The Dynamics of Complex Systems

#1 Introduction to complex systems

Fred Hasselman & Maarten Wijnants

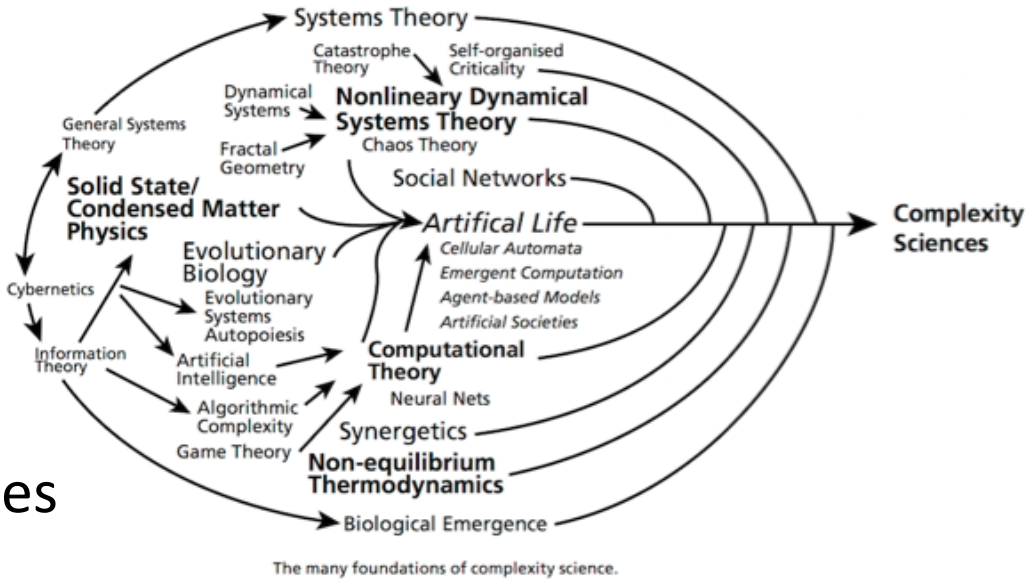
This course

- 8 lectures
  - Theory and methods
- 8 practical sessions
  - Hands-on data analysis
- Literature
  - Course guide
  - Papers
- Bb discussions
- Take-home exam

There are a number of characteristic features that are shared by almost all complex systems. A complex system can often be seen as a large collection of small elements that interact with each other at a micro-level. Such elements may be atoms in physics, molecules or cells in biology, or consumers in socio-economics. However, *'more is different'* (Anderson, 1972) in complex systems. Phenomena observed at a global, *macro*-level, typically cannot be reduced to the properties of the constituent elements: these are *emergent* properties that arise through *'self-organizing'* local interactions. This is in sharp contrast to the classical reductionistic idea that nature can only be understood by reducing or decomposing its processes into elementary building blocks that can be studied independently.
The concept of *'Complexity'* has been introduced as the associated paradigm shift in the study of natural phenomena. »

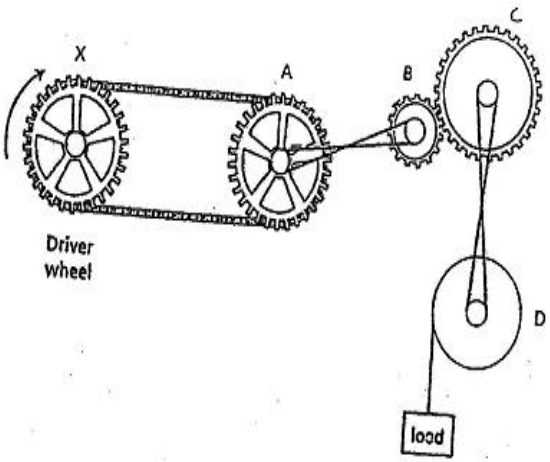
Complex systems

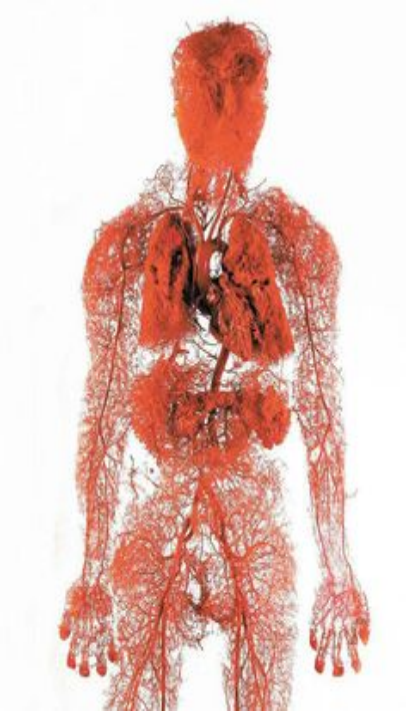
- Interactions more dominant than components
- Nonlinear
- Micro-macro
- Dynamic
- Self-organization
- Multiple causality
- Across multiple levels/scales
- Context-specific



The many foundations of complexity science.

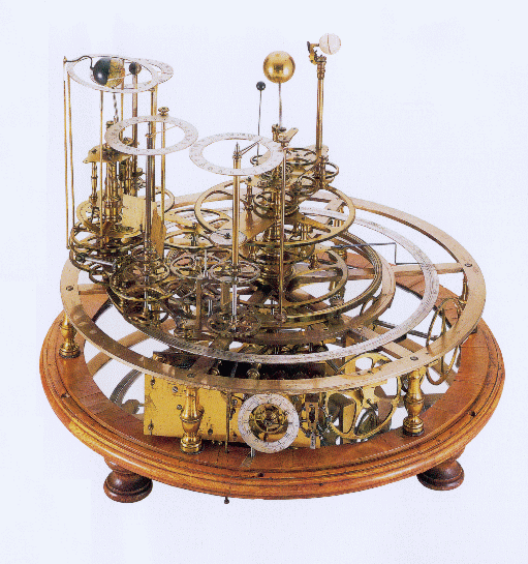
Complicated Systems vs. Complex Systems

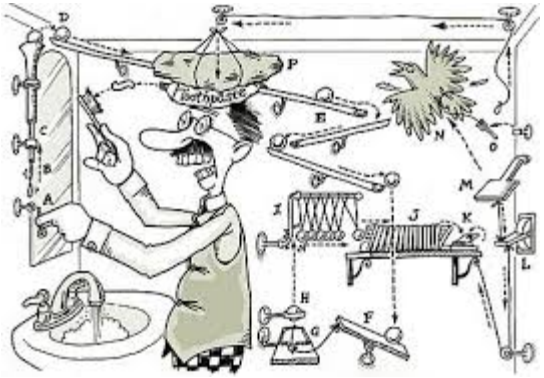




Mechanistic view

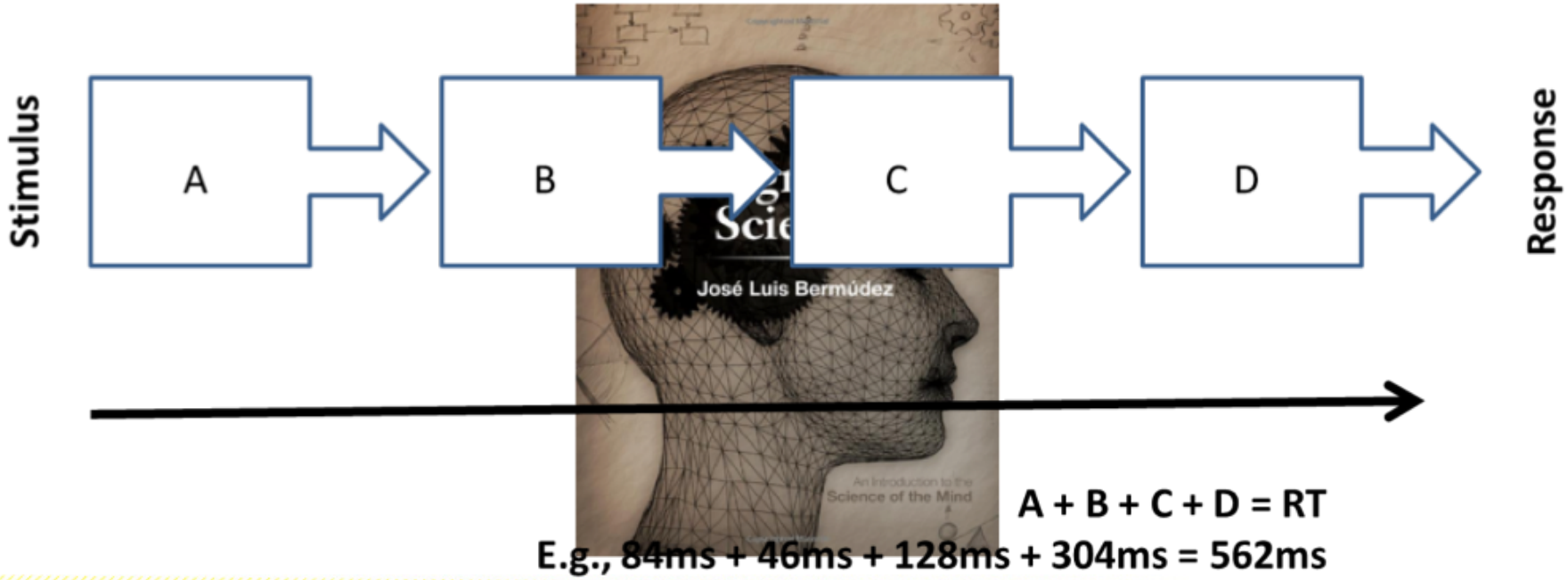
- Reductionism:
  - Reduce complicated problems into simpler parts



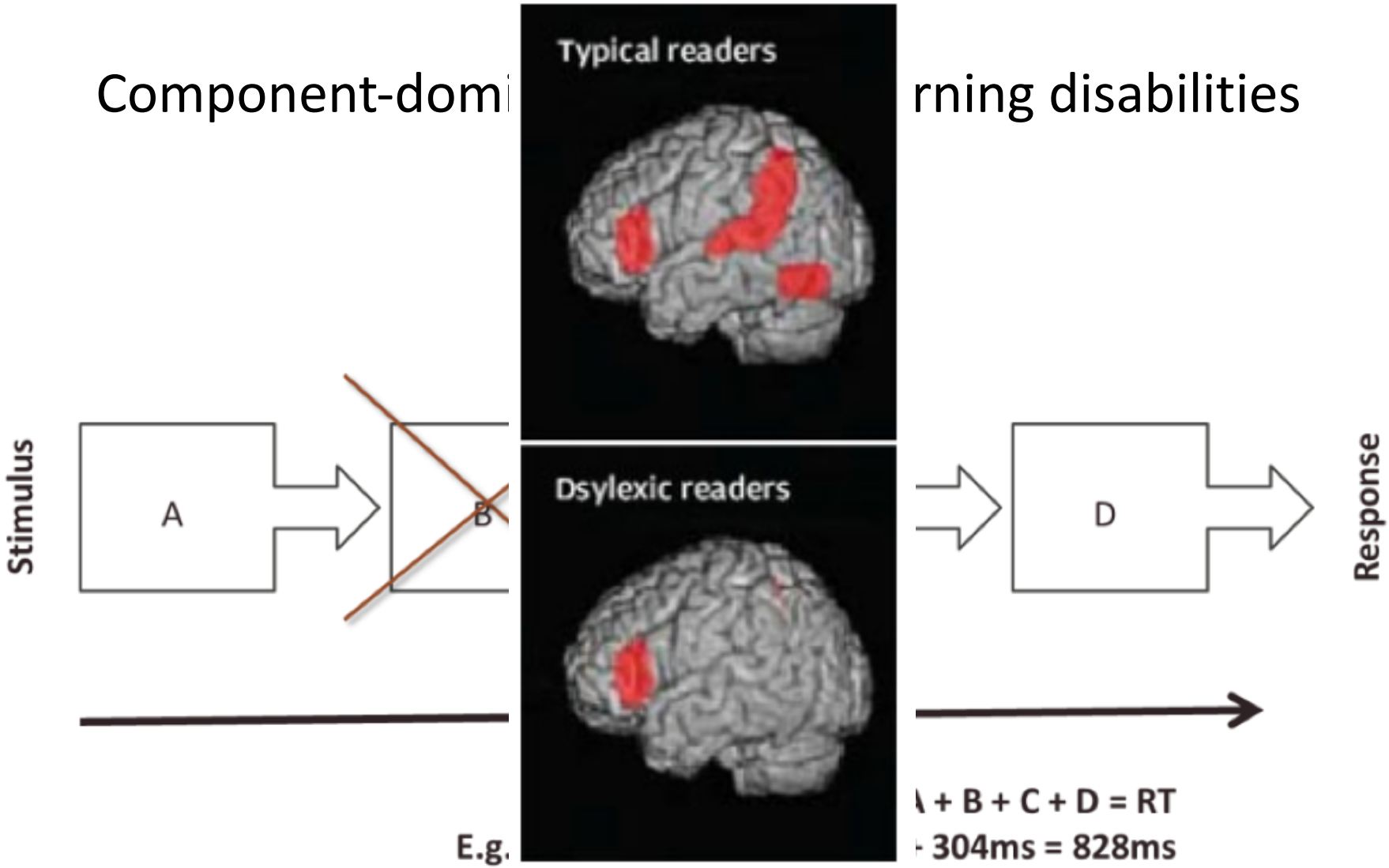


Component-dominant dynamics

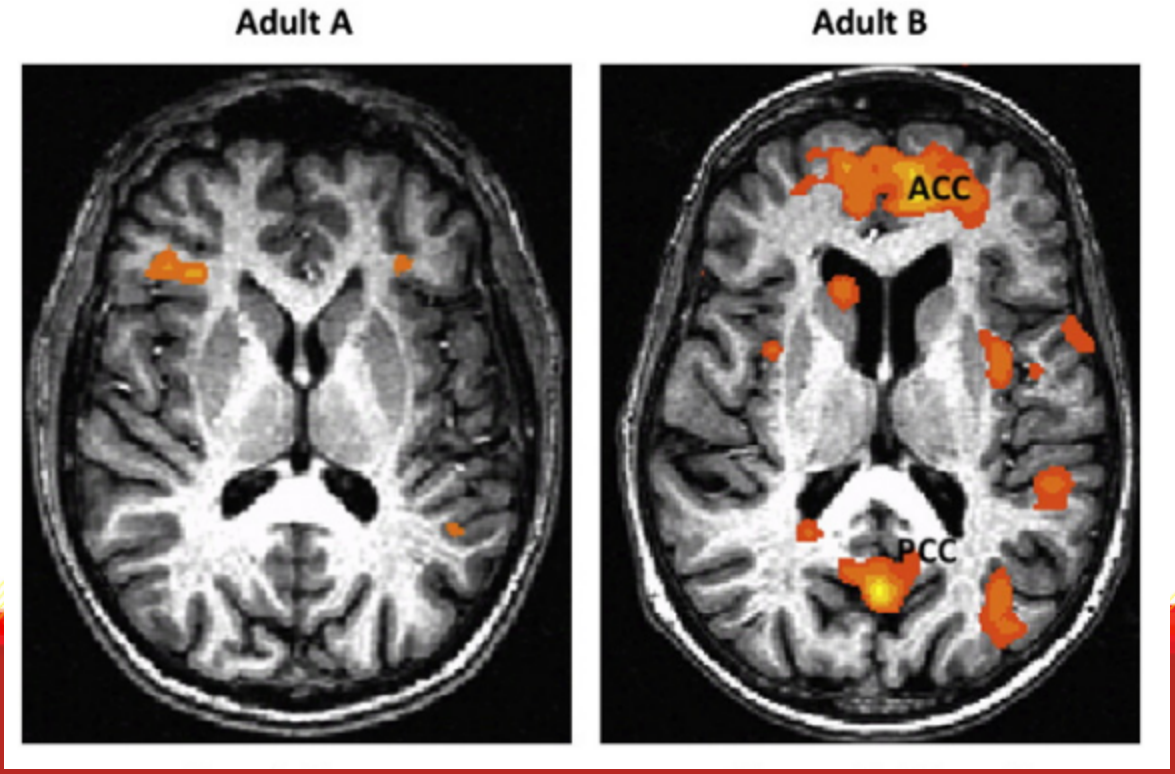
Additive component interactions



Component-domi rning disabilities



Behavioral, fMRI, ERP, and molecular genetic studies at the group level often mask important findings associated with sub-groups of individuals.



Parasuraman & Jiang, 2011

Alternative

Emergence

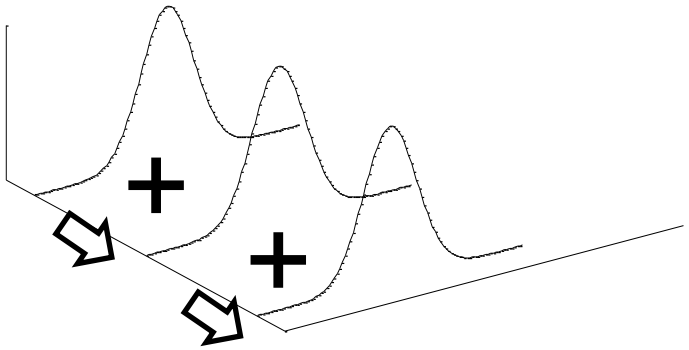
- Local interactions → global order
- Not reducible to component behavior

Table 1. *Results of PubMed search for articles on major depressive disorder 1980-2014*

Terms	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Cognitive bias	331													
2. Rumination	11	263												
3. Memory	3	8	296											
4. Social isolation	4	4	1	363										
5. Financial stress	0	0	2	9	280									
6. Immune response	0	0	1	5	0	816								
7. Cortisol	2	3	12	27	2	56	1884							
8. Hippocampus	0	0	3	4	1	2	15	151						
9. Sleep	3	6	4	7	11	43	127	0	2820					
10. Gene	2	5	5	8	2	58	38	10	37	1552				
11. Personality disorder	9	5	3	6	0	3	17	1	19	7	1225			
12. Diet	2	0	0	0	0	24	5	0	11	15	3	294		
13. Exercise	1	0	3	7	2	16	15	0	47	5	0	15	547	
14. Early adverse experiences	6	4	0	2	0	10	21	8	1	40	16	0	1	347

Wittenborn, Rahmandad, Rick, & Hosseinichimeh (2016). Depression as a systemic syndrome: Mapping the feedback loops of major depressive disorder. *Psychological Medicine, 46*, 551-562.

Statistical components

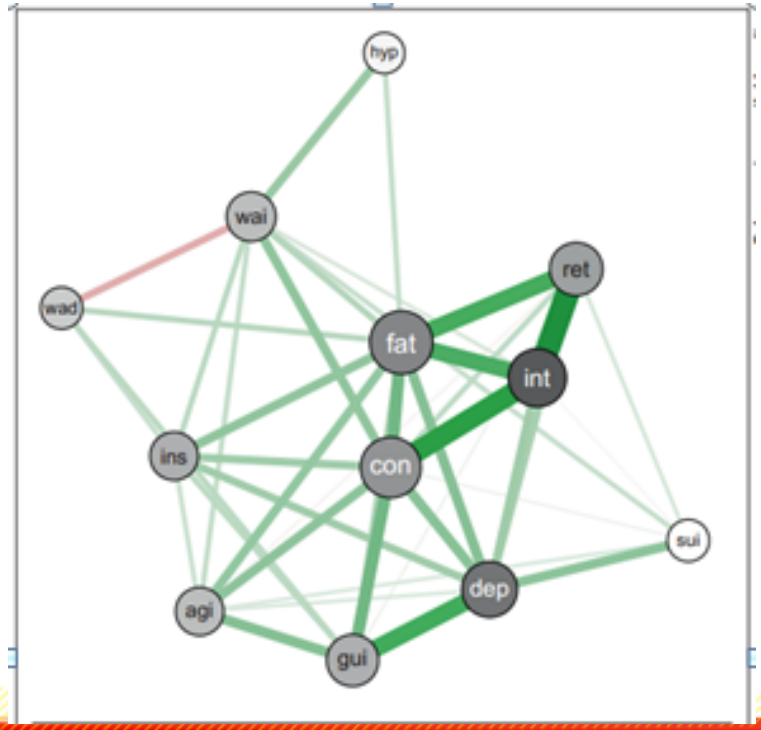


≈ Behaviour

- Each component explains part of variance
- As single causes for behavior

Causality in complex systems

Factors are not necessarily independent of one another



	Description	Centrality of symptom in the network	Association of symptom with the onset of MDD	
		symptom strength	OR	p
dep	depressed mood	0.77	3.68	<0.001
int	loss of interest/pleasure	0.83	4.30	<0.001
wad	decrease in weight/appetite	0.22	1.75	0.030
wai	increase in weight/appetite	0.48	2.06	0.001
ins	insomnia	0.44	2.43	<0.001
hyp	hypersomnia	0.13	1.18	0.456
ret	psychomotor retardation	0.65	2.66	<0.001
agi	psychomotor agitation	0.44	2.09	<0.001
fat	fatigue	1.04	3.34	<0.001
gui	feelings of guilt/worthlessness	0.62	2.36	<0.001
con	concentration problems	0.95	3.02	<0.001
sui	suicidal thoughts	0.21	1.04	0.903

Medical model: Independent problems

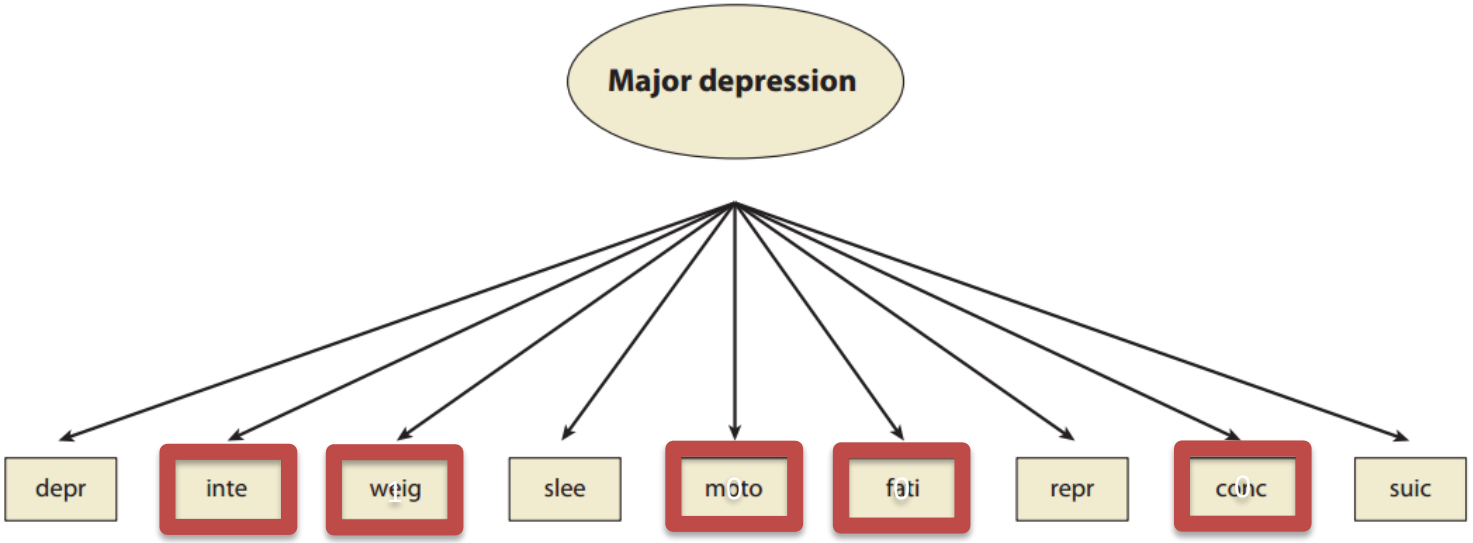
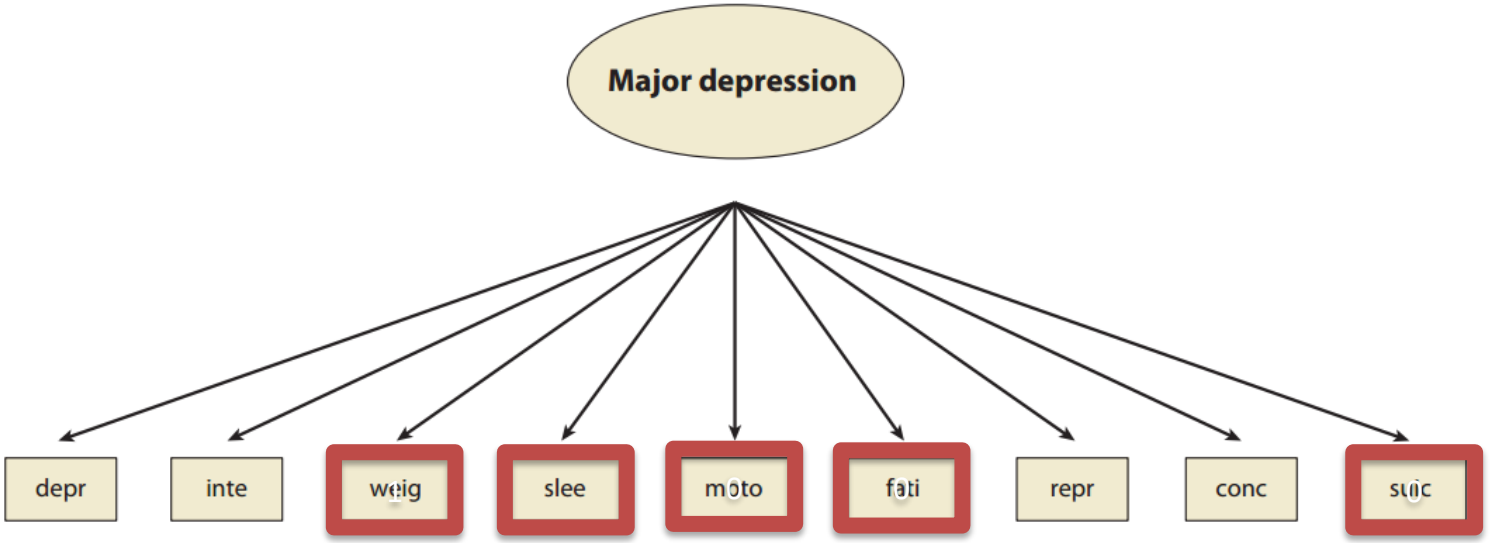


Figure 1

The relation between the disorder major depression (MD) and its observable symptoms according to a medical disease model. According to this model, MD (the oval at the top of the figure) is the root cause of its observable symptoms (the boxes at the bottom of the figure). Arrows point from the root cause (MD) to its observable symptoms, but not the other way around. See **Table 1** for definitions of abbreviated terms.

Medical model: Independent problems



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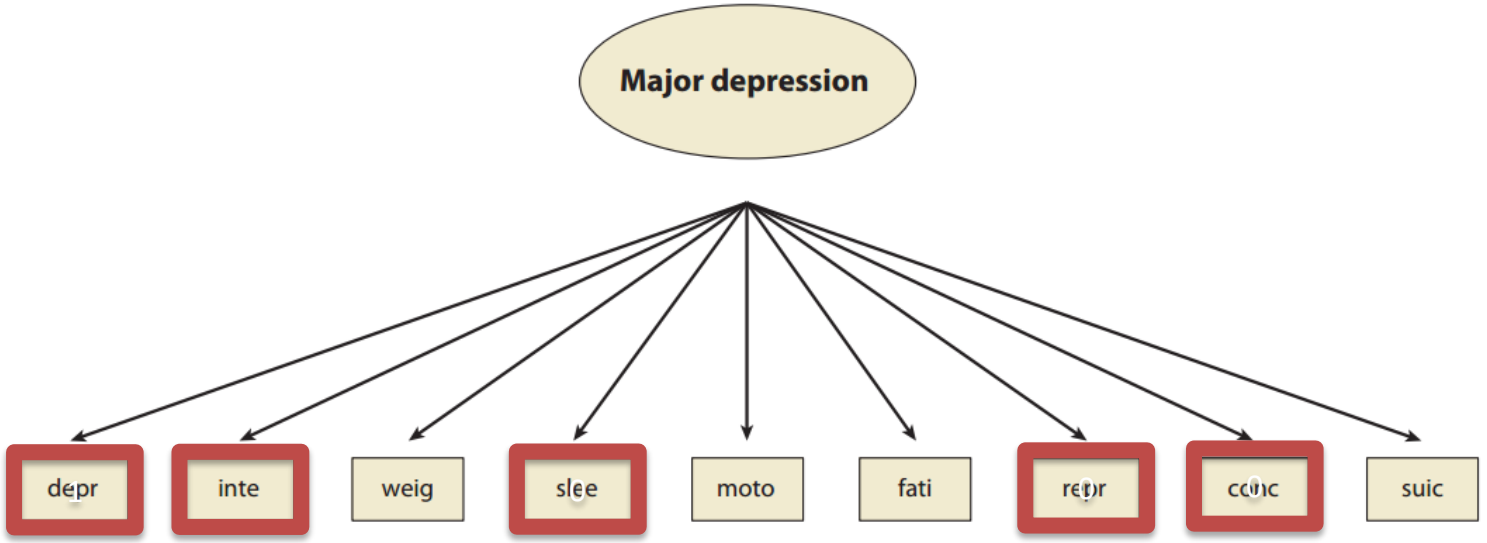

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Complexity vs categorization

Fried & Nesse (2014)

Participants

- N = 3703
- 18 to 75 years old
- Seeking treatment for MDD
- Fulfill DSM-IV criteria for MDD
- Received antidepressants

12 depression symptoms

- 4096 possible unique combinations of symptoms
- **1030** actual unique profiles

Table 1
Depression symptoms.

QIDS-16 symptoms	Mean	Median	*SD*
Sad mood	2.14	2	0.83
Loss of energy	2.00	2	1.15
Concentration problems	1.83	2	1.00
Insomnia	1.69	2	0.82
Loss of interest	1.69	2	1.11
Appetite problems	1.42	1	1.22
Self-blame	1.37	1	1.25
Weight problems	1.16	1	1.22
Psychomotor agitation	1.05	1	1.00
Psychomotor retardation	0.90	1	0.97
Suicidal ideation	0.74	1	0.85
Hypersomnia	0.44	0	0.83

Not just a few subtypes with some exceptions…

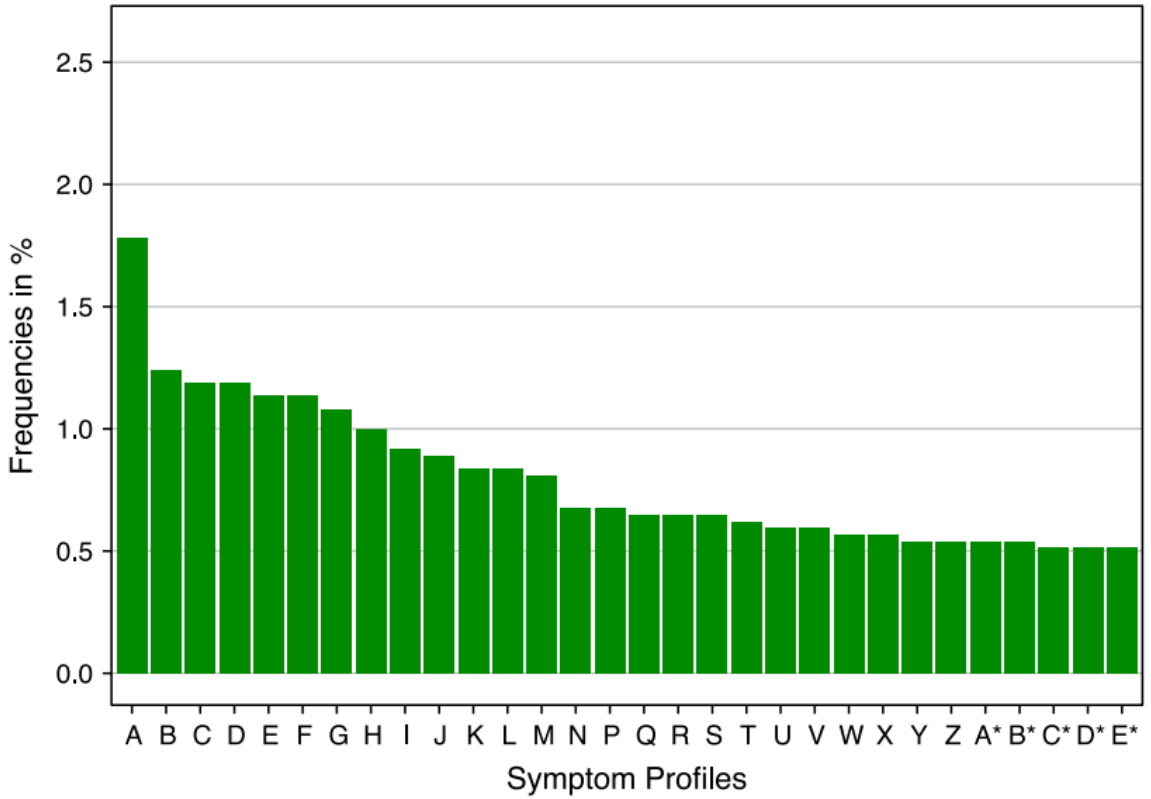
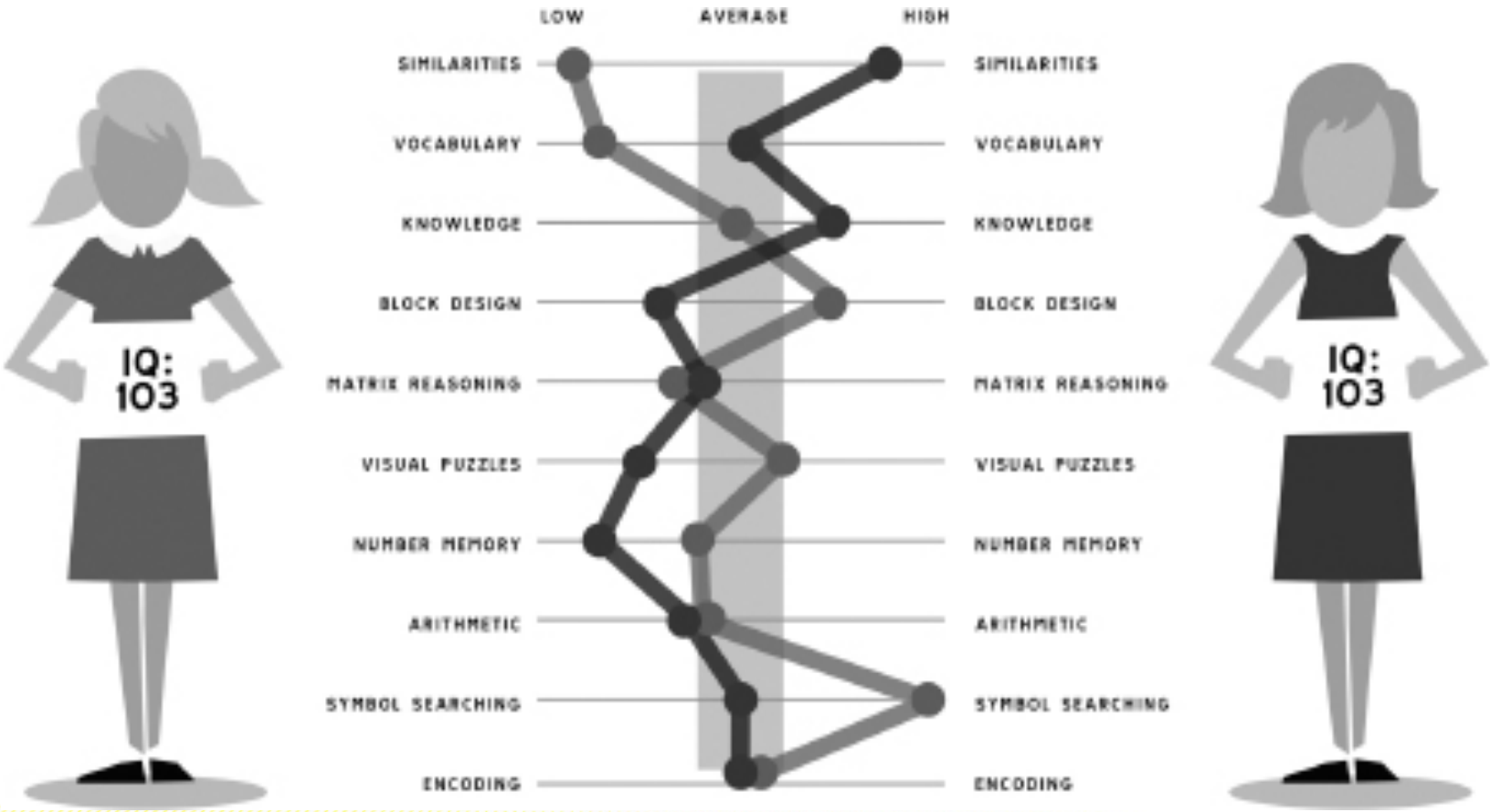


Fig. 1. Frequencies of the 30 most common depression symptom profiles during the beginning of the first treatment stage of the STAR*D study ($n=3703$).

Jagged profiles (Rose, 2016)



Group research

Statements about groups (that characterise individuals)

- Women are more emotional than men
- Baby's don't have a ToM, whereas toddlers do
- Lesbians only have sex with women
- Adolescents are more impulsive than young adults
- People with ADHD have weakly developed EF
- Children with ASD display repetitive behaviour
- Teachers are prejudiced against pupils from low-SES backgrounds

Statements are often linear (or all-or-none)

- The older one gets -> the less impulsive the behaviour will be
- The weaker EF -> the stronger ADHD
- The more repetitive the behaviour -> the clearer the autism

What did we learn so far from applying statistics?

Compare means

Mean EF_{adhd} < Mean $EF_{control}$

20 (*SD* = 3.4) < 24 (*SD* = 4.2) *T* test is significant, $p < .05$

Compute correlation

Age * Impulsiveness: $r = -0.30$, $p < .05$, 9% variance explained

Run ANOVA

IV = Gender (Female vs. Male) and Group (Autistic vs. Control)

DV = Score on a test of Theory of Mind (ToM)

Effect of Gender: $ToM_{female} > ToM_{male}$ 3% of variance explained

Effect of Group: $ToM_{autistic} < ToM_{control}$ 10% of variance explained

Conclusions about the individual? OOPS, maybe later!!!

On what is traditional statistics based?

only concern the group, not the individual

linear relationships (e.g., correlation, *t* test, ANOVA, regression)

components explain behaviour (IV + Noise => DV): Autism + noise -> ToM

independent components added together explain behaviour:
3% of the variance caused by gender + 10% by the group = 13% in total

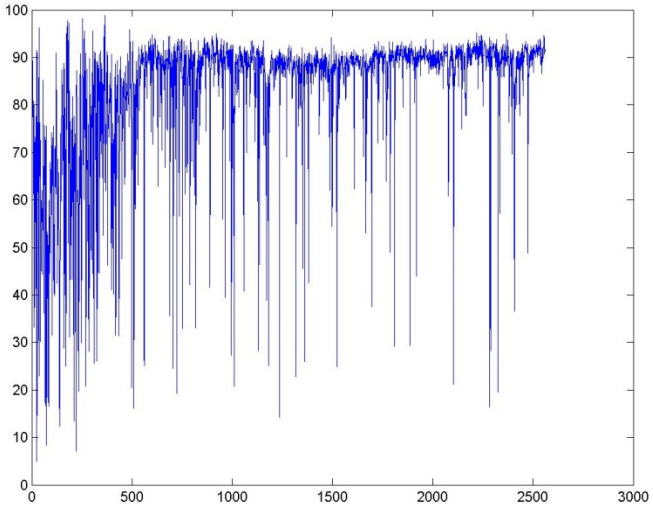
Observed score = true score + noise; In formula: $X_1 = T + \varepsilon_1$

Dynamics vs. classical test theory

Depressive symptoms

Time

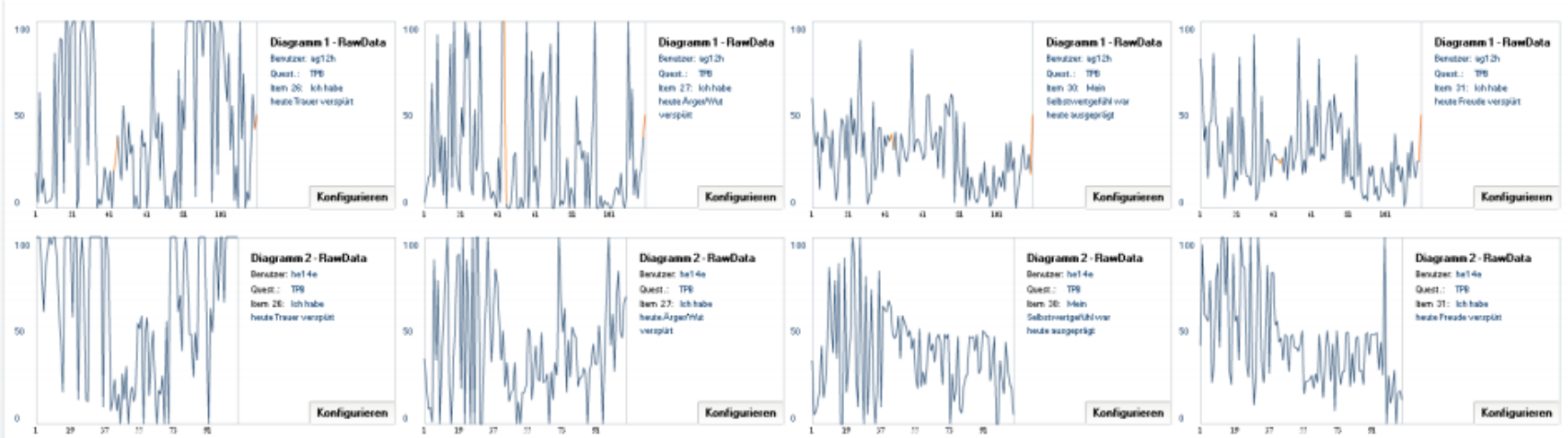
Self-reports of mood



Time

$X = T + E$?

Nonlinear and nonstationary dynamics in time series from daily ratings on the Therapy Process Questionnaire by an Internet-based device (Synergetic Navigation System)



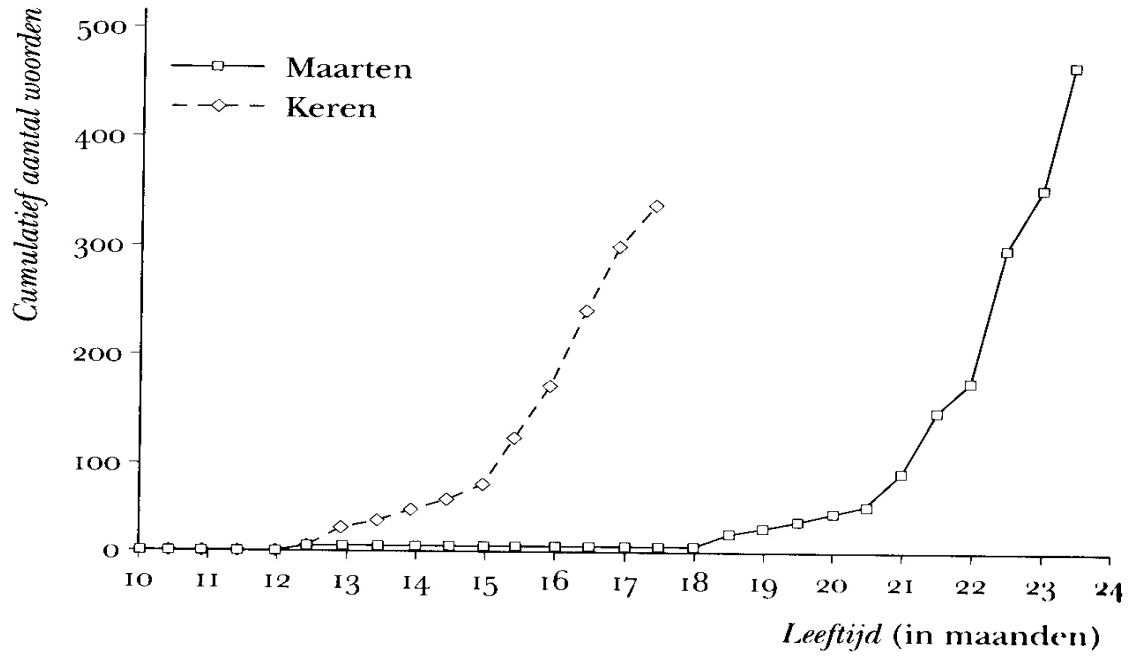
grief anger/rage self-esteem joy

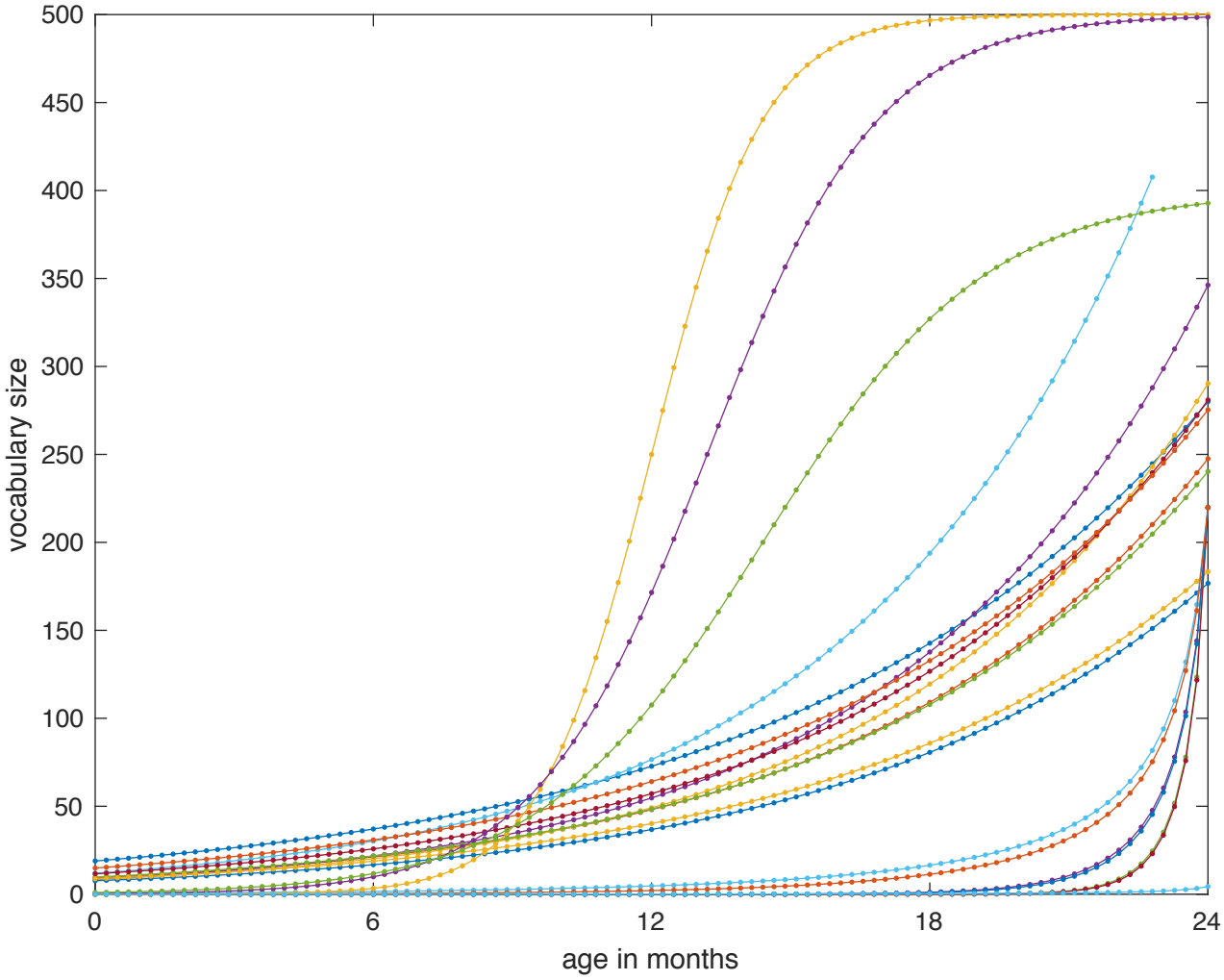
Vocabulary development

- Nonlinear and dynamic
  - E.g. vocabulary spurt

FIGUUR 5.1

Cumulatief overzicht van de woordenschat van Maarten en Keren

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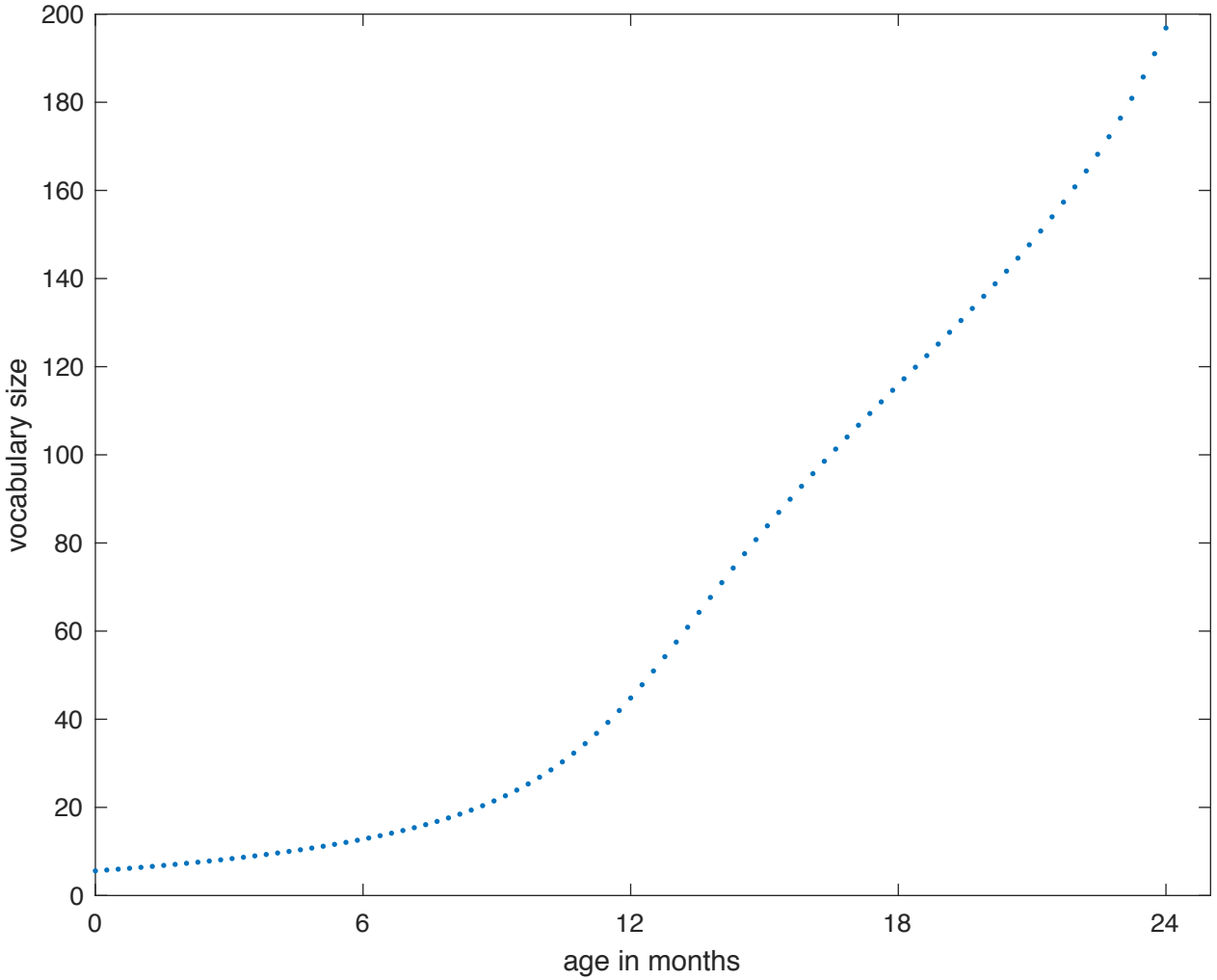
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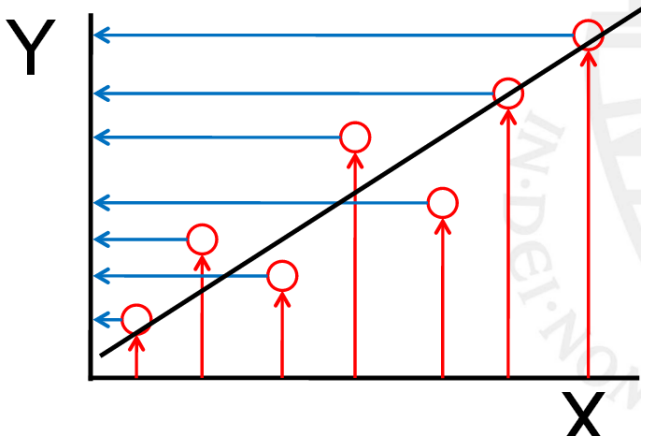
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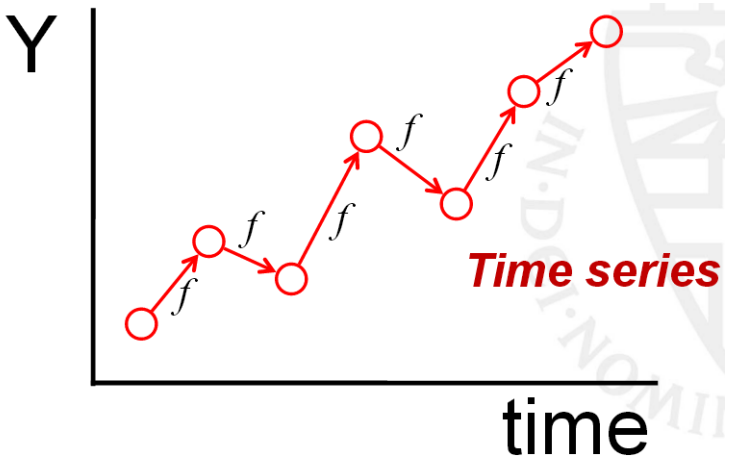
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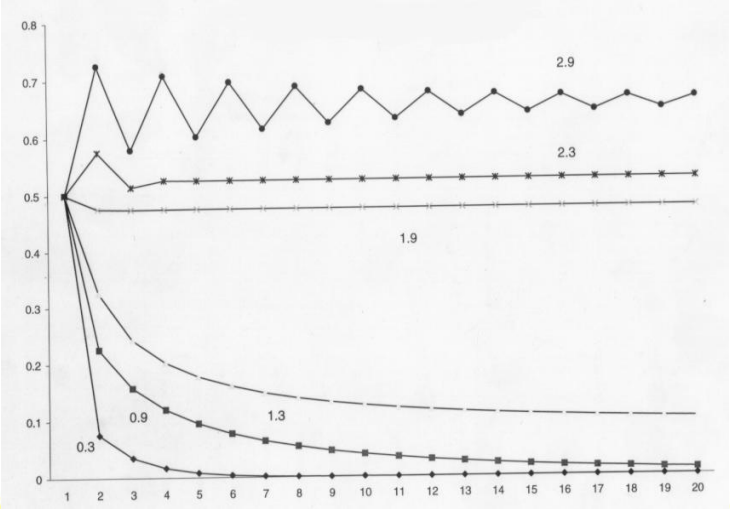
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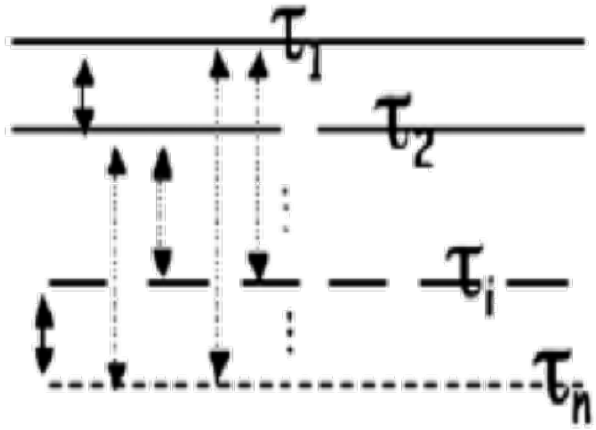
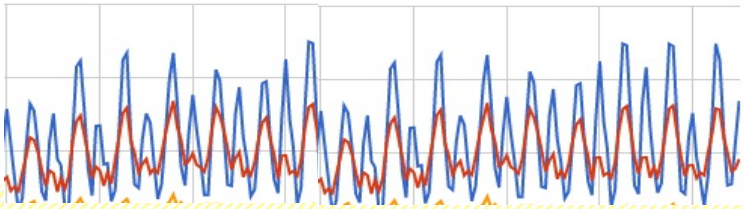
Multiple timescales

- Micro: seconds and minutes
- Days and weeks
- Months and years, even decades



Local variance



Global variance

Concepts of complexity science

- Fluctuations
- Wavelets
- Attractors
- Entropy
- Metastability
- Phase transitions
- Critical points
- Degrees-of-freedom
- Emergence
- …

Topics

- **I. Introduction to the mathematics of change**
  - Modelling (nonlinear) growth and Deterministic Chaos (C1)
  - Multivariate systems: Predator-Prey dynamics (C2)
  - Potential theory, Agent-based models, Dynamic field models (C2)
- **II. Time Series Analysis: Temporal Correlations and Fractal Scaling**
  - Nonlinear regression: Fitting analytic solutions and Catastrophe Theory (C3)
  - Basic timeseries analysis: Quantifying temporal correlations and order transitions (C4)
  - Scaling phenomena: Fluctuation analyses and (multi-)fractal geometry (C5)
- **III. Quantifying Recurrences in State Space**
  - Takens' Theorem and State-Space reconstruction (C6)
  - Recurrence Quantification Analysis of continuous and categorical data (C6)
  - Cross-Recurrence Quantification Analysis of dyadic interaction (C7)
  - Early Warning Signals of order transitions (C7)
- **IV. Complex Networks**
  - Small-world and Scale-free networks (C8)
  - Symptom networks and Networks of Quantified Recurrences (C8)
  - Early Warning Signals of order transitions in clinical settings (C8)

Hope you will find it great fun!

- Any questions?

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