

Causal networks in neural systems: from brain-based devices to consciousness

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Causal network analysis

Causal networks in brain-based devices

Consciousness

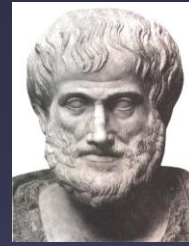
Synthetic and comparative approaches

Causal network analysis



Gods, people,
and animals

~50 AD



material, formal, efficient,
and final causes.

~350 BC

The Bible

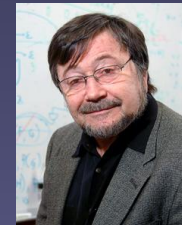
Aristotle

Hume



treatise on
human nature

Pearl



algebra of
intervention

Galileo

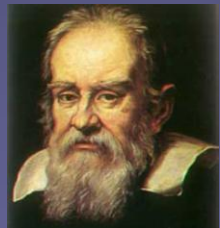
~1600

1740

1911

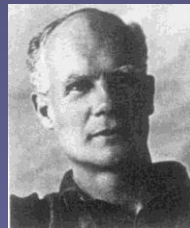
1936

2000

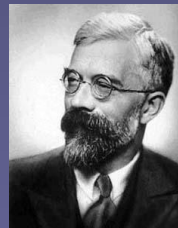


engineering
and algebra

Pearson

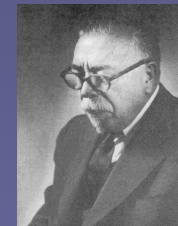


correlation,
not causation!



Fisher
randomized
experiment

Wiener



1954

Granger



1969

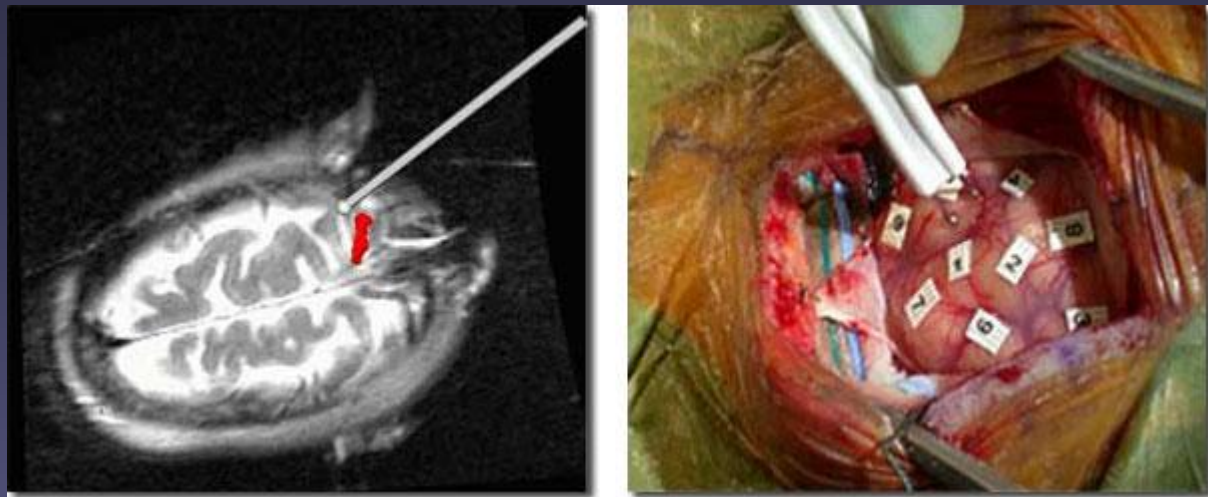
time series inference

“[the] development of Western science is based on two great achievements: the invention of the formal logical system ... and the possibility to find out causal relationships by systematic experiment”

Albert Einstein (1953)

Assessing causality

Stimulation



Ablation

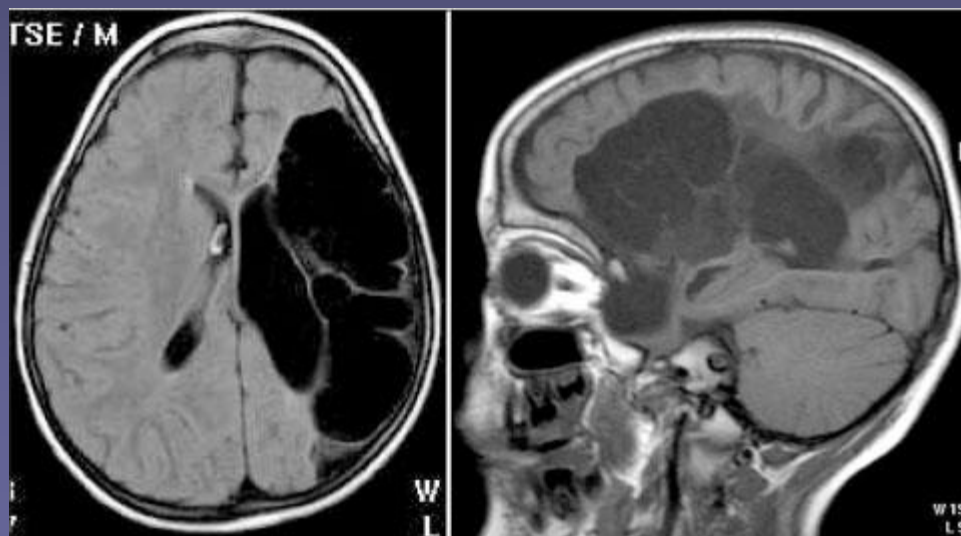
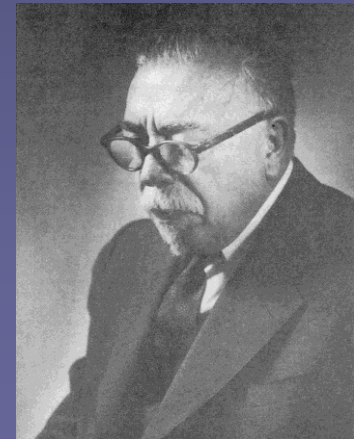


Fig 1. Female, 6 years old. MR showing left side multicystic encephalomalacia

Time series inference

- Causality based on temporal relations among recorded time series.
- No need for intervention/perturbation.
- Well (less badly) suited to complex, nested, hierarchical networks.
- Norbert Wiener: If knowing 'A' helps predict the future of 'B', then, in some sense, 'A' can be said to cause 'B'.



Granger (G-)causality

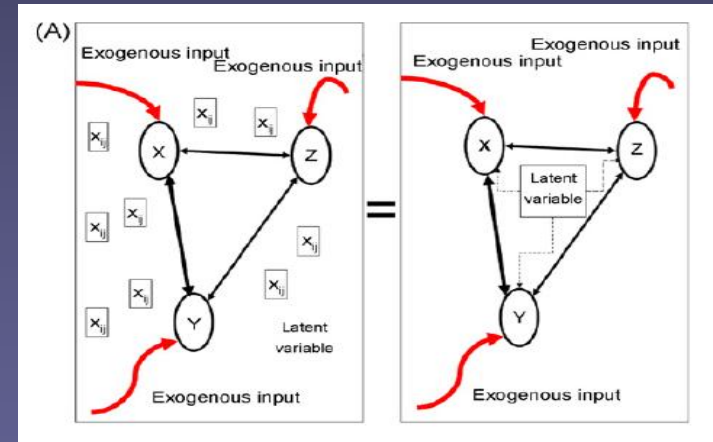
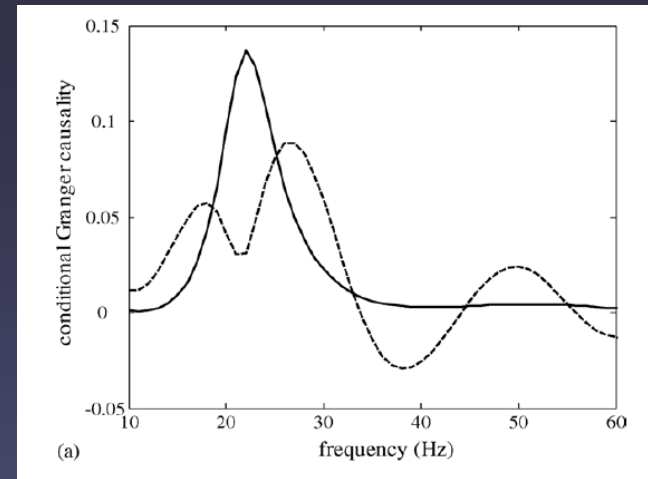
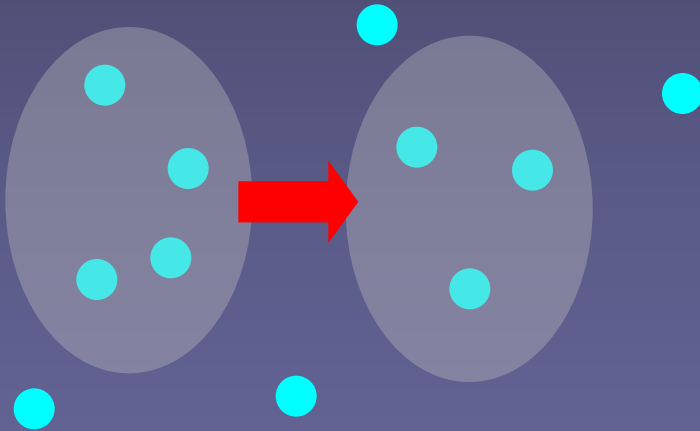
Causality based on prediction: If a signal X_1 causes a signal X_2 , then knowledge of the past of both X_1 and X_2 should improve the predictability of X_2 , as compared to knowledge of X_2 alone.

$$X_1(t) = \sum_{j=1}^m A_{11} X_1(t-j) + \xi_1(t)$$
$$X_2(t) = \sum_{j=1}^m A_{22} X_2(t-j) + \xi_2(t)$$

$$\mathcal{F}_{X_1 \rightarrow X_2} = \ln \frac{\text{var}(\varepsilon_t)}{\text{var}(\varepsilon'_t)}$$

G-causality extensions

- Spectral G-causality
- Partial G-causality
- Transfer entropy
- Multivariate G-causality



$$F_1 = \ln \left(\frac{|R_{XX|Z}^{(1)}|}{|R_{XX|Z}^{(2)}|} \right) = \ln \left(\frac{S_{11} - S_{12}S_{22}^{-1}S_{21}}{\Sigma_{11} - \Sigma_{12}\Sigma_{22}^{-1}\Sigma_{21}} \right)$$

Barrett, Barnett, & Seth (in press). *Phys. Rev. E*.

Barnett, Barrett, & Seth (2009). *Phys. Rev. Lett.*

Guo, Seth, et al (2008). *J. Neuro. Meth.* 172(1):72-93

Kaminski et al. (2001). *Biol Cyb.* 85:145-157.

G-causality extensions (2)

- G-autonomy
- G-emergence

$$X_1(t) = \sum_{j=1}^m A_{11} X_1(t-j) + \sum_{j=1}^m A_{12} X_2(t-j) + \varepsilon_1(t)$$

$$X_2(t) = \sum_{j=1}^m A_{21} X_1(t-j) + \sum_{j=1}^m A_{22} X_2(t-j) + \varepsilon_2(t)$$

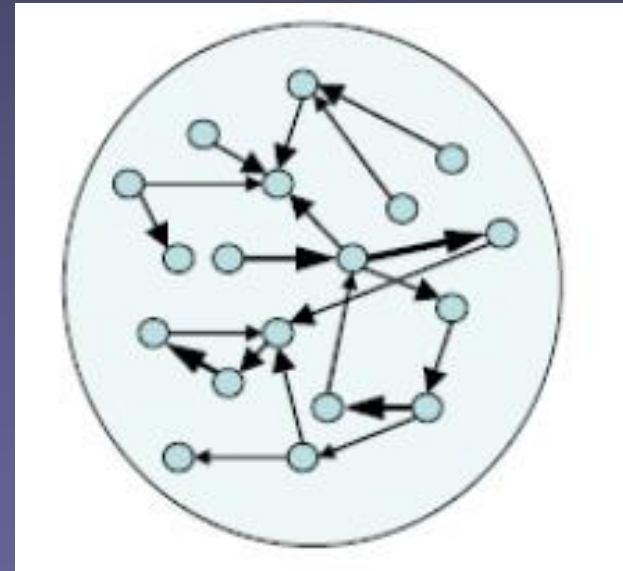
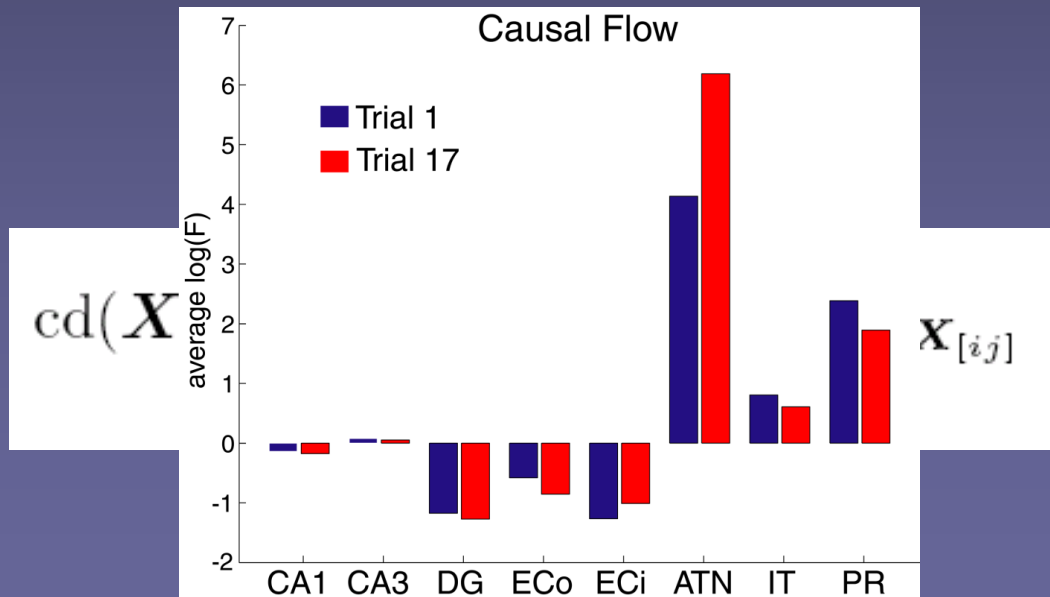
$$ga_{X_1|X_2} = \log \frac{\text{var}(\xi_{1R(11)})}{\text{var}(\xi_{1U})},$$



$$ge_{M|\mathbf{m}} = ga_{M|\mathbf{m}} \left(\frac{1}{N} \sum_{i=1}^N \mathcal{F}_{m_i \rightarrow M} \right)$$

Causal networks

- Represent G-causality interactions as a directed graph
- Allows useful summary statistics, e.g.,
 - Causal flow
 - Causal density

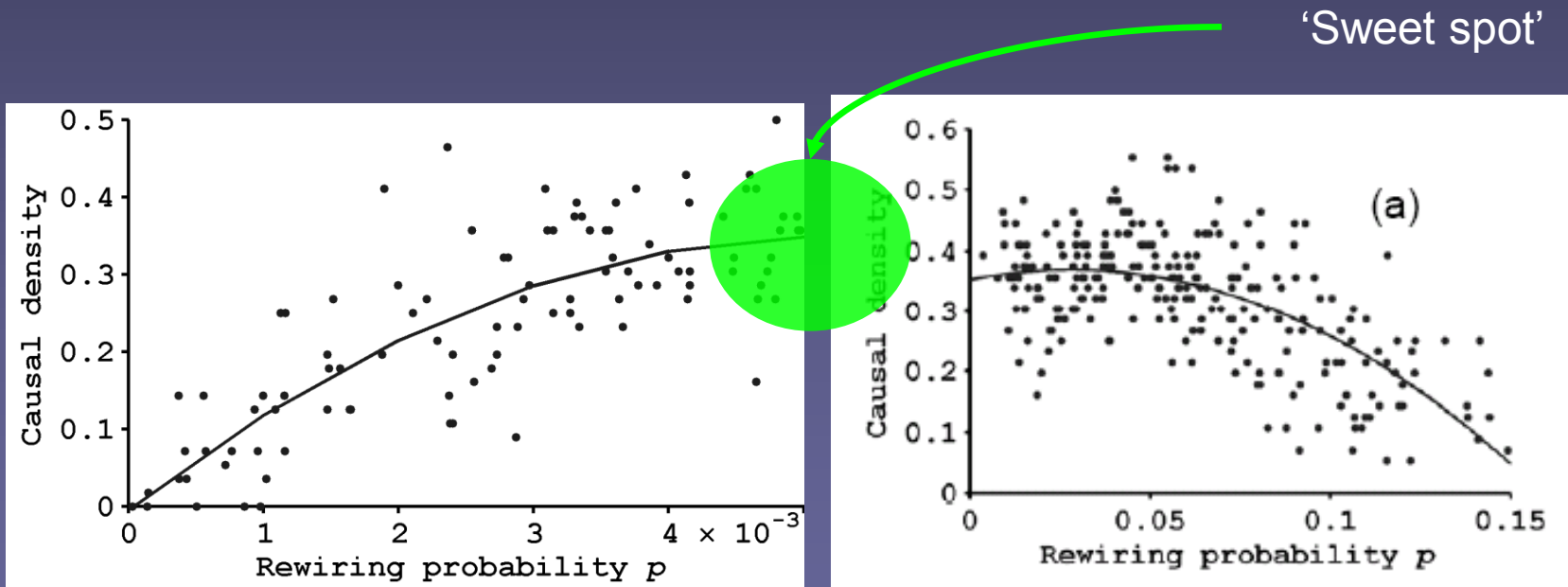


Seth, A. (2008). *Cogn. Neurodyn.* 2:49-64.

Seth, A. (2005). *Network: Comp. Neur. Sys.* 16(1):35-54.

Causal density

- Causal density provides a useful measure of **complexity**.
- Independent elements will have low causal density, as will elements that behave identically.




Do it yourself

- MATLAB toolbox containing multiple easy-to-use functions for implementing G-causality analysis.


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A MATLAB toolbox for Granger causal connectivity analysis

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ABSTRACT

Assessing directed functional connectivity from time series data is a key challenge in neuroscience. One approach to this problem leverages a combination of Granger causality analysis and network theory. This article describes a freely available MATLAB toolbox – ‘Granger causal connectivity analysis’ (GCCA) – which provides a core set of methods for performing this analysis on a variety of neuroscience data types including neuroelectric, neuromagnetic, functional MRI, and other neural signals. The toolbox includes core functions for Granger causality analysis of multivariate steady-state and event-related data, functions to preprocess data, assess statistical significance and validate results, and to compute and display network-level indices of causal connectivity including ‘causal density’ and ‘causal flow’. The toolbox is deliberately small, enabling its easy assimilation into the repertoire of researchers. It is however readily extensible given proficiency with the MATLAB language.

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Summary (1)

- Neural systems (or any complex time-varying system) can be analyzed in terms of causal networks (without assumptions of information processing, neural coding, etc.)
- G-causality does not require intervention/perturbation.
- Many extensions both to G-causality *per se* and to graph/network theoretic interpretations.

Causal networks in brain-based devices



The Neurosciences Institute, San Diego, CA

The Darwin series

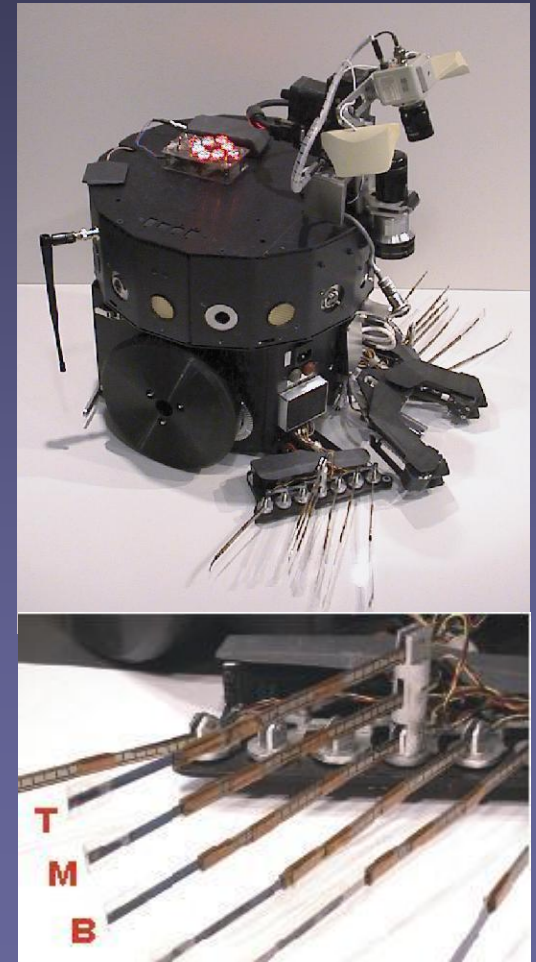
Darwin IV-VI
1992 - 1998



Darwin VII-VIII
1999 - 2002

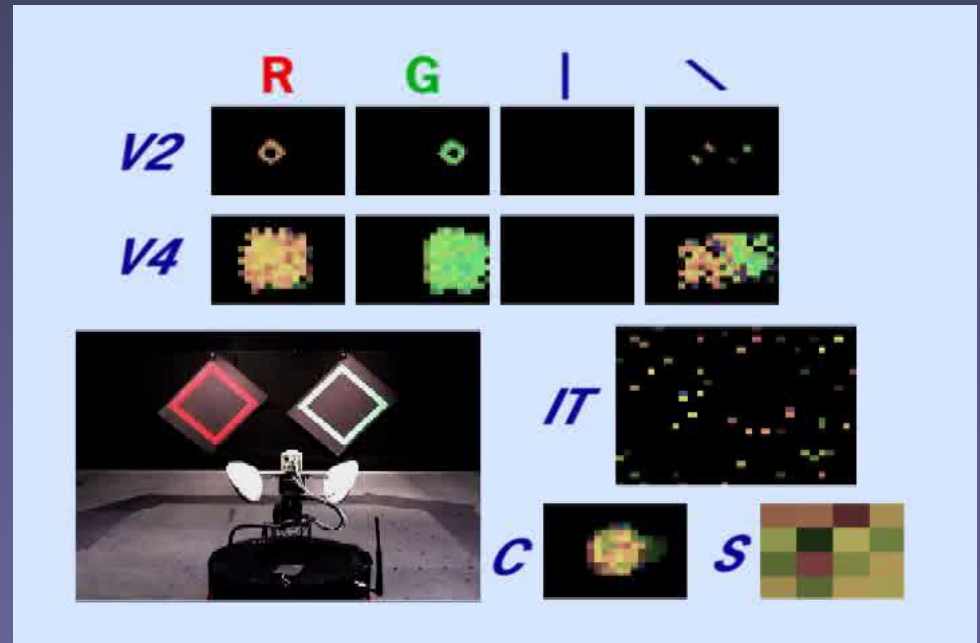
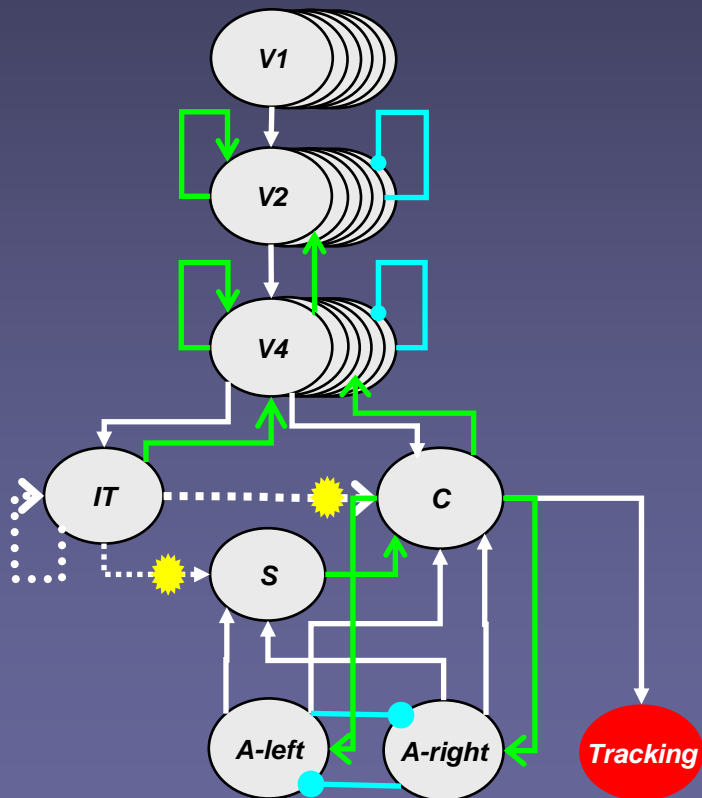


Darwin IX-XI
2003 - 2009



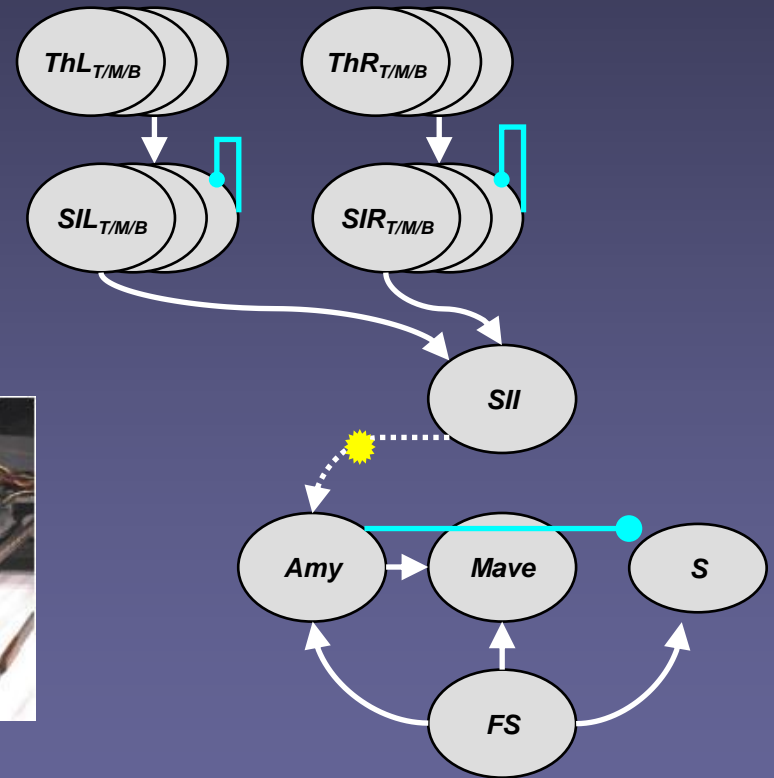
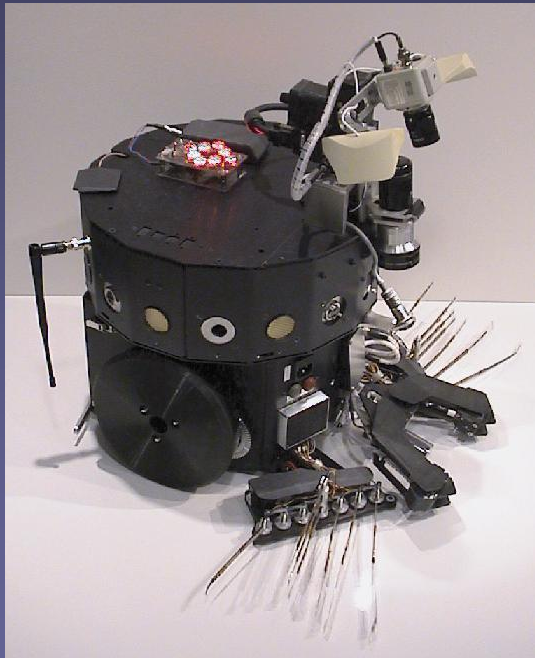
Darwin VIII: visual binding

- Visual binding as the result of the dynamic synchronization of neural activity via reentrant connections among distributed areas.

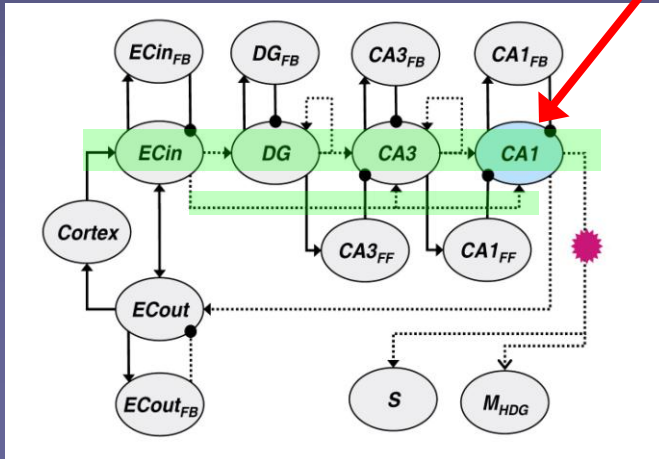
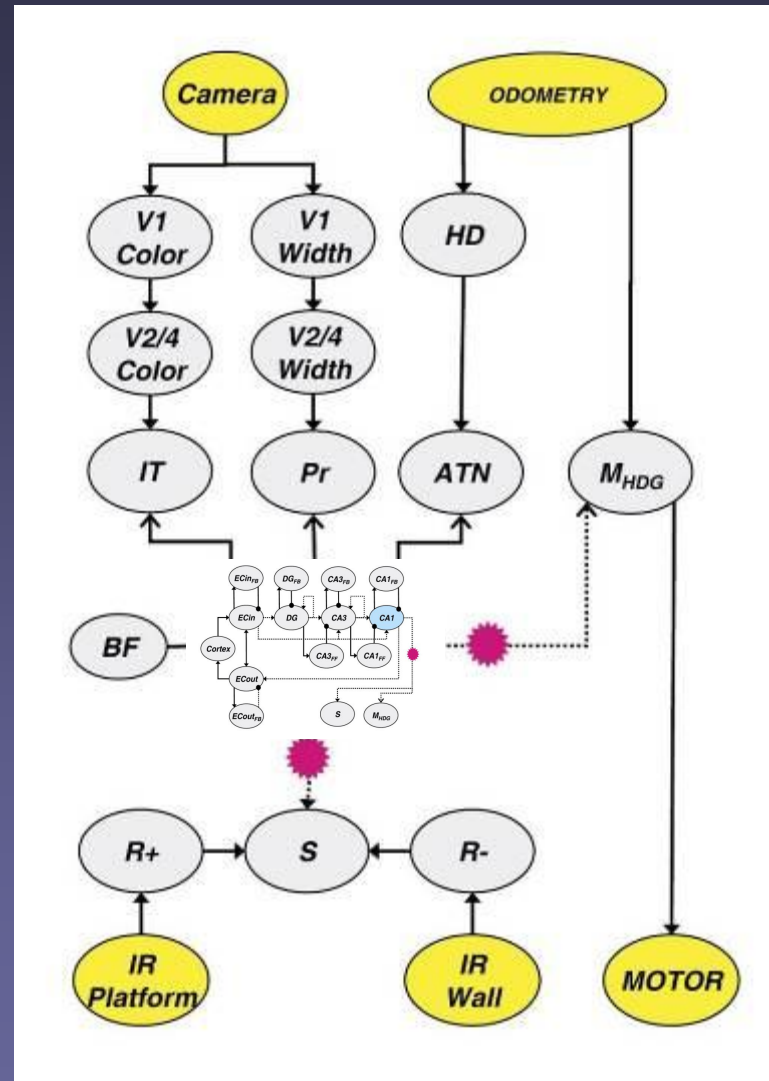
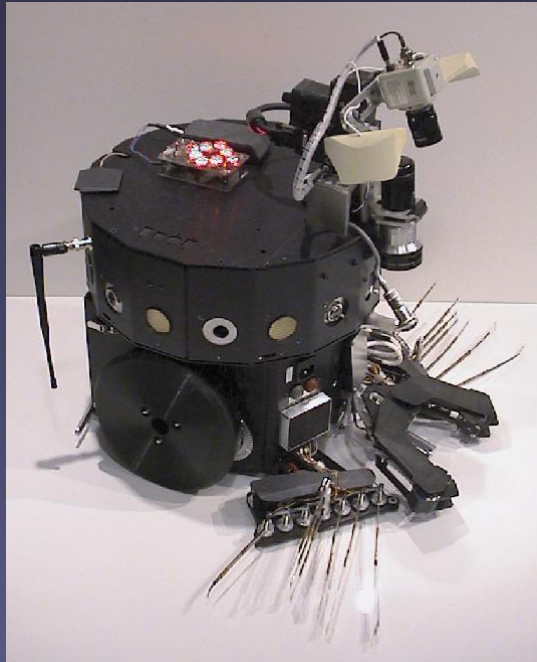


Darwin IX: haptic perception

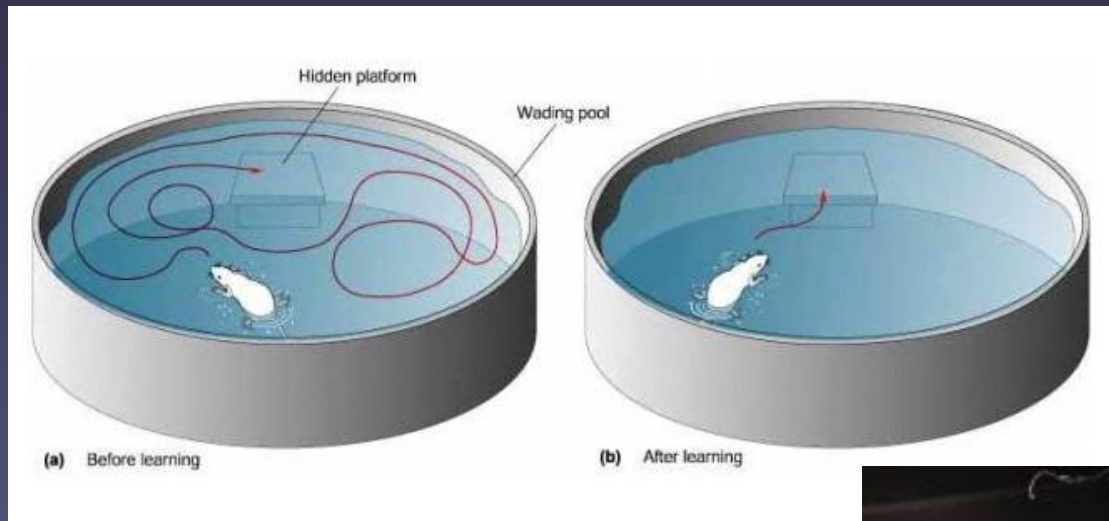
- Texture discrimination via spatiotemporally specific receptive fields and a model of aversive conditioning.



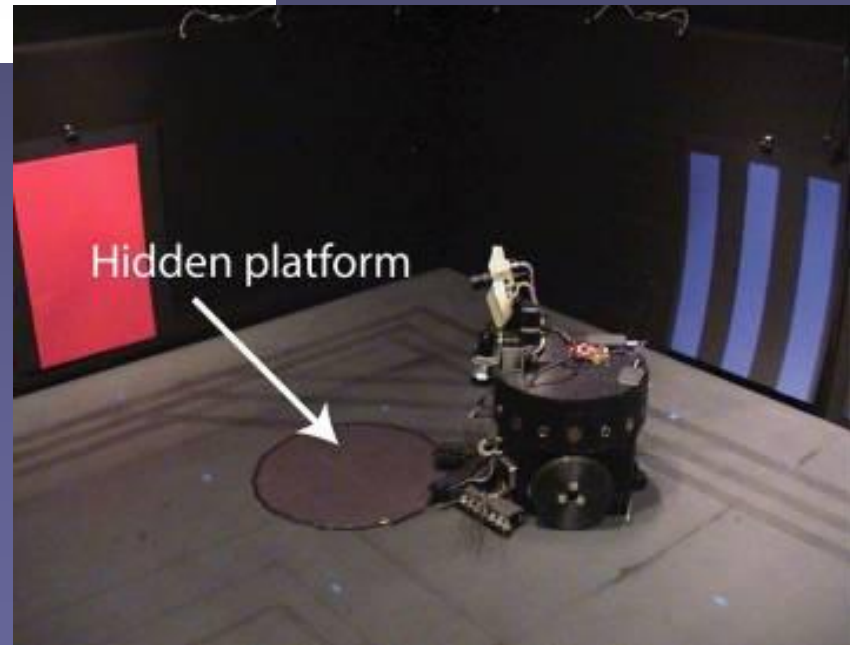
Darwin X: spatial navigation



Darwin X

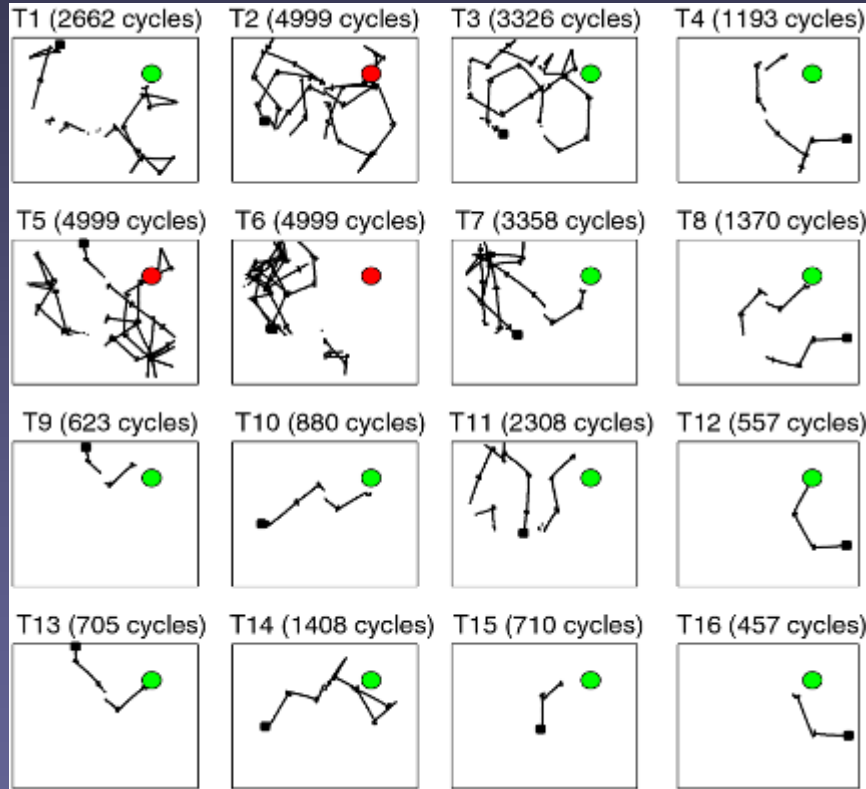


“Morris” water maze



Darwin X

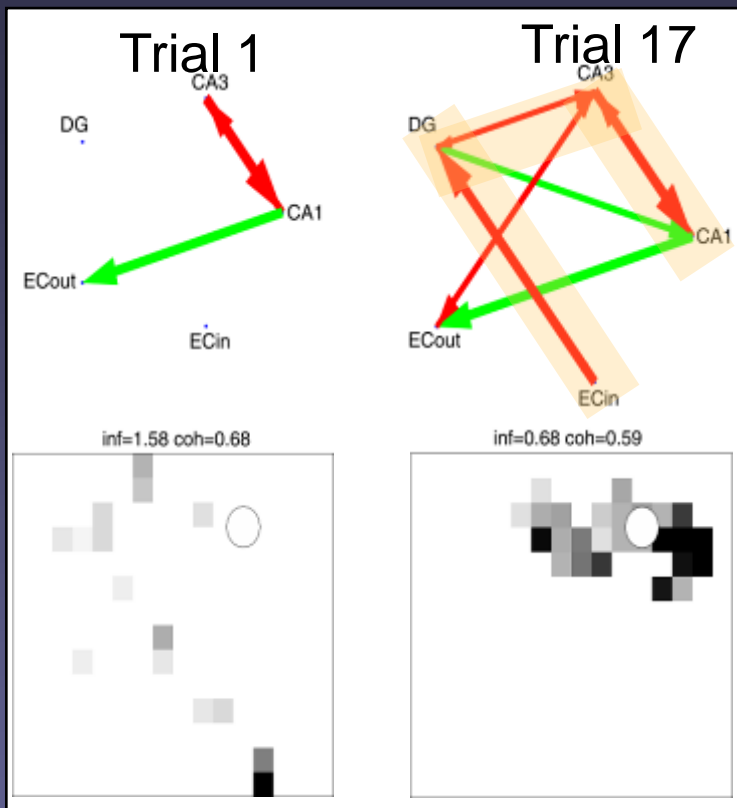
The device learns the task ...



... and develops 'place cells'

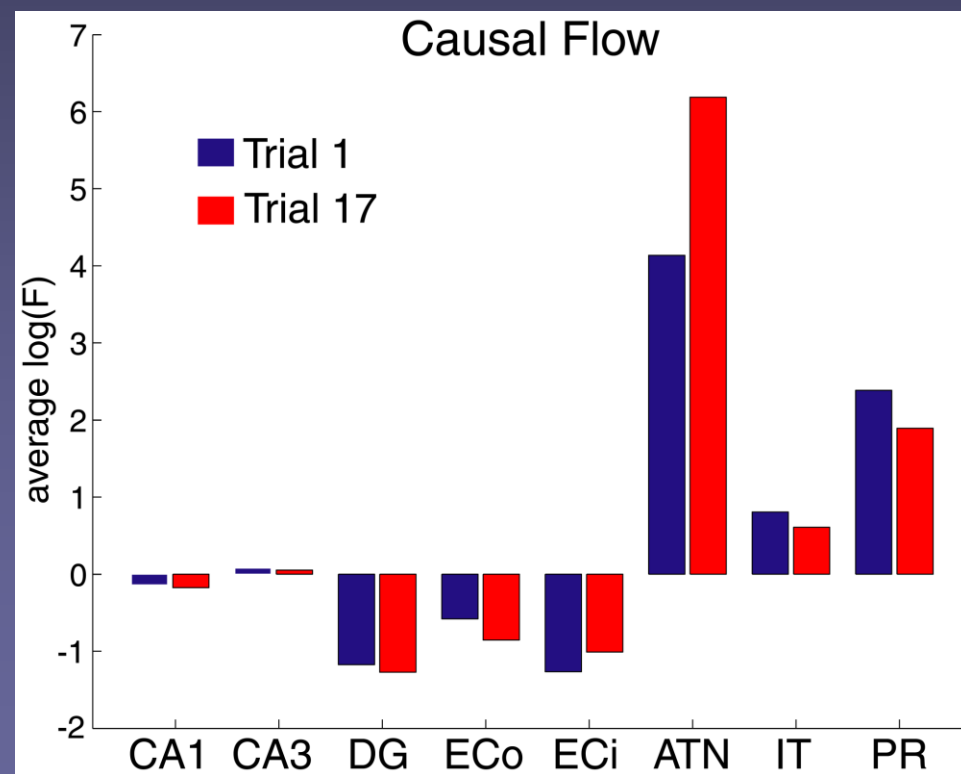


Darwin X



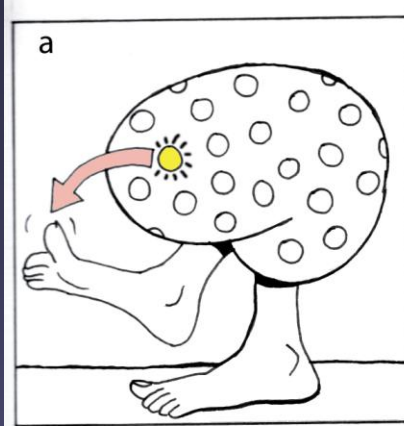
% of causal pathways

	Trial 1	Trial 17
Tri-synaptic	42.0	29.0
Perforant	14.8	21.6



Causal cores

a. Pick a 'Neural Reference' (NR)



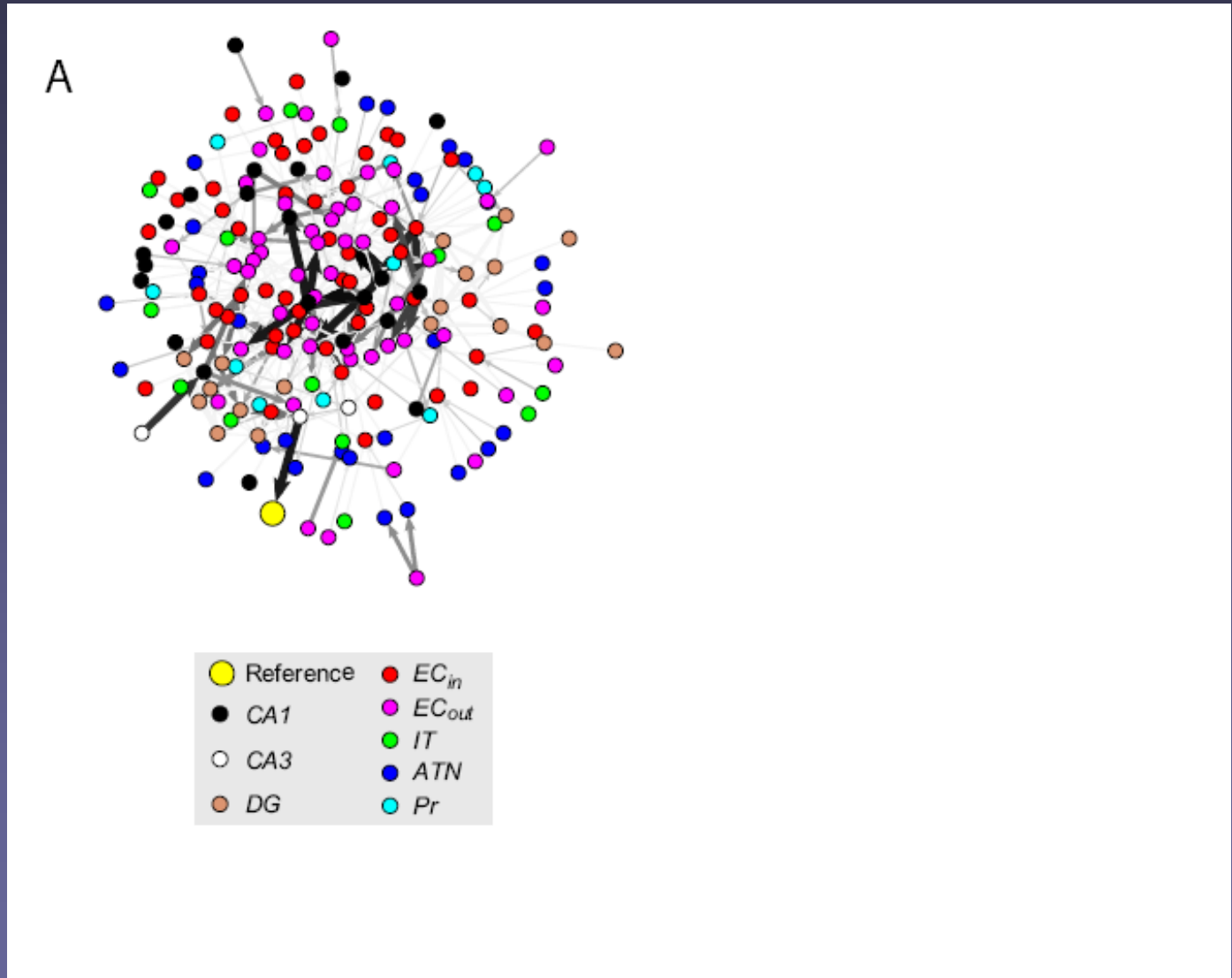
b. Identify the 'context network'

c. G-causality analysis for each synapse.

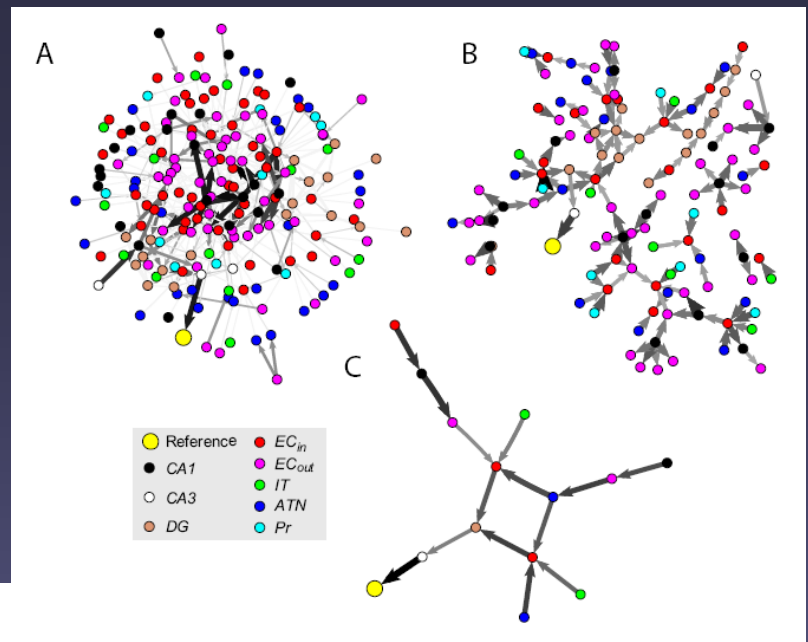
d. Identify causal core.

e. The causal core '*in vivo*'.

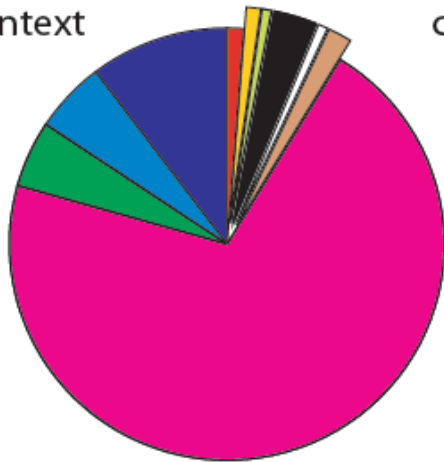
Causal cores



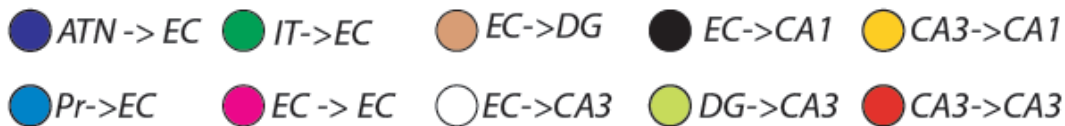
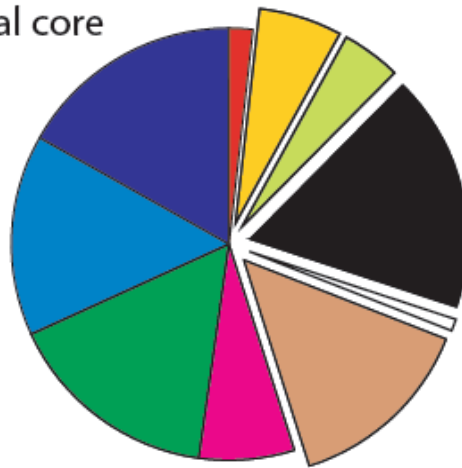
Causal cores



context

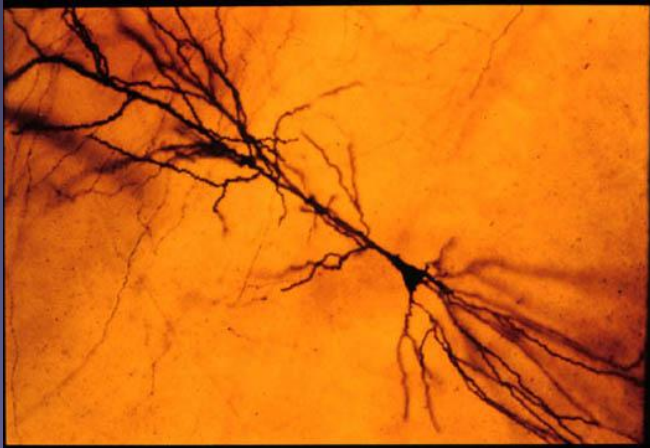


causal core



Multilevel dynamics of learning

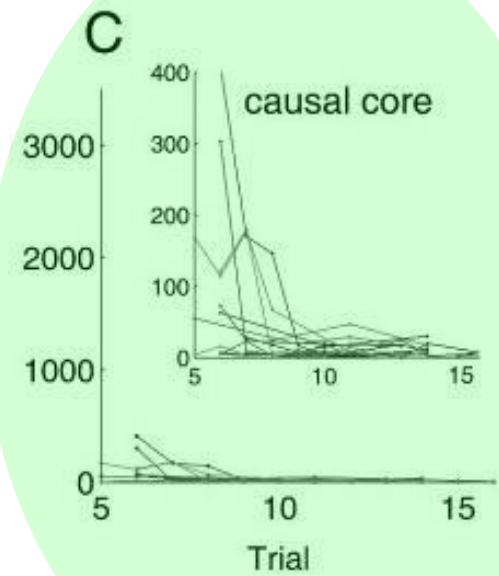
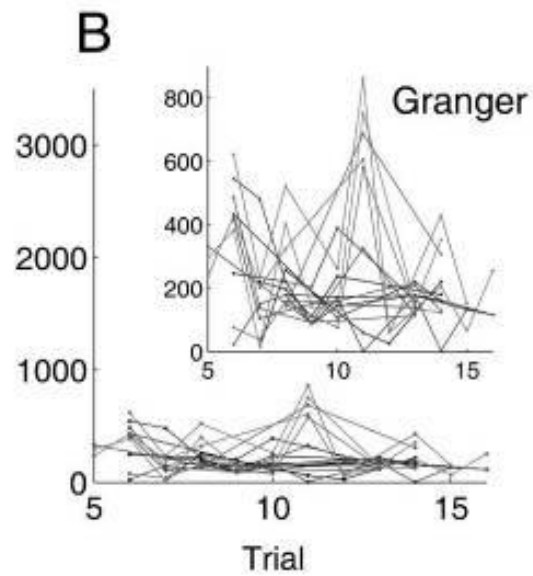
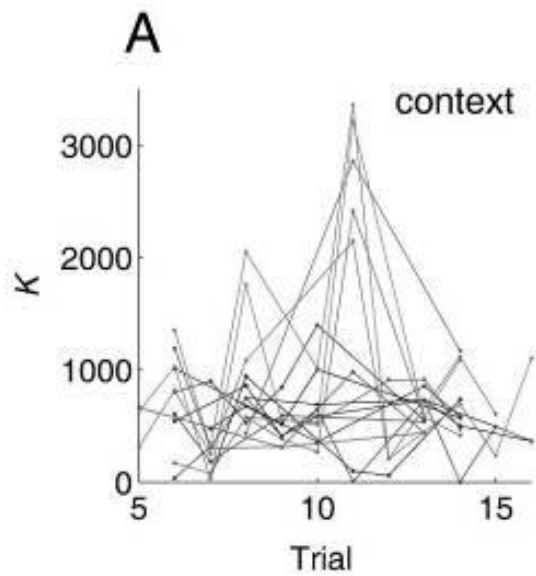
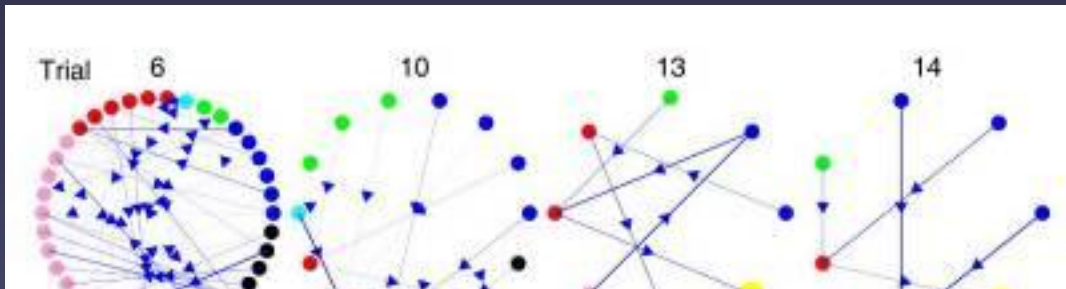
How to get from this ...



... to this



Causal cores



Summary (2)

- Brain-based devices (BBDs) implement large-scale simulated nervous systems as embodied, embedded agents.
- A BBD of the hippocampus shows spatial navigation learning accompanied by place cell formation.
- Causal network analysis of this BBD shows shifting functional connectivity during learning.
- ‘Causal core’ concept identifies simple pathways within complex networks: causal core refinement may connect synaptic plasticity with behavioral learning.

Consciousness

For centuries, debating the nature of consciousness was the exclusive purview of philosophers. But if the recent torrent of books on the topic is any indication, a shift has taken place: Scientists are getting into the game.

Has the nature of consciousness finally shifted from a philosophical question to a scientific one that can be solved by doing experiments? The answer, as with any related to this topic, depends on whom you ask. But scientific interest in this slippery, age-old question seems to be gathering momentum. So far, however, although theories abound, hard data are sparse.

The discourse on consciousness has been hugely influenced by René Descartes, the French philosopher who in the mid-17th century declared that body and mind are made of different stuff entirely. It must be so, Descartes concluded, because the body exists in both time and space, whereas the mind has no spatial dimension.

Recent scientifically oriented accounts of consciousness generally reject Descartes's solution; most prefer to treat body and mind as different aspects of the same thing. In this view, consciousness emerges from the properties and organization of neurons in the brain. But

ness entirely, leaving them in a coma or a persistent vegetative state. Although these regions may be a master switch for consciousness, they are unlikely to be its sole source. Different aspects of consciousness are probably generated in different brain regions. Damage to visual areas of the cerebral cortex, for example, can produce strange deficits limited to visual awareness. One extensively studied patient, known as D.F., is unable to identify shapes or determine the orientation of a thin slot in a vertical disk. Yet when asked to pick up a card and slide it through the slot, she does so easily. At some level, D.F. must know the orientation of the slot to be able to do this, but she seems not to know she knows.

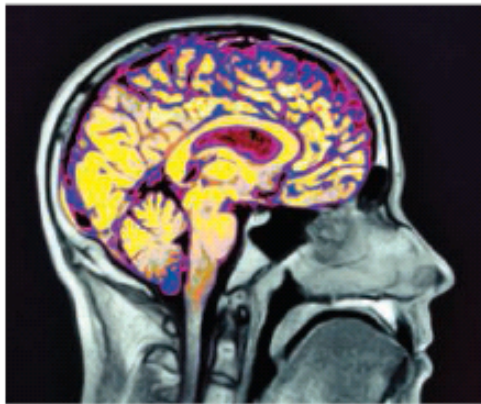
Cleverly designed experiments can produce similar dissociations of unconscious

hunt for neurons that track the monkey's perception, in hopes that these neurons will lead them to the neural systems involved in conscious visual awareness and ultimately to an explanation of how a particular pattern of photons hitting the retina produces the experience of seeing, say, a rose.

Experiments under way at present generally address only pieces of the consciousness puzzle, and very few directly address the most enigmatic aspect of the conscious human mind: the sense of self. Yet the experimental work has begun, and if the results don't provide a blinding insight into how consciousness arises from tangles of neurons, they should at least refine the next round of questions.

Ultimately, scientists would like to understand

What Is the Biological Basis of Consciousness



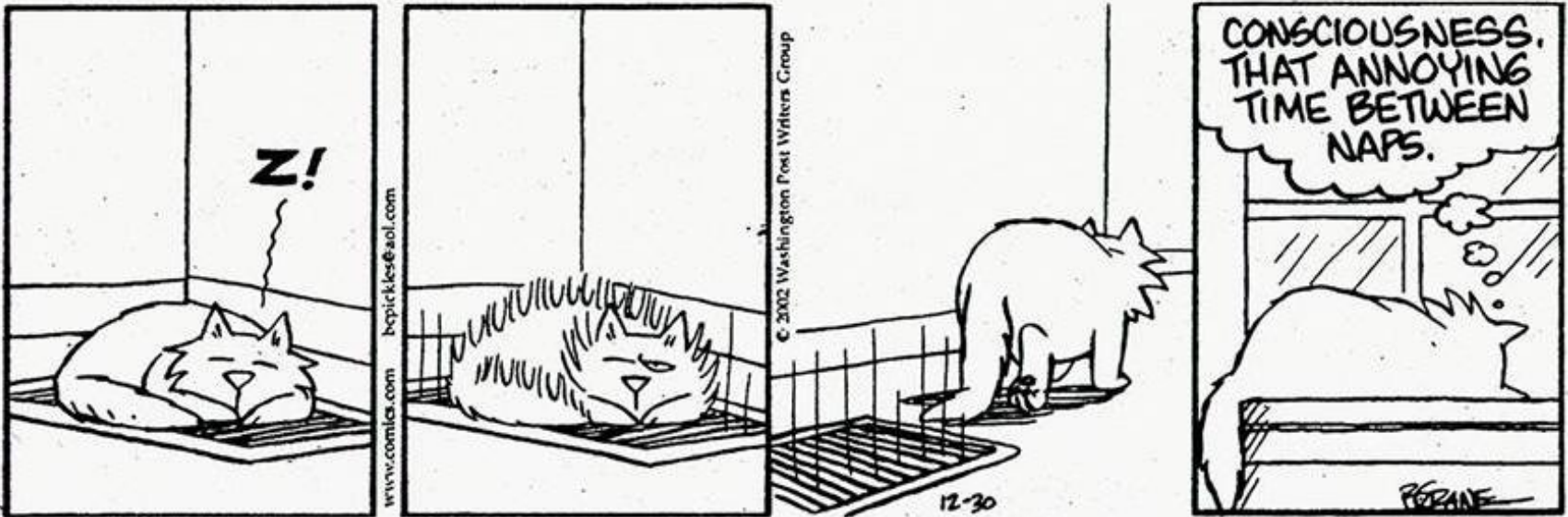
and conscious knowledge in people without neurological damage. And researchers hope that scanning the brains of subjects engaged in such tasks will reveal clues about the neural activity required for conscious awareness. Work with monkeys

not just the biological basis of consciousness but also why it exists. What selection pressure led to its development, and how many of our fellow creatures share it? Some researchers suspect that consciousness is not unique to humans, but of course much depends on how the term is defined. Biological markers for consciousness might help settle the matter and shed light on how consciousness develops early in life. Such markers could also inform medical decisions about loved ones who are in an unresponsive state.

“Consciousness is the appearance of a world.”

Metzinger (2009)

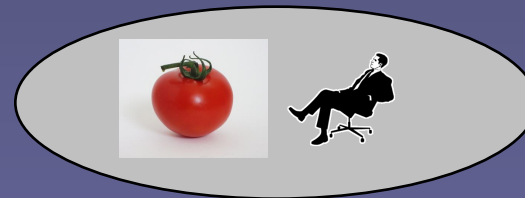
Pickles by Brian Crane



- Conscious *content* vs conscious *level*



- Primary* consciousness vs. *higher-order* consciousness

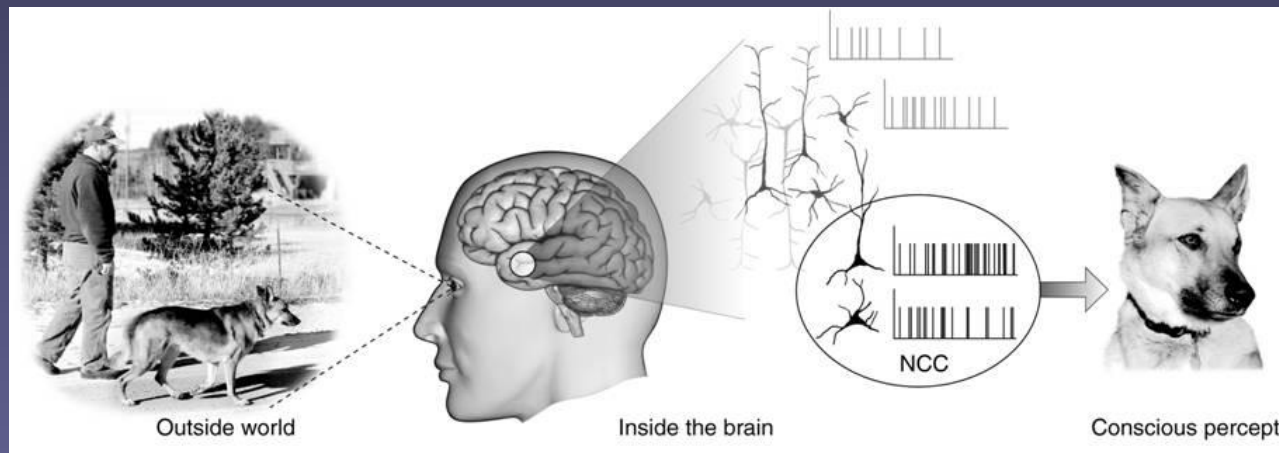


“tomato!”



Correlates of consciousness

- **Neural correlates:** activity in groups of neurons or brain regions that has a privileged relationship with consciousness.



Koch (2007), *Scholarpedia*

- **Explanatory correlates:** brain processes that account for fundamental (structural) aspects of conscious experience.



Dynamical complexity

Every conscious scene is *differentiated*

Every conscious scene is *integrated*



Gottfried Wilhelm Leibniz

“The most perfect world is the one in which there is the greatest diversity, in combination with the greatest order and harmony.”

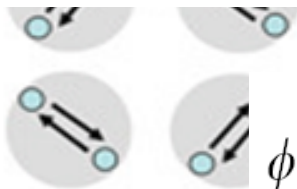
(only God can appreciate this)

Measuring complexity

- Neural complexity (Tononi, Sporns, Edelman, 1994)
- Information integration (Φ) (Tononi, 2004, 2008)
- Causal density (Seth, 2005, 2008)

$$C_N(\mathbf{X}) = \sum_k \langle MI(X_j^k; \mathbf{X} - X_j^k) \rangle,$$

neural complexity



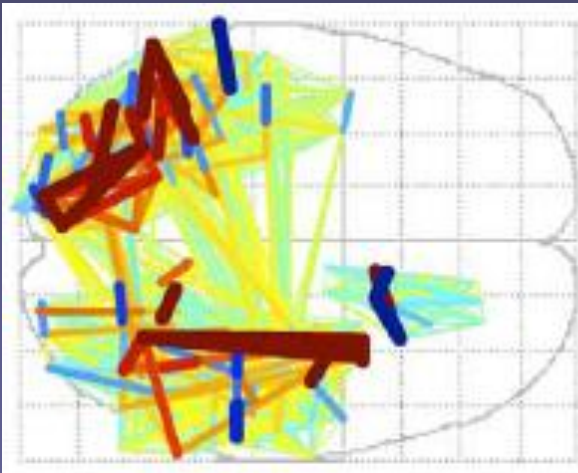
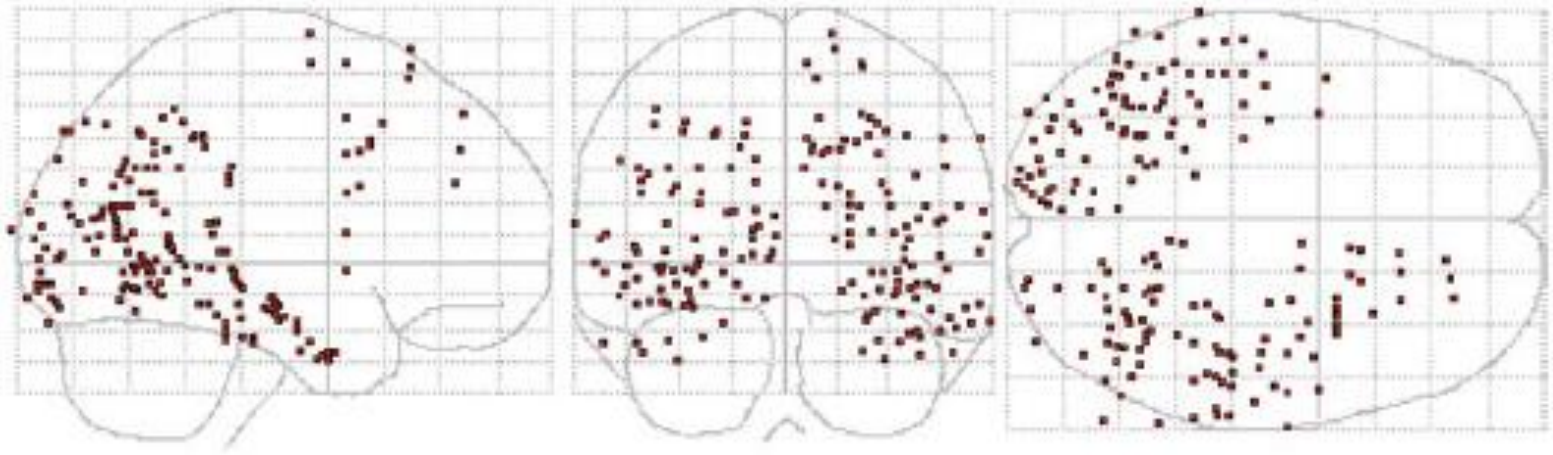
information integration

$$\phi(x_1) = H \left[p(X_0 \rightarrow x_1) \right] \left\| \prod_{M^k \in PMIP} p(\mathbf{M}_0^k \rightarrow \mu_1^k) \right]$$

$$cd(\mathbf{X}) \equiv \frac{1}{n(n-1)} \sum_{i \neq j} \mathcal{F}_{X_i \rightarrow X_j | \mathbf{X}_{[ij]}}$$

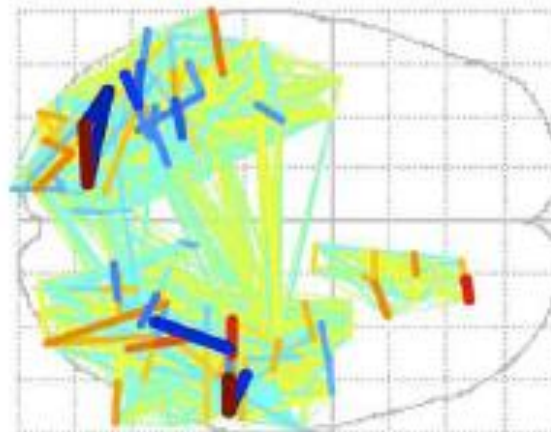


causal density

A

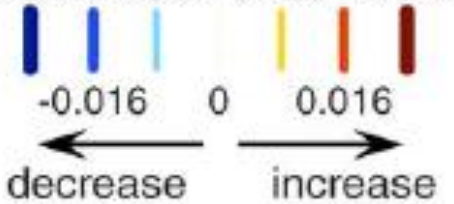
Unmasked
Causal gain

-0.024 -0.008 0.008 0.024

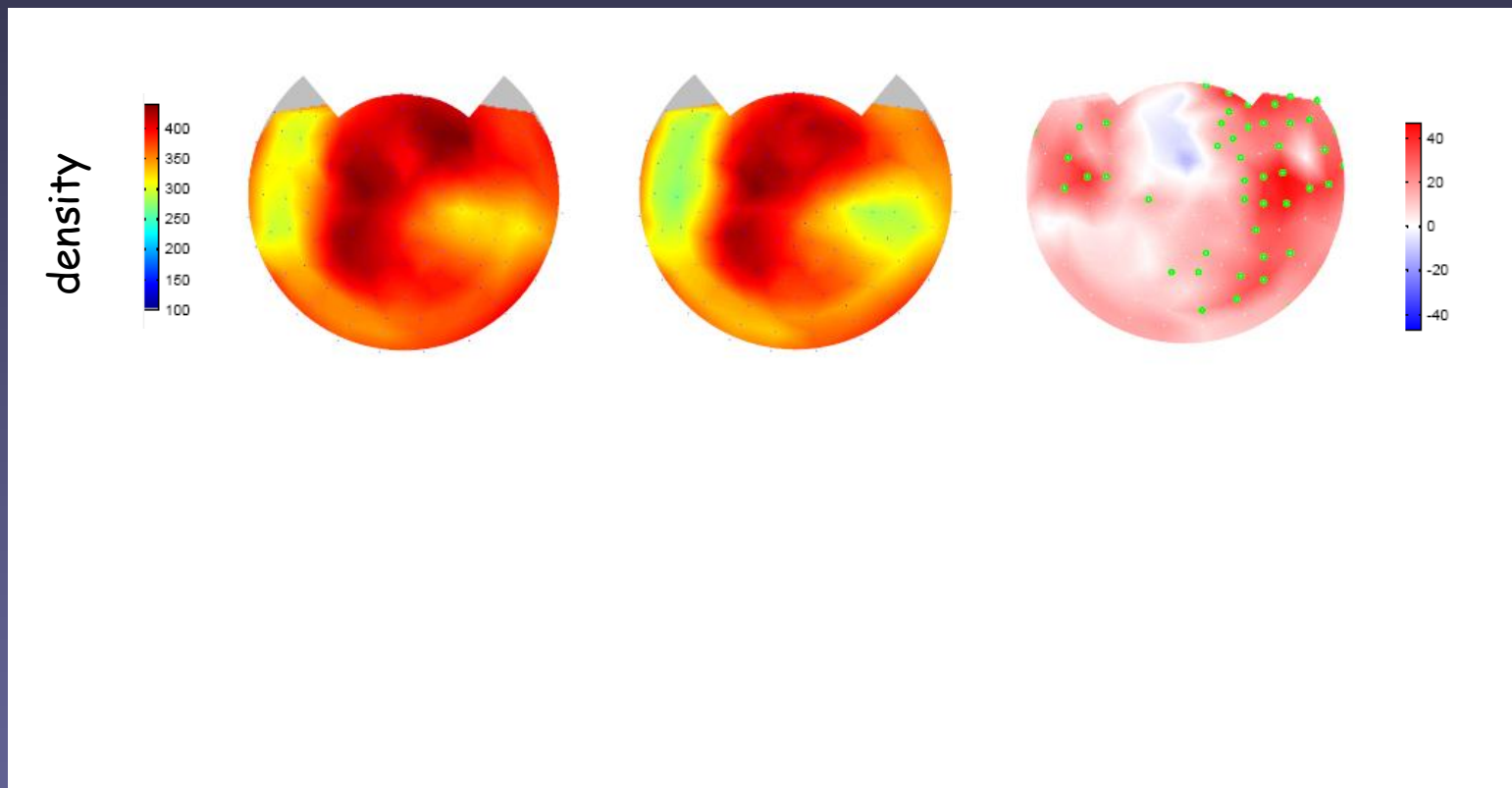


Masked
Causal gain

-0.024 -0.008 0.008 0.024



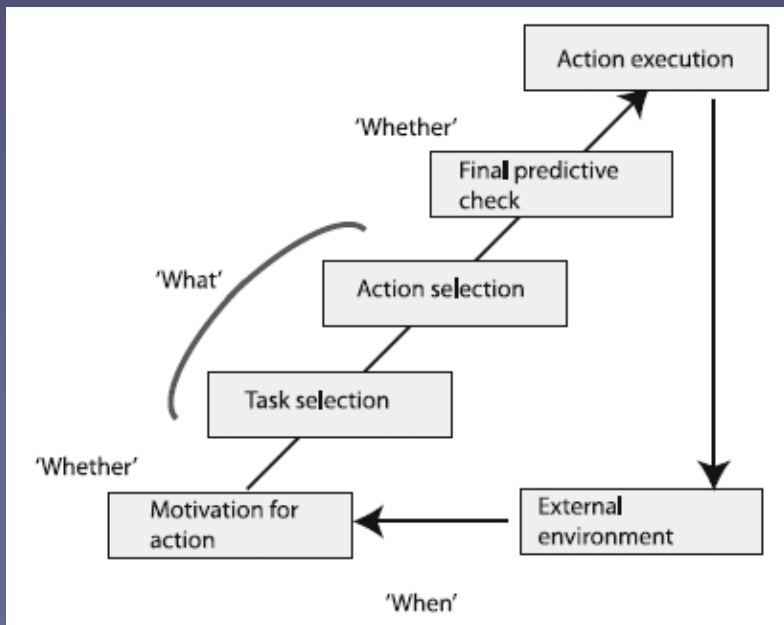
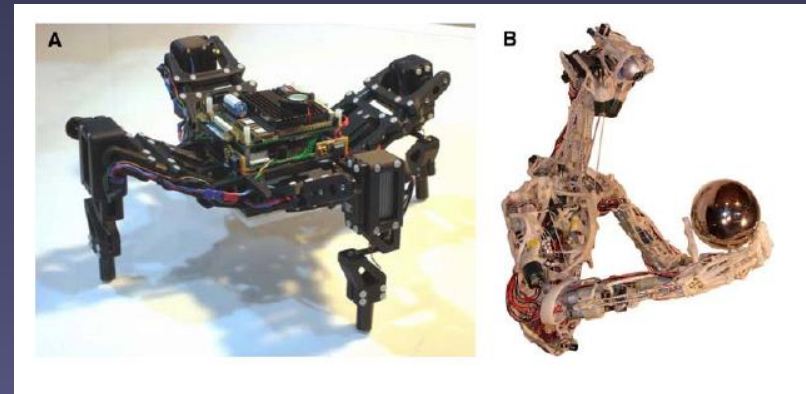
Causal density in MEG data



Moving on ...

- Theoretical/computational models should **account for** 'structural properties' of consciousness in terms of neural system dynamics.

- Perspectivalness
- Emotion/mood
- Volition/agency



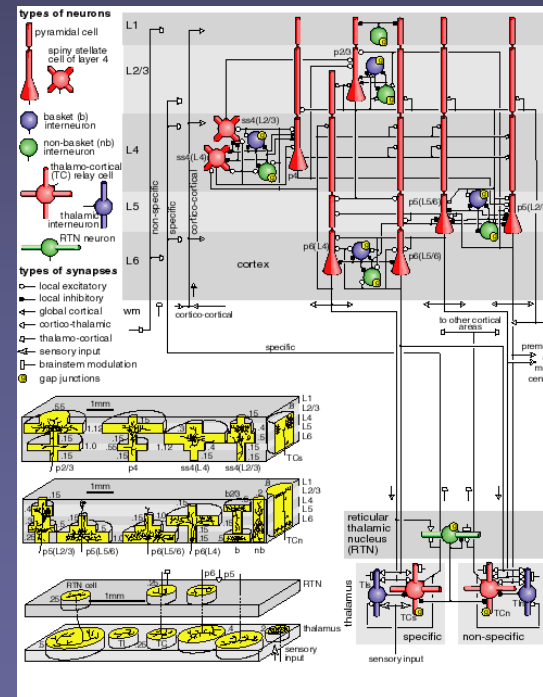
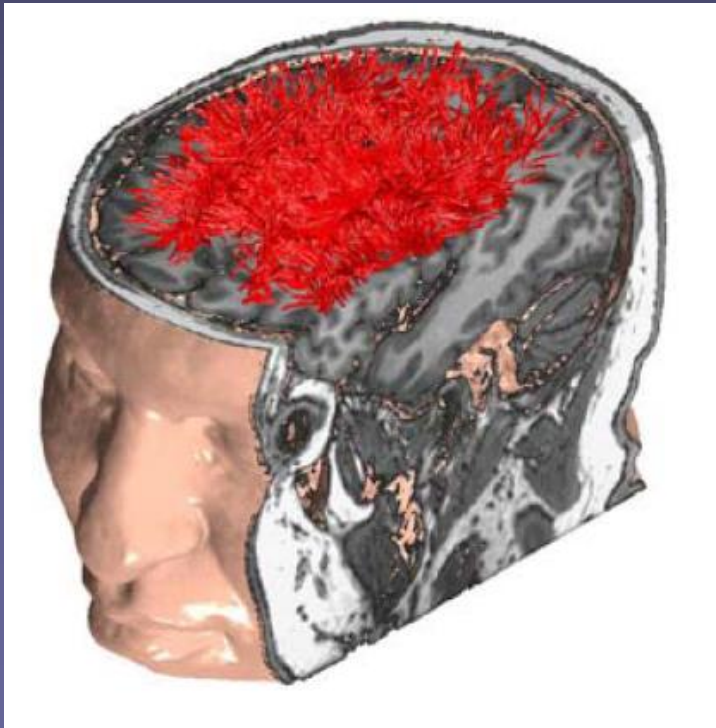
Summary (3)

- Consciousness: a legitimate scientific objective.
- Explanatory correlates aim to *account for* phenomenological properties in terms of neural processes and mechanisms.
- Causal density may account for the ‘dynamical complexity’ of conscious experience (thus providing a potential measure)
- Other ‘structural properties’ include agency, volition, perspectivalness, etc.

Synthetic and comparative approaches

Synthetic models

- Large-scale thalamocortical models
 - Refinement/extension of neural theories of consciousness
 - Generation of fine-grained predictions



Artificial /machine consciousness (AC)

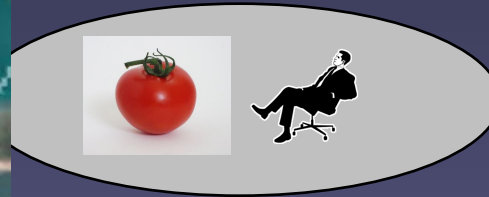
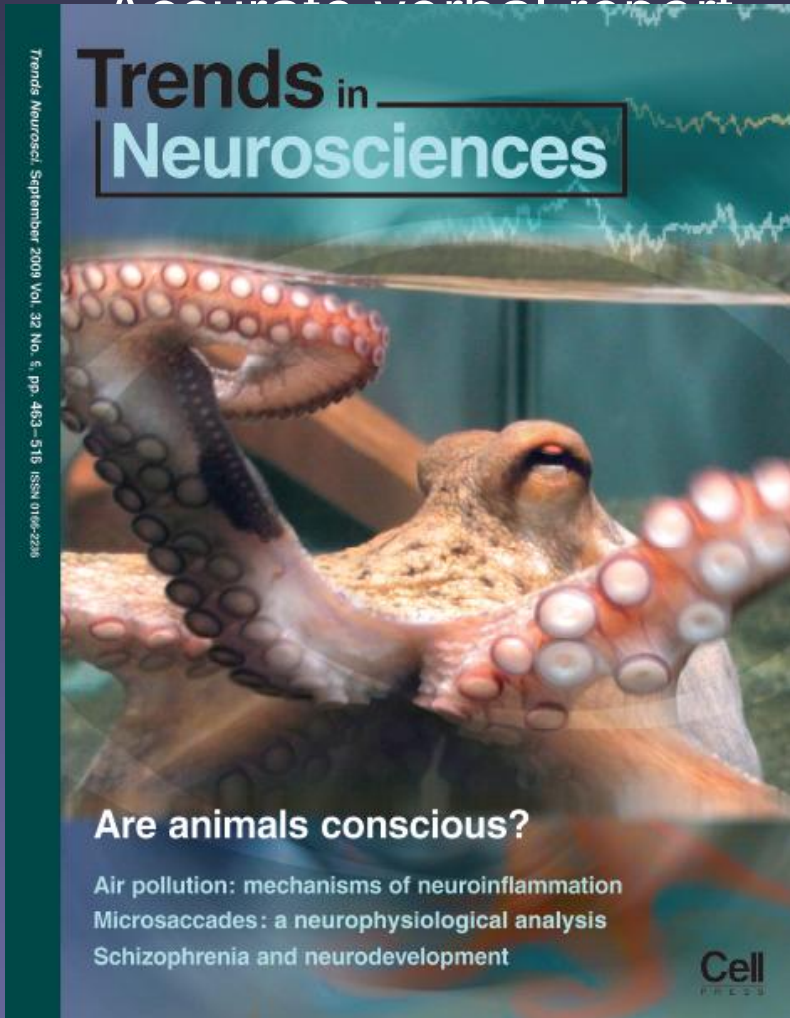
- Strong AC: instantiation
 - Axiomatic approaches (Aleksander, Tononi)
 - Status of data equal to biology (possible circularity)
 - Consciousness 'as it could be'? (deferral of comparisons)
- Weak AC: simulation
 - Refinement/prediction/interpretation of explanatory correlates
 - Transform *properties* into *criteria*
 - Show how apparently distinct properties arise from common mechanisms?
 - Will weak AC inevitably lead to strong AC?

Animal consciousness



- *Octopus vulgaris* in action

Accurate verbal reports: the **gold** standard:



“tomato!”



“
—
”

Edelman D.B., & Seth (2009). *Trends. Neurosci.*
Seth, Baars, & Edelman D.B. (2005). *Consc. Cogn.*

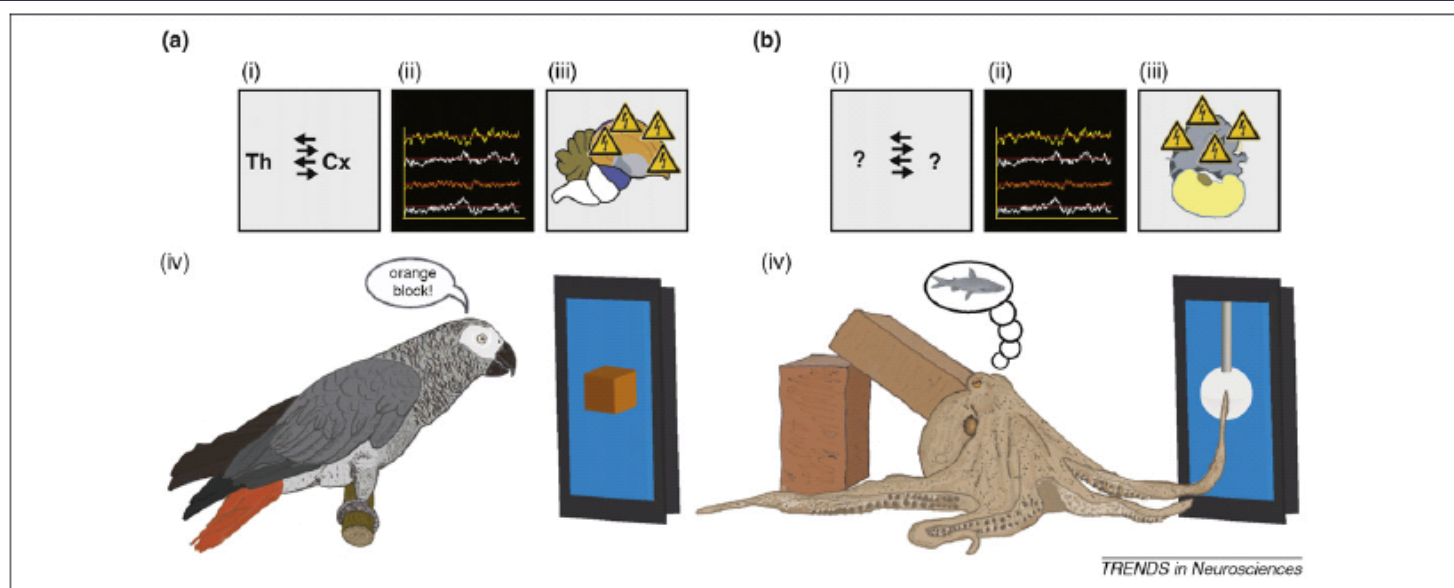
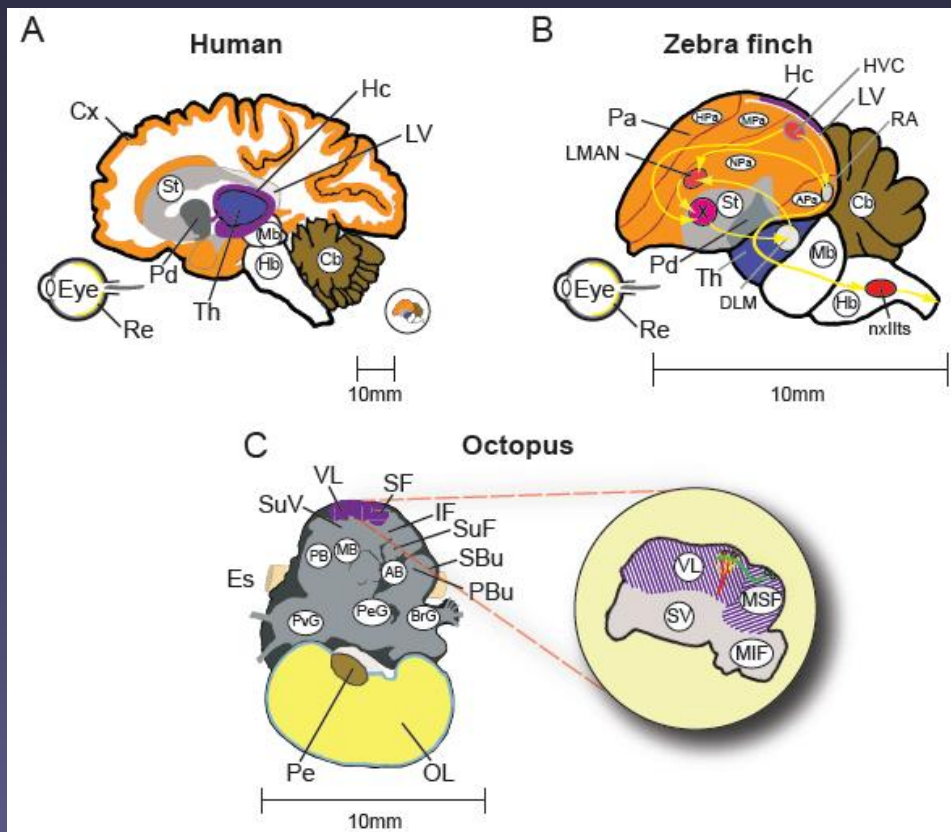


Figure 1. The investigation of possible conscious states in non-human species as disparate as birds (a) and cephalopods (b) can be informed by searching for neural properties that have been correlated with consciousness in humans, including reentrant signaling between thalamus and cortex (a, i) or putative functional analogs (b, i), fast, irregular, low-amplitude EEG signals (a and b, ii), and widespread electrical activity in cortex (a, iii) or functionally analogous structures (b, iii). Such processes in animals can best be related to consciousness when they can be correlated with accurate reports. Relevant forms of report include vocalizations in the case of African grey parrots (a, iv) or coloration and body patterning in the case of cephalopods (b, iv). In the figure, an African grey parrot and a common octopus (*O. vulgaris*) respond to salient artificial stimuli presented on video displays: an orange block in a discrimination task (a, iv) and a white ball that has been previously associated with food (herring) during training (b, iv) (see Box 1).

- Use humans as a benchmark to derive (and apply) behavioral, cognitive, and neural criteria.
- Relevant neural evidence must *account for* phenomenal properties.
- Mammalian case (relatively) uncontroversial (!), but more challenging are e.g. birds and octopuses.



- **Birds:** Homologous neural circuitry (striatopallidothalamic) underlying motor sequencing (mammals) and vocal learning (birds). Dorsal ventricular ridge may be homologous to 6-layer mammalian cortex.
- **Octopus:** ~500 million neurons; lobular organization; preserved neurotransmitters (5-HT, DA etc); *foxP2* expression in chromatophore lobes; evidence for LTP.

Conclusions

- Causal network analysis (CNA) is a powerful framework for analyzing both biological and artificial systems, and for translating insights between domains.
- Applied to 'brain-based-devices', CNA predicts aspects of the functional architecture of hippocampal mediation of spatial navigation (and, separately, tactile perception, and visual binding ...)
- Applied to consciousness, CNA can operationalize explanatory correlates (e.g., dynamical complexity).
- Synthetic approaches (artificial consciousness) can transform properties into criteria.
- A major challenge: consciousness in animals (and in infants, and in vegetative patients ...)

US

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Sackler Centre for Consciousness Science



Anil K. Seth D.Phil.

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Research Interests:

Theoretical Neuroscience, [Consciousness](#), Cognitive Science, Artificial Life, Neurobotics



I was born in Oxford, England. After receiving a First from [Cambridge University](#) (UK) in Natural Sciences, I gained an M.Sc. with distinction from [Sussex University](#). I received my D.Phil. also from Sussex on the subject of artificial evolution. In 2001 I joined [The Neurosciences Institute](#), La Jolla, California, where I was a Postdoctoral fellow and then an Associate Fellow. As of October 2006 I am back at Sussex on the [Informatics](#) faculty.

26 June 2008: Awarded an EPSRC [Leadership Fellowship](#), value ~GBP 1.3 million, 'Towards a [next-generation computational neuroscience](#)'. Postdoctoral/PhD opportunities to follow.

www.sussex.ac.uk/sackler

www.anilseth.com

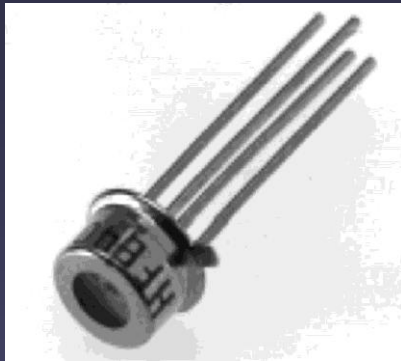
www.frontiersin.org/consciousnessresearch/

Sackler Centre opening event: April 21st, 3pm-8pm

NSI: Jeff Krichmar, Gerald Edelman, Jeff McKinstry, Jason Fleischer

Sussex: Adam Barrett, Lionel Barnett

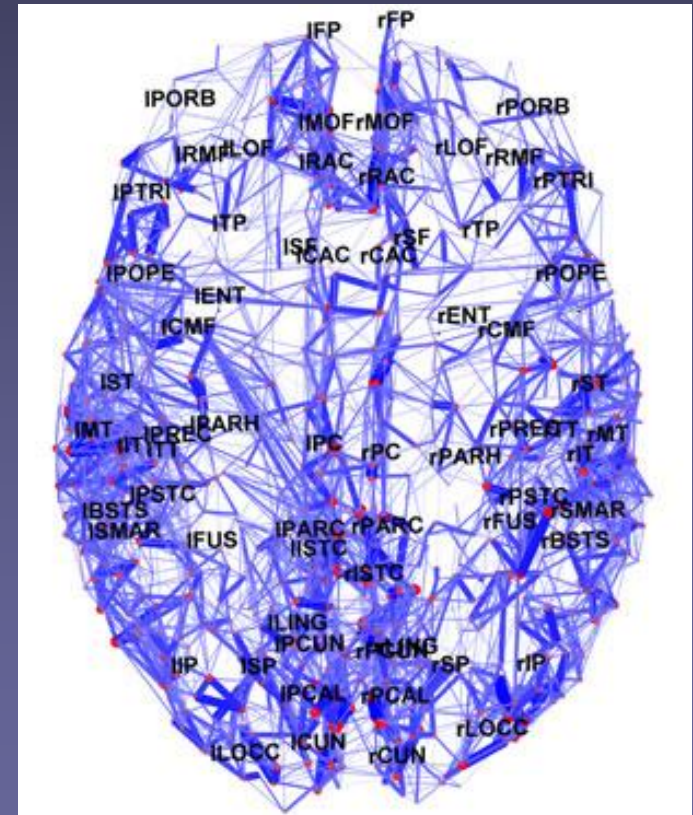
Funding: Neuroscience Research Foundation, EPSRC, Dr. Mortimer and Theresa Sackler Foundation



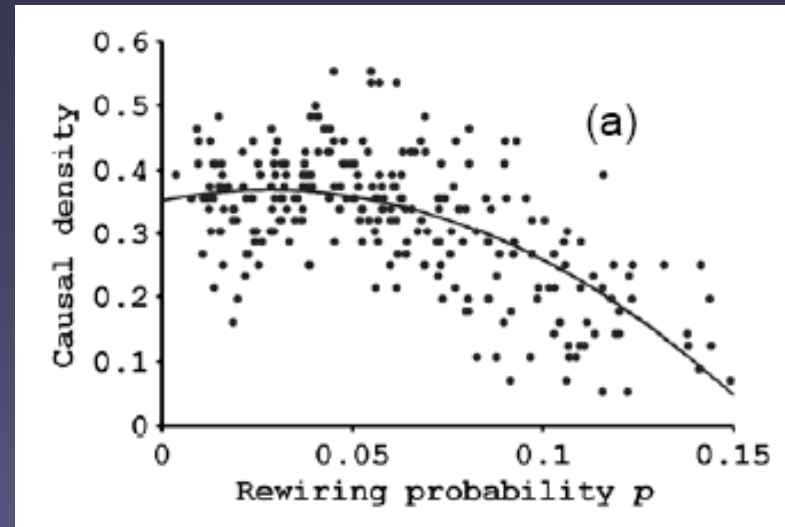
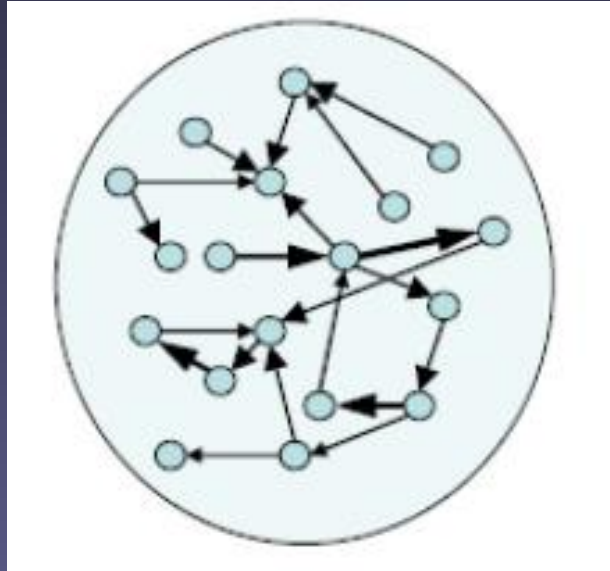
not differentiated

not integrated

differentiated *and* integrated



Causal density



$$\text{cd}(\mathbf{X}) \equiv \frac{1}{n(n-1)} \sum_{i \neq j} \mathcal{F}_{X_i \rightarrow X_j | \mathbf{X}_{[ij]}}$$