] & Antonio Benítez-Burraco[3]

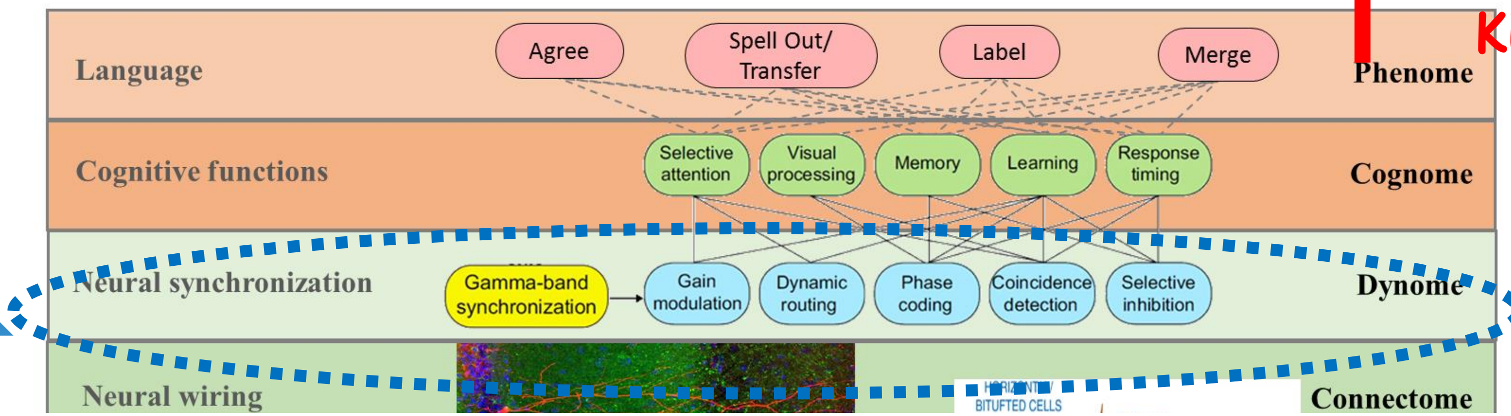


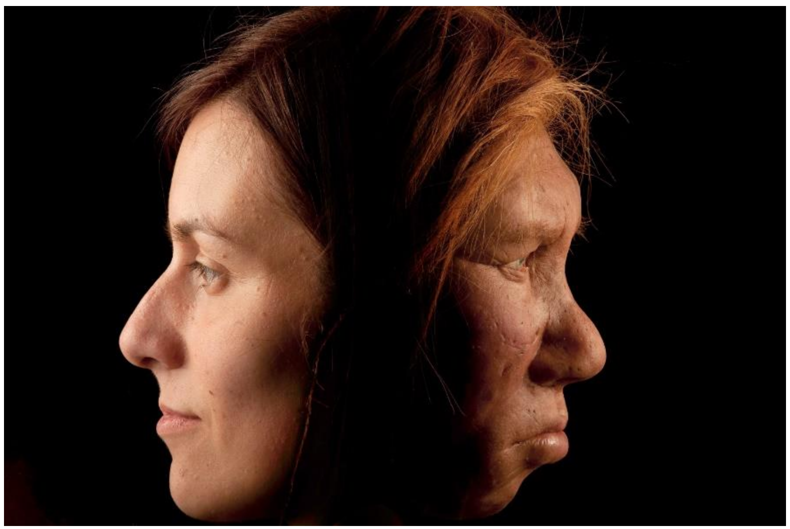
International Journal of Language & Communication Disorders
Discussion

Rigidity in autism spectrum disorder (ASD): A unified (evolutionary) account of linguistic and non-linguistic symptoms

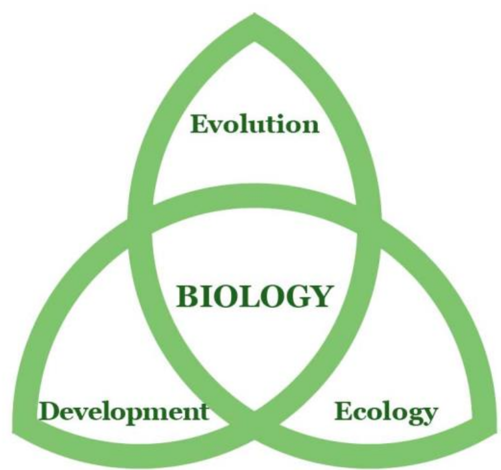
Submission Status Under Review

KEIO/1

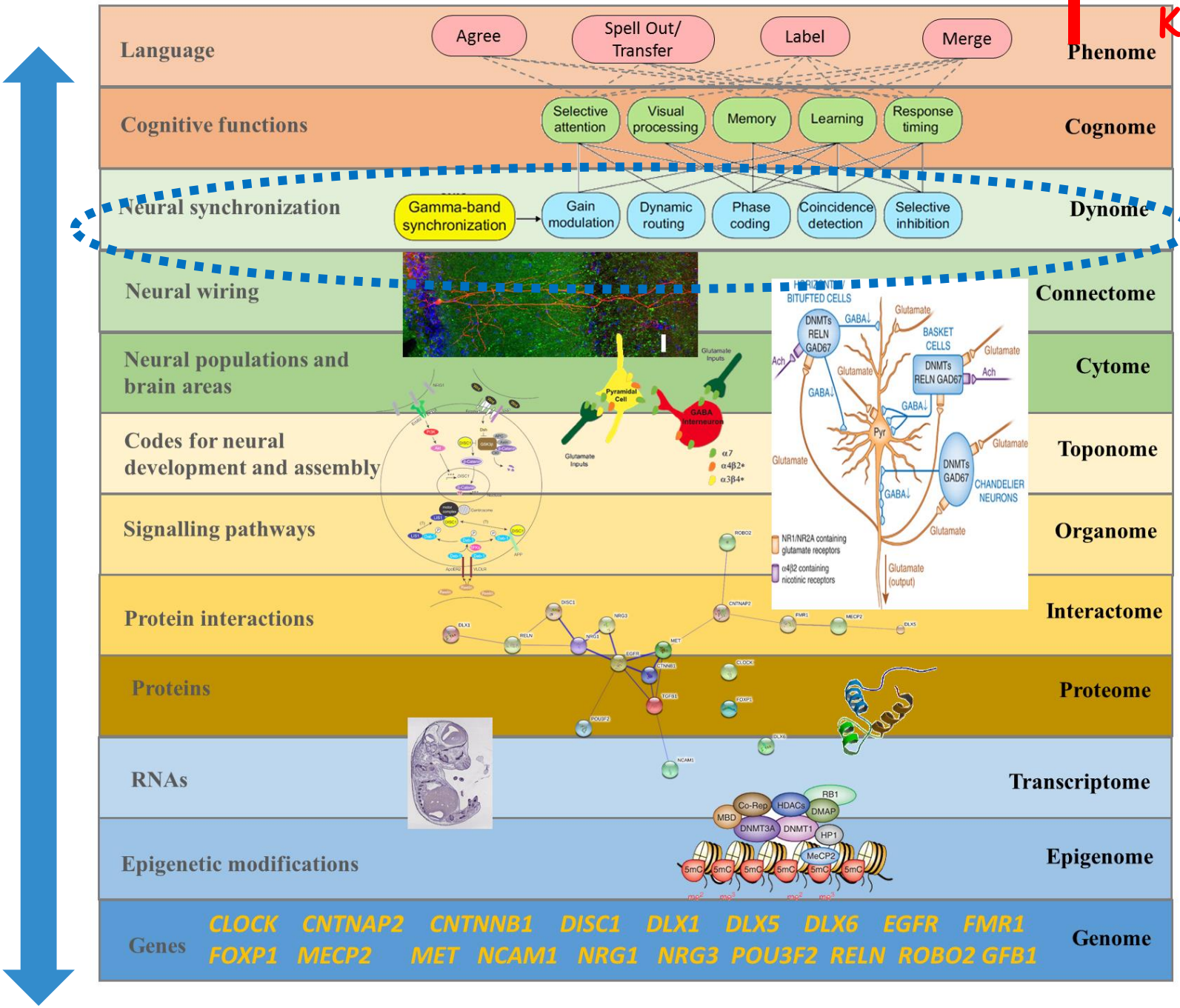

Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome
Neural wiring
Connectome




autism
genetic
daily activities
disorder
behavior
self-injury
interest
perception
dressing ritual
ritualistic behavior
unchanging
head rolling
repetitive movement
limited

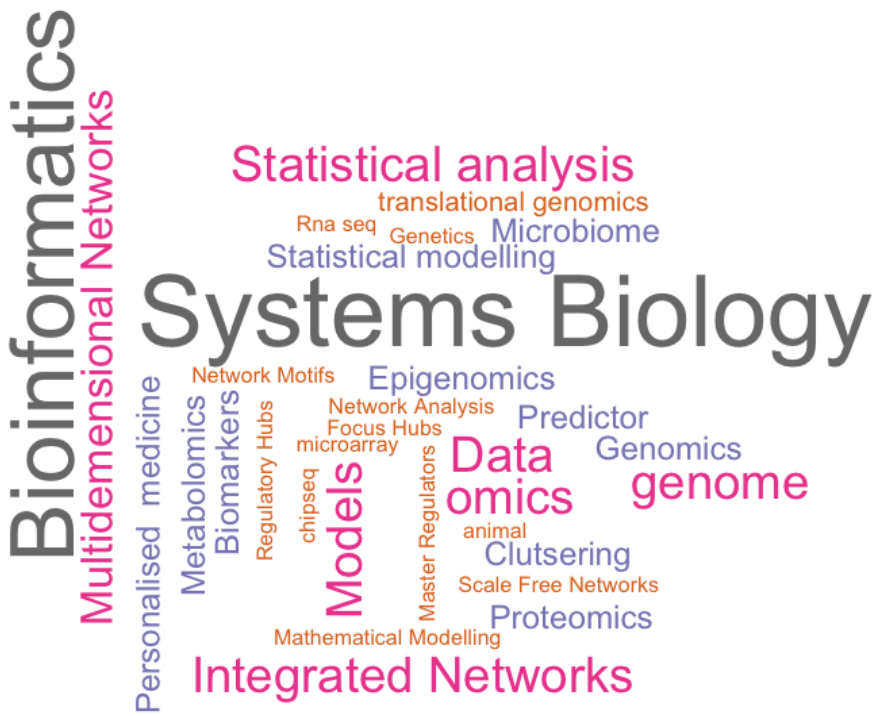

Evolution
BIOLOGY
Development
Ecology

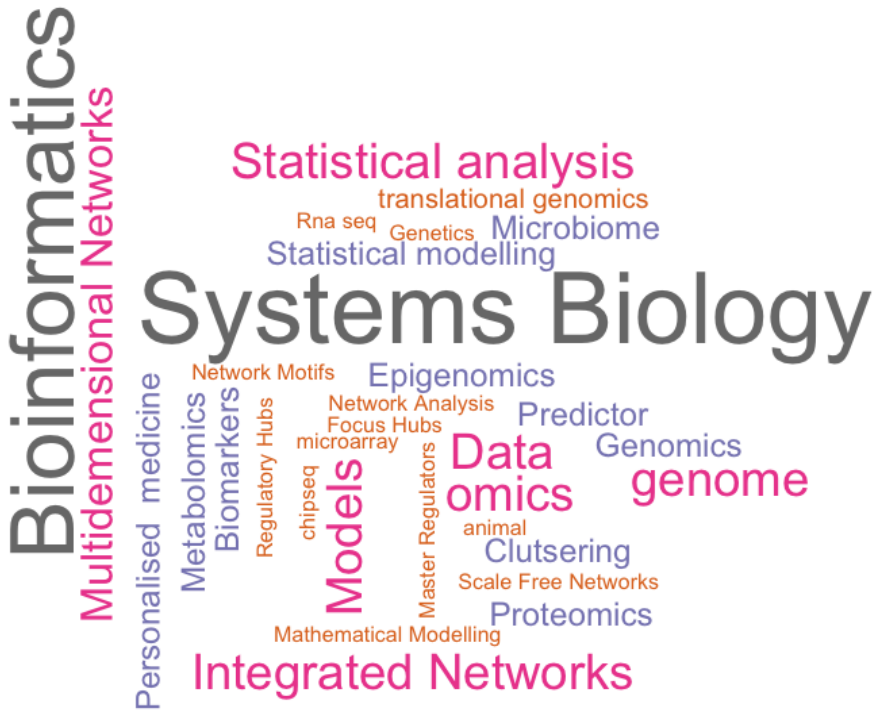
KEIO/1

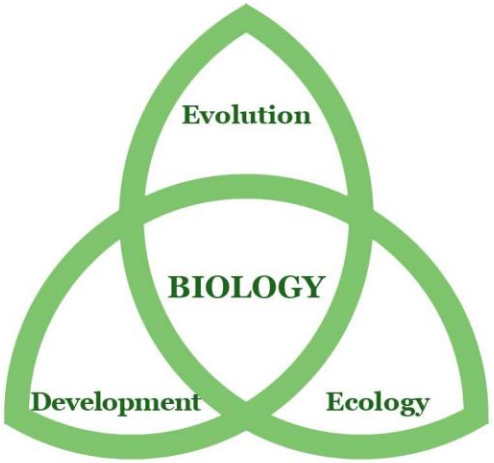

Language
Agree
Spell Out/ Transfer
Label
Merge
Phenome
Cognitive functions
Selective attention
Visual processing
Memory
Learning
Response timing
Cognome
Neural synchronization
Gamma-band synchronization
Gain modulation
Dynamic routing
Phase coding
Coincidence detection
Selective inhibition
Dynome
Neural wiring
Connectome
Neural populations and brain areas
Pyramidal Cell
GABA interneuron
Glutamate Inputs
Cytome
Codes for neural development and assembly
Toponome
Signalling pathways
Organome
BITUFTED CELLS
DNMTs RELN GAD67
BASKET CELLS
CHANDELIER NEURONS
Pyr
Glutamate
GABA
Ach
NR1/NR2A containing glutamate receptors
α4β2 containing nicotinic receptors
Protein interactions
Interactome
Proteins
Proteome
RNAs
Transcriptome
Co-Rep
HDACs
RB1
DMAP
MBD
DNMT3A
DNMT1
HP1
MeCP2
5mC
Epigenetic modifications
Epigenome
Genes
CLOCK CNTNAP2 CNTNNB1 DISC1 DLX1 DLX5 DLX6 EGFR FMR1
FOXP1 MECP2 MET NCAM1 NRG1 NRG3 POU3F2 RELN ROBO2 GFB1
Genome

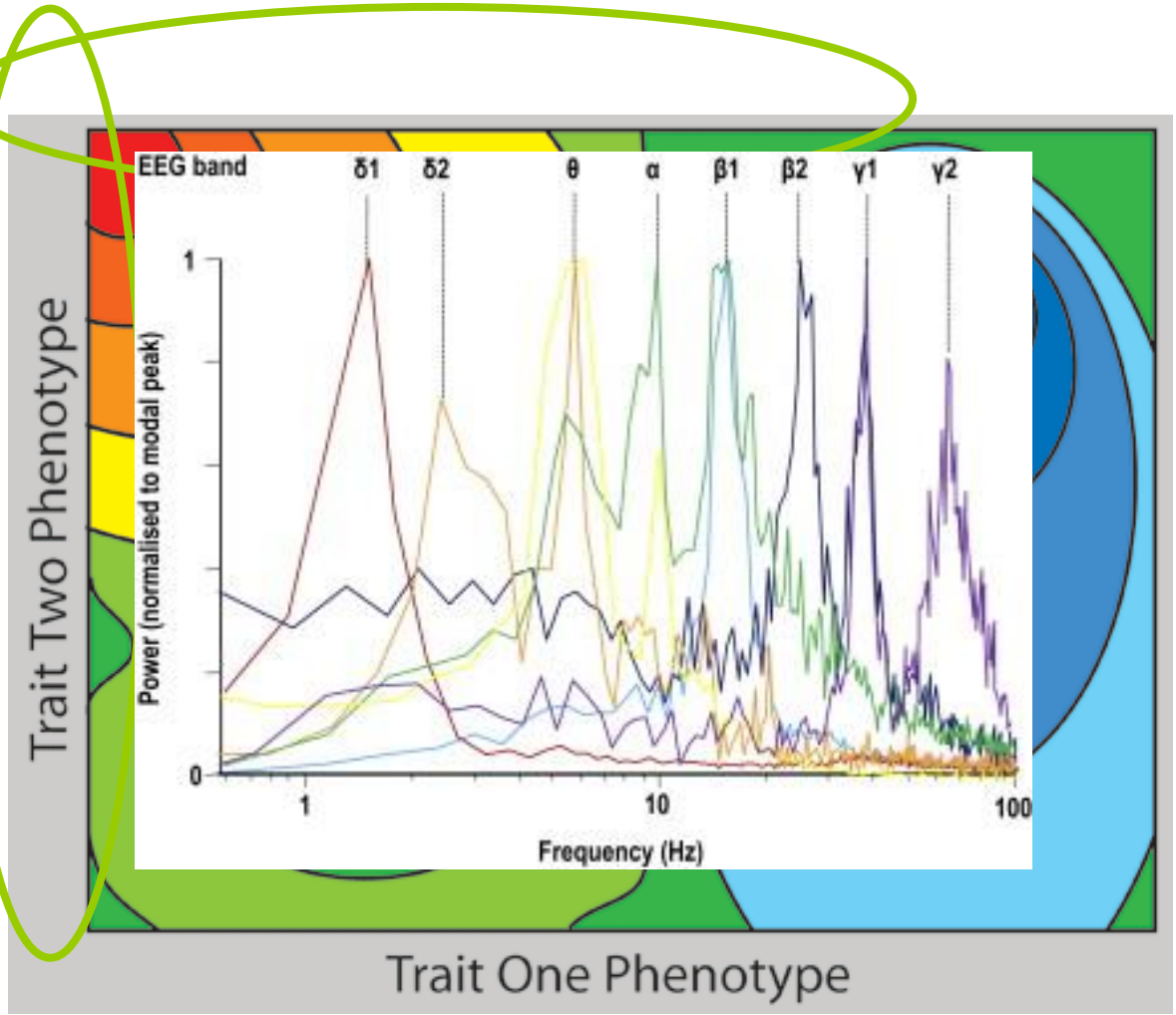
HOW I HAVE STRUCTURED MY TALK

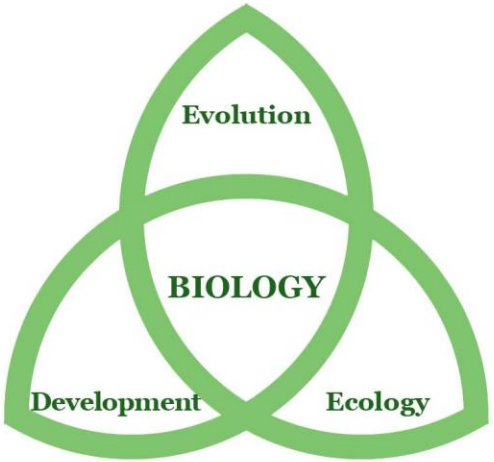
1. Introduction
2. Clinical linguistics: struggling with symptoms and causes
3. Bridging genes to language (and vice versa)
4. Putting the focus on brain oscillations
5. Conclusions and future prospects

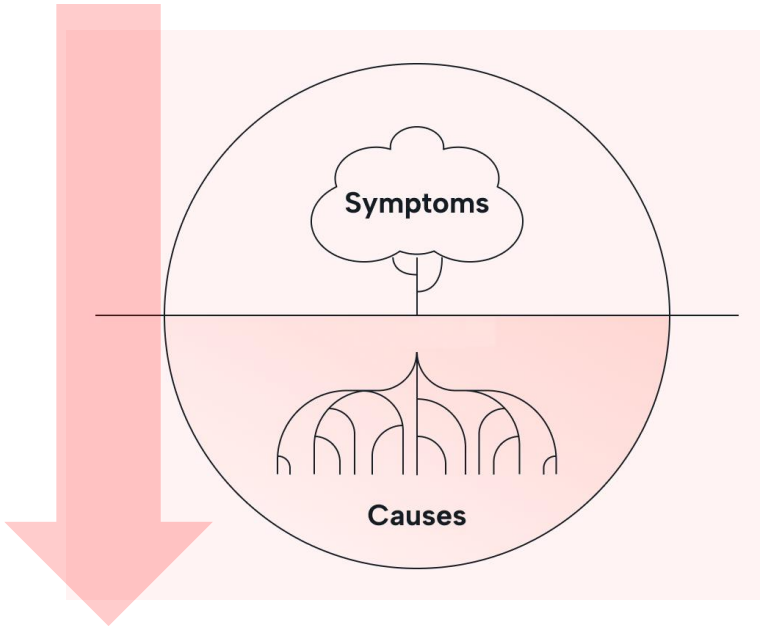

Bioinformatics
Multidemensional Networks
Personalised medicine
Statistical analysis
translational genomics
Rna seq
Genetics
Microbiome
Statistical modelling
Systems Biology
Network Motifs
Epigenomics
Metabolomics
Biomarkers
Regulatory Hubs
Network Analysis
Focus Hubs
microarray
Predictor
Genomics
Data
genome
omics
chipseq
Models
Master Regulators
animal
Clutsering
Scale Free Networks
Proteomics
Mathematical Modelling
Integrated Networks

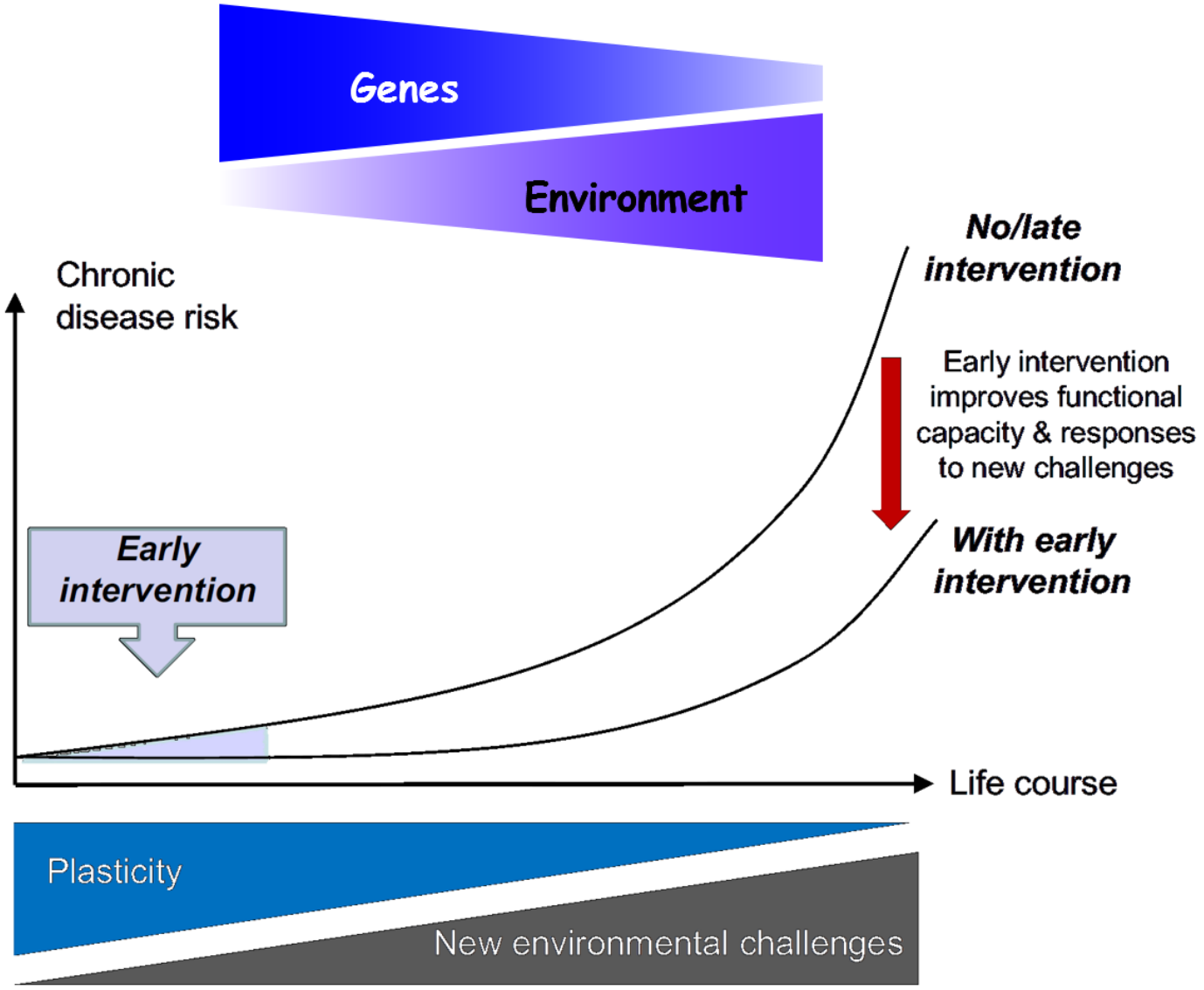

Bioinformatics
Multidemensional Networks
Statistical analysis
translational genomics
Rna seq
Genetics
Microbiome
Statistical modelling
Systems Biology
Network Motifs
Epigenomics
Personalised medicine
Metabolomics
Biomarkers
Regulatory Hubs
Network Analysis
Focus Hubs
microarray
Predictor
Genomics
Data
genome
chipseq
Models
Master Regulators
omics
animal
Clutsering
Scale Free Networks
Proteomics
Mathematical Modelling
Integrated Networks

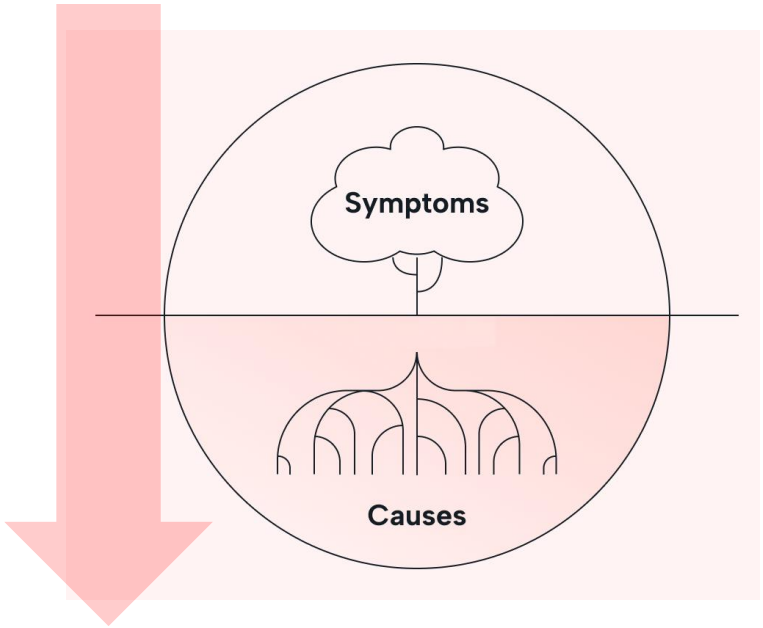

Evolution
BIOLOGY
Development
Ecology

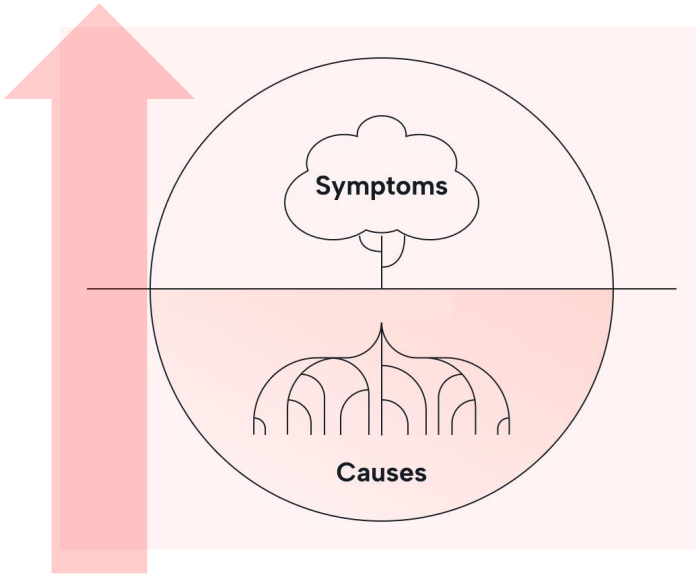

EEG band
δ1
δ2
θ
α
β1
β2
γ1
γ2
Power (normalised to modal peak)
1
0
1
10
100
Frequency (Hz)
Trait Two Phenotype
Trait One Phenotype

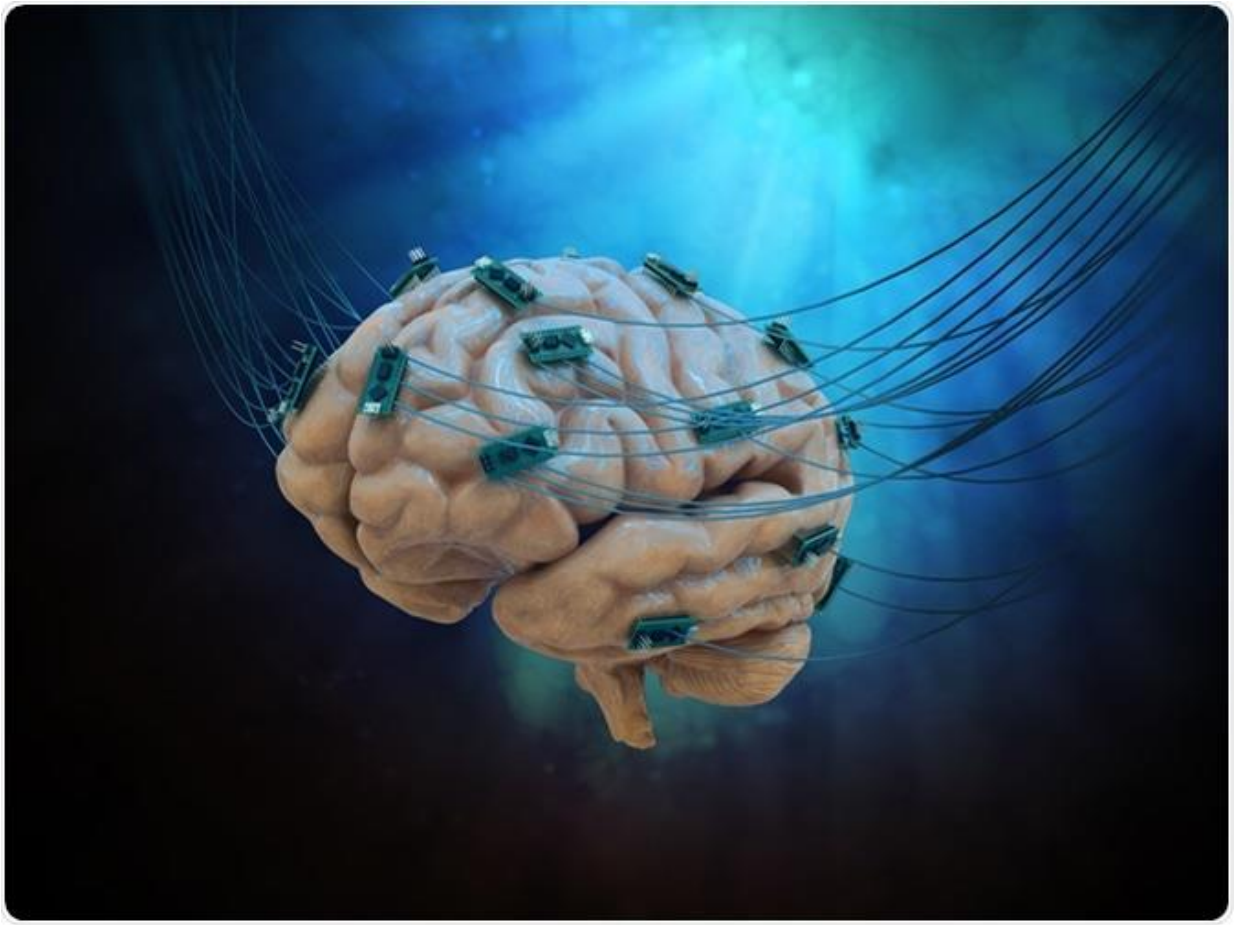

Evolution
BIOLOGY
Development
Ecology


Symptoms
Causes


Genes
Environment
Chronic disease risk
No/late intervention
Early intervention improves functional capacity & responses to new challenges
With early intervention
Early intervention
Life course
Plasticity
New environmental challenges


Symptoms
Causes


Symptoms
Causes



Thanks a lot for your attention!

Dr. Antonio Benítez Burraco
Full profesor of Linguistics
Área de Lingüística General. Departamento de Lengua Española, Lingüística y Teoría de la Literatura. Facultad de Filología.
Universidad de Sevilla
e-mail: abenitez8@us.es
web: http://antoniobenitez.wix.com/benitez-burraco
Twitter: @abenitezburraco

Principal Investigator. Research Project: "La evolución de las lenguas modernas: fundamentos genéticos y sociológicos del continuo lingüístico esotérico-exotérico" (PID2020-114516GB-I00))

Principal Investigator. Research Group "Biolingüística" (HUM 972) (Junta de Andalucía, spain)