Computational modelling of developmental disorders

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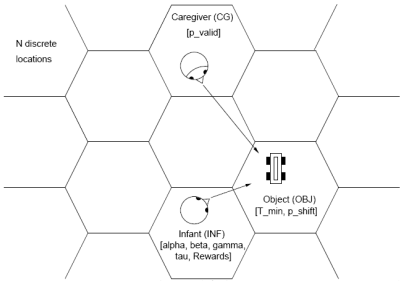
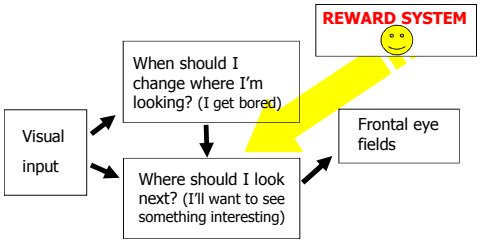
Emergence of Gaze following



Triesch et al. (2006)

Build a model



Learn to fixate rewarding objects (make use of where mummy is looking?)

Reinforcement learning: State-action tables (networks)

Estimate the reward for each action in each situation

Table 1 *A state-action table for the When module*

		States (time slices)									
		T1	T2	T3	T4	T5	T6	T7	T8	T9	T10
Actions	Maintain fixation	90	80	55	40	30	18	10	5	3	1
	Shift fixation	10	20	45	60	70	82	90	95	97	99

Note: Values show the system's reward estimates for each action in each state. Values are illustrative and depict the idea that habituation conditions the system to change fixation after some point in time. Note, the action that is selected is determined probabilistically, depending on the system's bias for exploring the environment versus its bias for exploiting its existing knowledge (see Triesch *et al.*, equation 7).

Table 2 *A state-action table for the Where module*

		States						
		Where caregiver is looking						
		Region 1	Region 2	Region 3	Region 4	Region 5	Caregiver looking at infant	No info. (not looking at caregiver)
Actions	Fixate 1	80	5	5	5	5	20	10
	Fixate 2	5	80	5	5	5	20	10
	Fixate 3	5	5	80	5	5	20	10
	Fixate 4	5	5	5	80	5	20	10
	Fixate 5	5	5	5	5	80	20	10
	Fixate caregiver	0	0	0	0	0	0	50

Note: Values are illustrative and depict a system that has successfully learned gaze following. Therefore the system gets most reward from looking to the region where the caregiver is looking. Other regions still have reward estimates because the caregiver may be unreliable, the infant may have misperceived direction of gaze, or may chance upon rewarding events by ignoring the caregiver. For states where the infant is already looking at the caregiver, 'Fixate caregiver' reward is zero, since the When table has already requested a change in fixation. The right hand column ('No info.') corresponds to the system's 'sociability', i.e. its tendency to spontaneously look at the caregiver rather than search for an object.

Richardson & Thomas (2006)

Developmental trajectory of the model

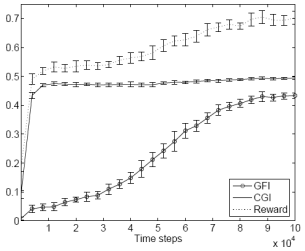


Figure 2. Emergence of gaze following in simple environment with just one interesting *target* present at any time. The solid curve plots the caregiver index (CGI), the solid curve with circles plots the gaze following index (GFI), and the dotted curve plots average reward per time step, as functions of the number of learning iterations. Error bars indicate standard deviations across 15 simulations.

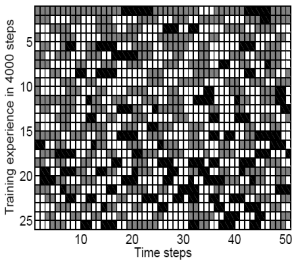
CGI = Do you look at the caregiver?

GFI = Do you look at the caregiver and then an object?

Reward = Are you happy? You know, deep down?

Triesch et al. (2006)

Sample sequences of actions



Each row of pixels shows the target of the infant's gaze as a function of time (for 50 steps). The gaze target is color coded, with white corresponding to the caregiver, black corresponding to the target, and grey corresponding to other regions of space. In particular, an instance of gaze following is represented by a black pixel lying to the right of a white pixel. Different rows show the behavior at different times during the learning process (every 4000 steps).

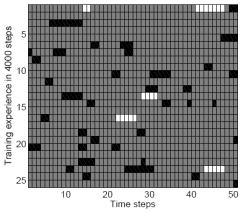
- Interesting object
- Caregiver
- Other region of space

Triesch et al. (2006)

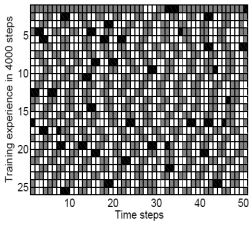
Normal vs. Atypical?

- We have a normal model of development
  - Not uncontroversial (e.g., Csibra, 2006)
- What if this systems lies within an individual with a developmental disorder?
- Autism
  - disinterest in social contact / faces
  - dyadic interactions observed to be atypical in young children with autism
- Williams syndrome
  - elevated interest in faces
  - triadic interactions observed to be atypical in atypical in toddlers with WS

Atypical development



Autism: Reduce reward of face



WS: Increase reward of face

Generate predictions: ADHD

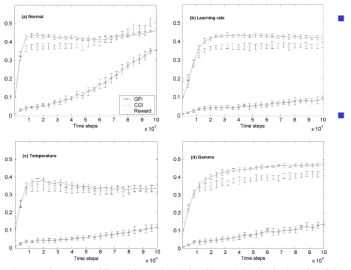


Figure 1 *Emergence of (a) normal gaze following behaviour (the equivalent of Figure 2 in Triesch et al.), shown alongside three parameter manipulations, (b) learning rate $\alpha = 0.001$, (c) temperature $\tau = 0.125$, and (d) discounting rate $\gamma = 0.3$, simulating a model with ADHD (default 'normal' parameter values: $\alpha = 0.0025$, $\tau = 0.095$, $\gamma = 0.8$). The caregiver index (CGI), gaze following index (GFI) and reward are shown. The error bars show standard deviations across 15 simulations.*

- ADHD (Williams & Dayan, 2005)
  - Reduced discounting rate
  - Too exploratory?
  - Slow reward learning?
- Prediction
  - Infant precursor of ADHD may be delayed gaze following (but normal interest in faces)

Richardson & Thomas (2006)

Modelling in the study of developmental disorders

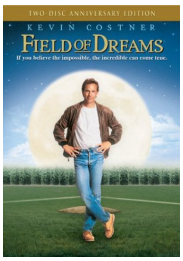
- Connectionist models of cognitive development
- Another case study – Specific Language Impairment
- Theoretical issues:
  - Where do modules comes from?
  - Can I really developmentally damage a single module?
- Conclusions

Mechanisms of change

- Developmental psychology cannot simply comprise a list of behaviours that children show at each age
- It must identify **mechanisms of change**
- Use of computational modelling: implemented learning systems suggest possible mechanisms of change to drive formation of developmental theories
- Trying to "imagine" how knowledge is acquired has produced very nativist theories…

developmental neurocognition lab

"Build it, and they will come."

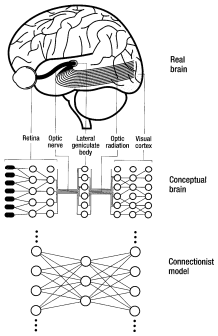


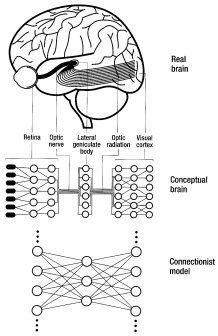
Brands of model

- Symbolic models
  - Rule-based / concept learning
  - Tree-learning
- Connectionist networks
  - Associative networks
  - Self-organising networks
  - Reinforcement learning
  - Adaptive resonance theory
- Bayesian networks
- Exemplar-based models
- Dynamical systems theory
- Single cell / neural spiking models
- Hybrid models (everything and the kitchen sink)

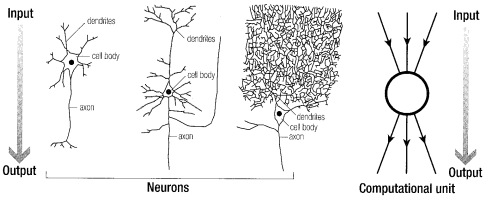
Connectionism

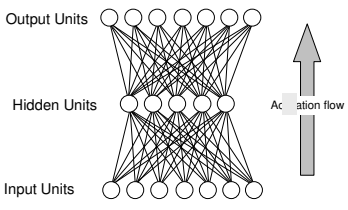
- Connectionist models use computational principles abstracted from neural computation
- Produce COGNITIVE level theories
- (a weakening of functionalism)





- **Simple processing units**
- **Networks**
- **Distributed (continuum)**
- **Parallel**
- **Layered**
- **Integrative**
- **Transformations**





Background

- "[With regard to connectionism] … nativism, associationism, empiricism, rationalism, reductionism, genetics, computer science, neuroscience, linguistics, ethology, and the mind/body problem (and I may have forgotten a few) have all somehow involved themselves in what started as just a psychologist's choice between theories of cognitive architecture"
  - Fodor (2000)

Connectionist models of development

- Demonstration of implemented systems
- Simulation of data
- Viability of theoretical proposals:
  - Stages from continuous change
  - Developmental lags from graded representations
  - Rule following behaviour from associative systems
- Models don't prove these are correct explanations
- Do widen *candidate inferences* from data

Connectionist models of typical development

- **Infancy**: categorisation, object-directed behaviour, memory
- **Childhood**: Piagetian reasoning tasks (balance scale problem, seriation, conservation), development of semantics
- **Language acquisition**: categorisation of speech sounds, segmentation of the speech stream into words, word learning and vocabulary development, inflectional morphology, syntax, metaphor, reading

Outstanding issues

	Timescale	Reversibility	Age of onset	Rate	Passive or Active?	Domain general vs. specific	Underlying mechanisms	Type of info processed	Inductive method
Learning	Quick One shot	More reversible (forgetting)	Later in life Episodic?	Can be increased through practice	Passive internalisation	Specific to task	Strengthen synapses within architectures?	Experiences of an individual	Search hypothesis space given experience
Development	Slow Several years	Less reversible	Earlier in life	Cannot be accelerated	Active exploration	Global across domains	Morphological changes of neural architectures?	Experiences common to all members of species	Alter / enrich hypothesis space

Developmental disorders

- Selective genetic developmental deficits most theoretically interesting
  - SLI
  - Dyslexia
  - Williams syndrome
  - Autism

Developmental disorders

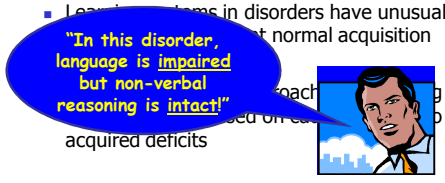
- Have isolated components failed to develop?
- Is this indeed evidence for [illegible] components, innately specified?

Reasonable recovery

- Appropriate explanation is not in terms of focal damage to normal system but atypical constraints on plasticity

Explanatory framework

- Explanations of deficits must be in terms of learning systems
- [illegible] disorders have unusual [illegible] normal acquisition



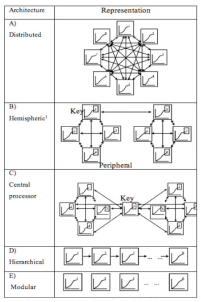
[illegible] from acquired deficits

Understanding the developmental process

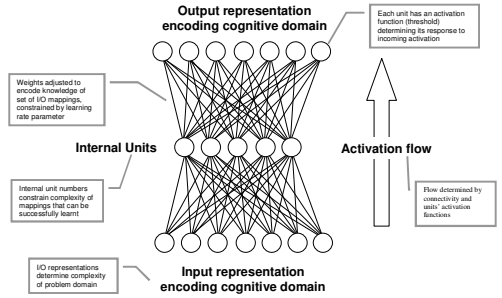
- The developmental process must be central to understanding of developmental disorders
  (Karmiloff-Smith, 1998)
- How can we explore the "developmental process"?

The developmental process

- Will include concepts such as plasticity, interactivity, redundancy, compensation
- Will specify relationships between components of cognitive system
- Most modelling work has focused on development within a single component or two linked components



3-layer feedforward network



Network parameters

- Models contain CONSTRAINTS which affect their ability to learn
  - Architecture
  - Activation dynamics
  - Representations
  - Learning algorithm
  - Training regime

Internal

Neurocomputational properties may be influenced by genetic differences

Environmental

Modelling disorders

- Assume a normal model
- Explore which parameter changes to the STARTSTATE lead to atypical trajectories and endstate deficits
- Use to assess viability of empirically driven hypotheses

The modelling enterprise

- Specific models
  - Test hypotheses in a particular domain using as many empirically driven constraints as possible
- Abstract models
  - Expand the range of candidate inferences from behaviour to structure
    - E.g., models used to assess the claim that double dissociations must arise from damaging modular systems
  - "This kind of behaviour can only be produced by these kinds of system"

A specific model

- English past tense
  - Talk-talked
  - Wug-wugged
  - Hit-hit
  - Sing-sang
  - Go-went
  - Over-regularisation errors ('I thinked it')
  - Omission errors ('yesterday, I talk to John')

"Rule"

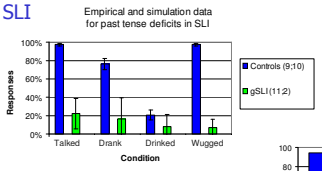
Exceptions

Early excitement (sells books)

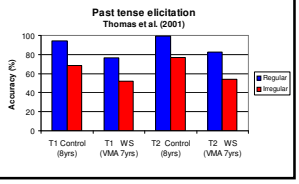
- Pinker (1999)
  - **SLI** = 'loss of certain genes [interferes] with the development of … the ability to inflect new and uncommon regular verbs' => Regular Deficit
  - **WS** = memory mechanism for storing exception verbs is specifically impaired => Exception Deficit
  - **WS** + **SLI** = 'a genetic double dissociation … the first group of children rarely generalise the regular pattern; the second group of children generalise it freely'

Some data

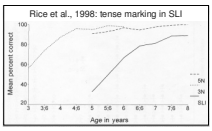
SLI

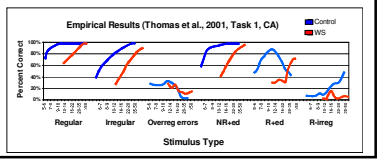


WS

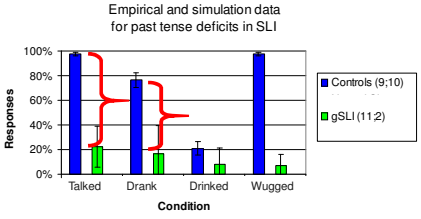


Study development…



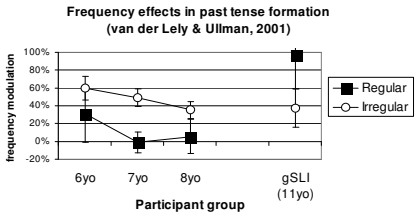


SLI=deficit in rule formation?



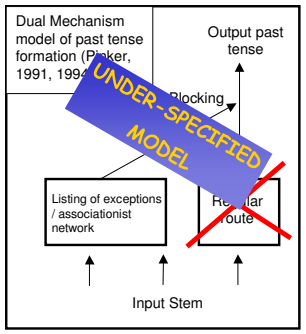
Van der Lely & Ullman (2001)

Is residual performance due to lexical memory?



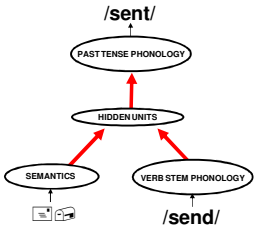
Assumption: Frequency effects are hallmark of memory systems
Absence of frequency effects implicates a rule

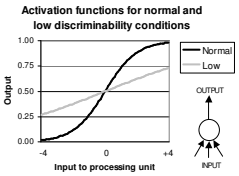
Pinker (1999): Box-and-arrow account



Specific Language Impairment

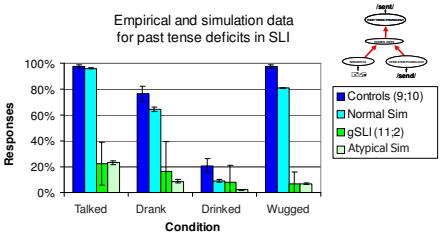
Vary learning properties of an implemented developmental model





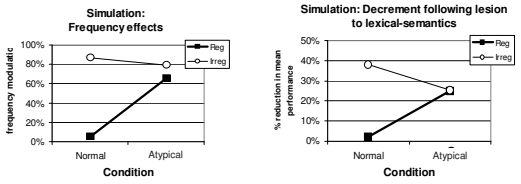
(Joanisse & Seidenberg, 1999)

English past tense model: Atypical developmental constraints

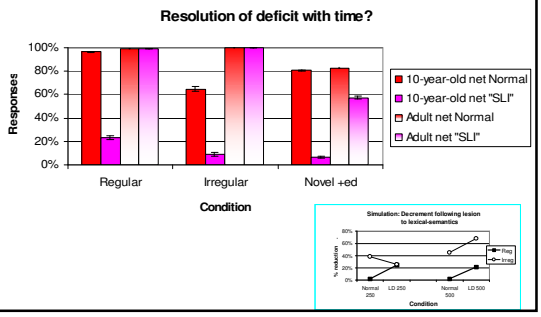


Thomas (2005), Thomas & Karmiloff-Smith (2003)

English past tense model: Atypical developmental constraints



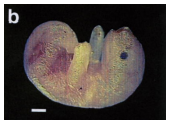
Delay and deficit resolution



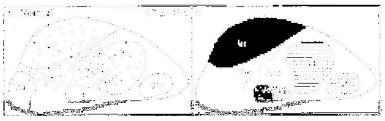
An Abstract model

- Addressing a theoretical issue on how the cognitive system develops

Animal studies



Huffman et al. (1999): reduce cortical sheet – what functional areas develop?



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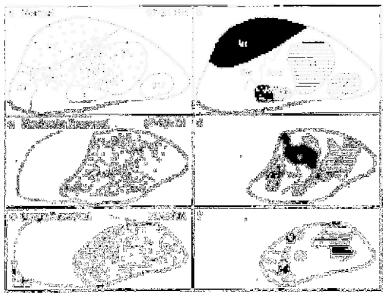
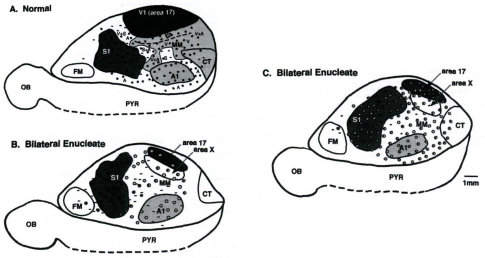
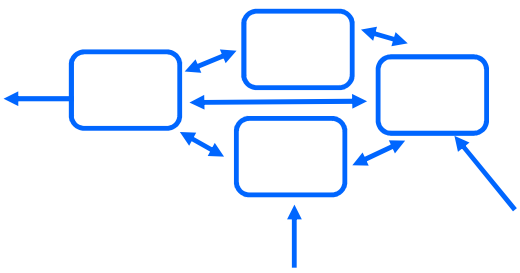


Kahn & Krubitzer (2002): change input



Where do modules come from?



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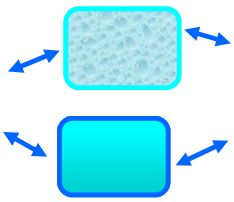
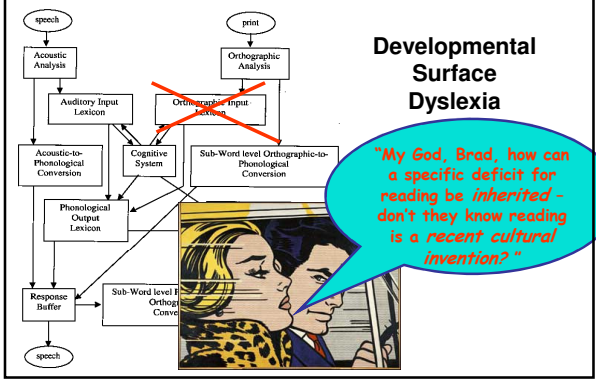
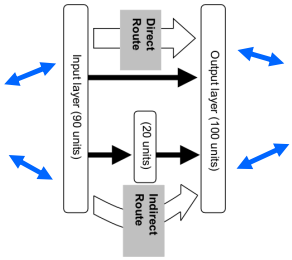


Reading



Origin of modules



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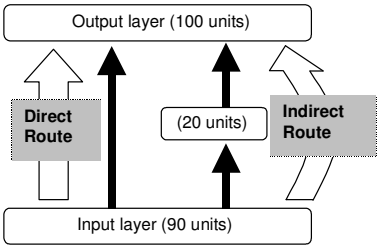
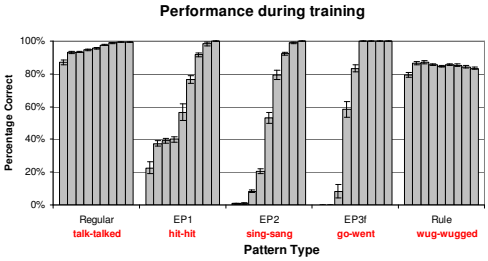
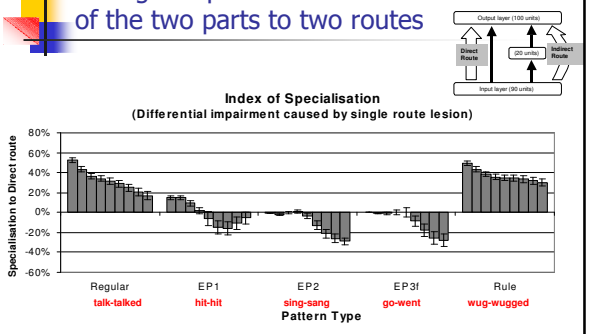


Learn a problem with two clear parts...

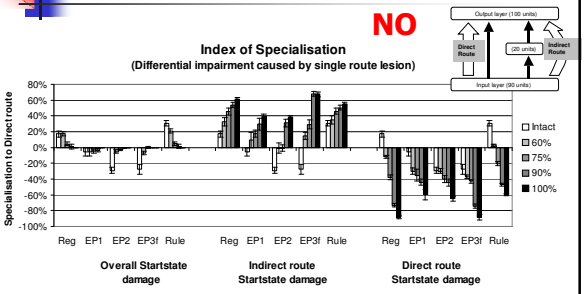


Emergent specialisation of function of the two parts to two routes



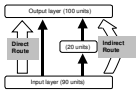
If one component is initially damaged, does other still develop normally?

NO



When would some components develop normally while others are damaged?

- Some developmental conditions for **Residual Normality**
  - Strong structure function-correspondences
  - Strong competition
  - Early commitment
  - Guided specialisation
  - Restrictions on resources



Thomas & Karmiloff-Smith (2002)

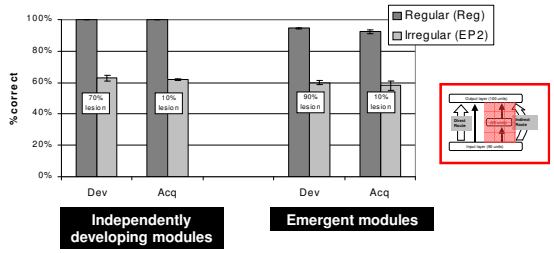
Is this important, at all?

- "The implication is that one cannot understand what is wrong in the cognitive system of a child or adult with a developmental disorder on their basis of apparently normal vs. impaired behaviour, unless one has a developmental account of where the cognitive structures comes from"

Inferences and disorders

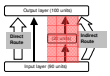
The effect of Residual Normality (RN)

Behavioural impairments after selective damage to Indirect route



Independently developing modules | **Emergent modules**

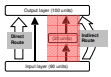
Inferences and disorders



What is producing normal (intact) *behaviour*? [Reg]

	Independently developing modules	**Emergent modules**
Acquired deficit	**Normal *process*** in Direct route	Mix of **Normal *process*** in Direct route and **residual *process*** in damaged Indirect route
Developmental deficit	**Normal *process*** in Direct route	**Atypical *processes*** in both Direct and residual Indirect route after development

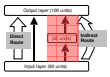
Inferences and disorders



What is producing impaired *behaviour*? [EP2]

	Independently developing modules	**Emergent modules**
Acquired deficit	**Impaired *process*** in residual damaged Indirect route	Predominantly **Impaired *process*** in residual damaged Indirect route
Developmental deficit	**Impaired *process*** in residual damaged Indirect route	**Atypical *processes*** in both Direct and residual Indirect route after development

Inferences and disorders



Does the dissociation imply independent underlying processes?

	Independently developing modules	**Emergent modules**
Acquired deficit	Yes	Yes
Developmental deficit	Yes	No

Dissociations in developmental disorders

- Inferences from impaired behaviour to underlying structure depend on having a developmental theory of the origins of the underlying structure
- Models help construct these theories

Three conclusions

- (1) Developmental disorders must be explained in terms of the developmental process itself
  - Plasticity, interactivity, redundancy, compensation
- (2) Implemented models allow detailed consideration of computational constraints operating on development
- (3) Models are a necessary intermediate step to relate behavioural deficits to neural substrate and genetic influences on its development

Tricky question

- If a model of a developmental disorder always assumes a model of normal development, how can such disorders change our understanding of normal development?
- Good question. If a normal model cannot in principle explain an observed developmental deficit, then maybe the normal model is wrong
- But difficult to discover this without building the model
- *Build it and they will come*