Is Psychology Ineliminable from Psychiatry, in Finding the Causal Structure of Disorders?

Three Types of Integration

Topics

1. Control Panels for Disorders
2. The Vision Science Model: Marr's 3 Levels
3. Control Variables for Psychological Clusters?

‘A central conundrum of the field is how to **integrate** this cacophony of scientific perspectives into a meaningful whole’

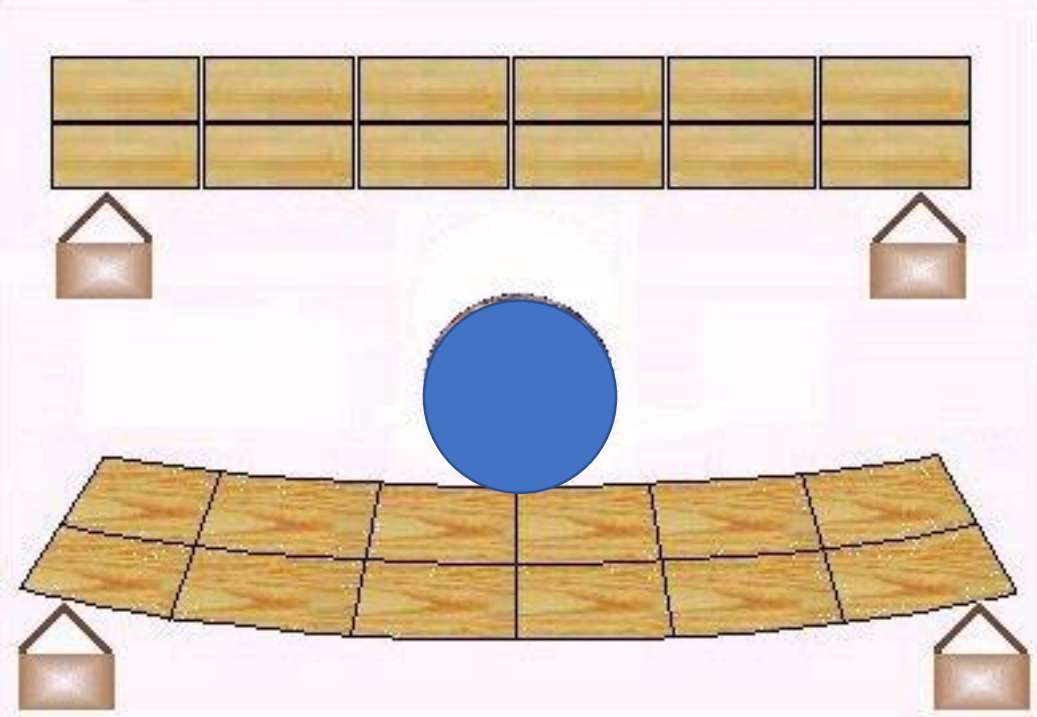
(conference web-site, my emphasis)

For the moment, not talk about integration, but only about how to get from correlation to causation

Variables proposed as implicated in alcohol dependence: (from Kendler (2012))

(1) Latent genetic risk,
(2) ALDH (a group of enzymes implicated in alcohol oxidation) variants,
(3) variants in the GABA (a neurotransmitter) receptor system,
(4) childhood sexual abuse,
(5) frontal lobe dysfunction,
(6) impulsivity,
(7) peer deviance,
(8) social norm expectations,
(9) taxation

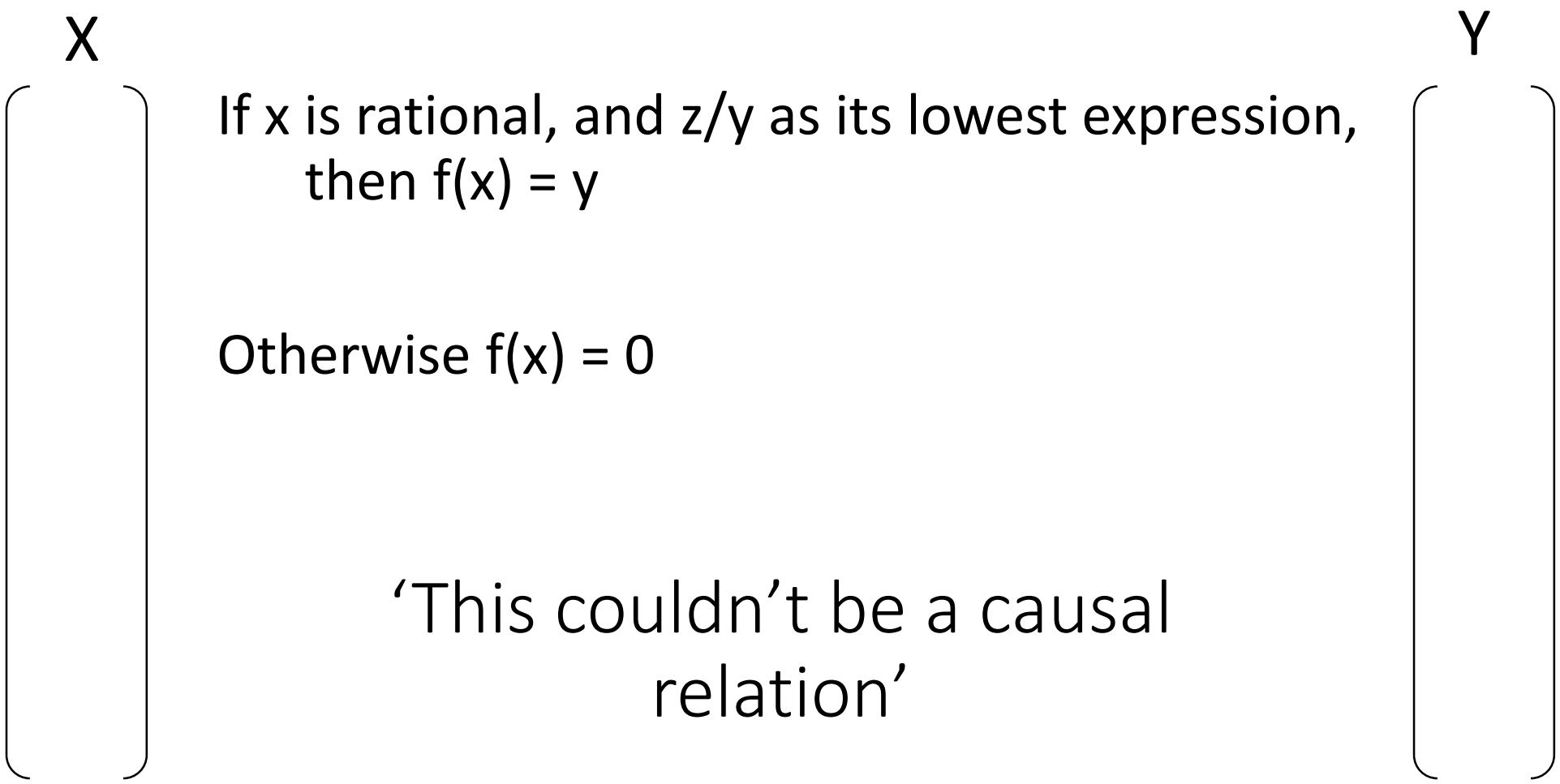
Why did the beam break?



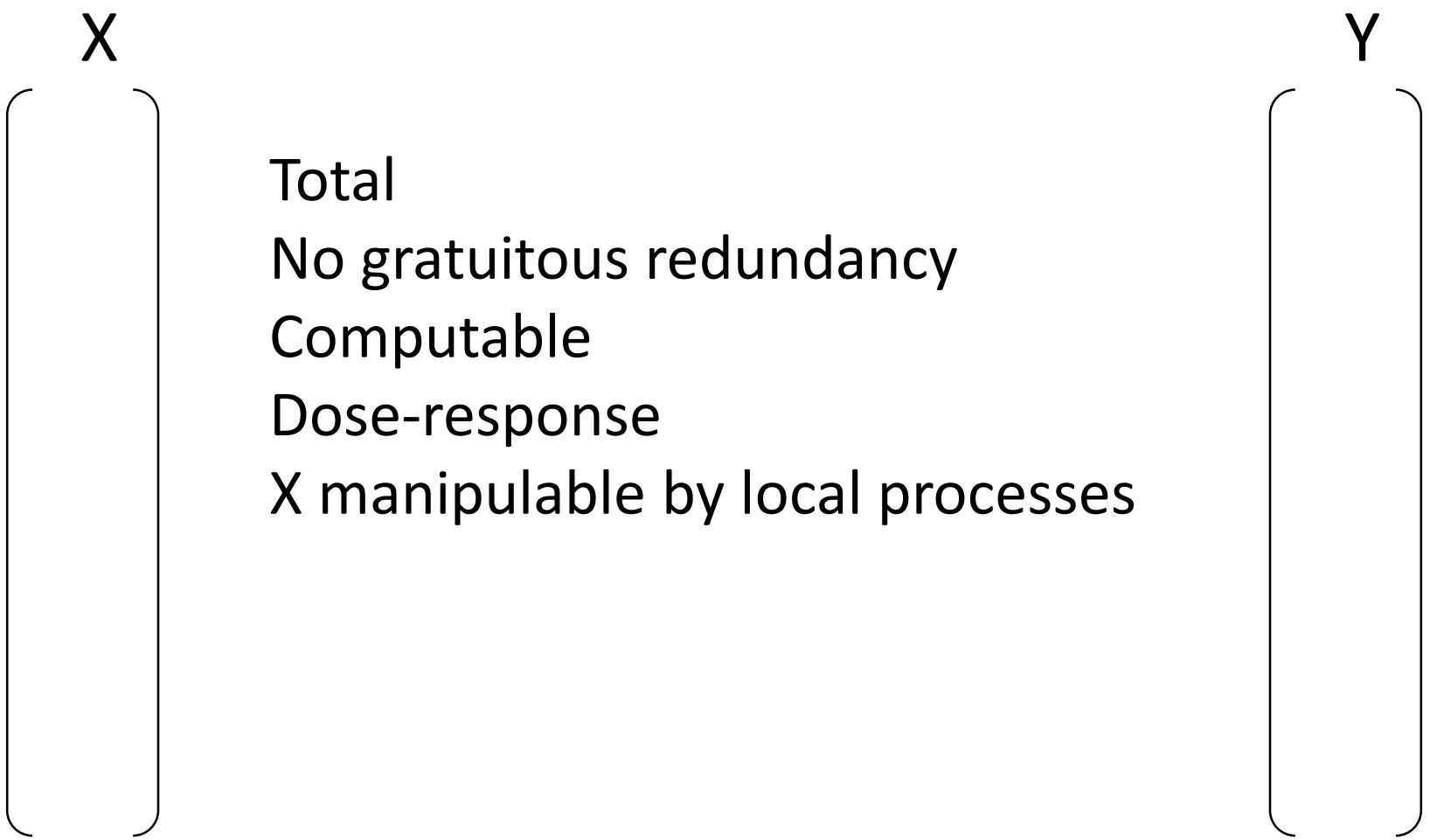
(1) Because the object weighed 14,503.23lb, or

(2) Because the object weighed more than 10,000lb.

What kind of function from cause to effect?



Map from values of X to values of Y under interventions on X



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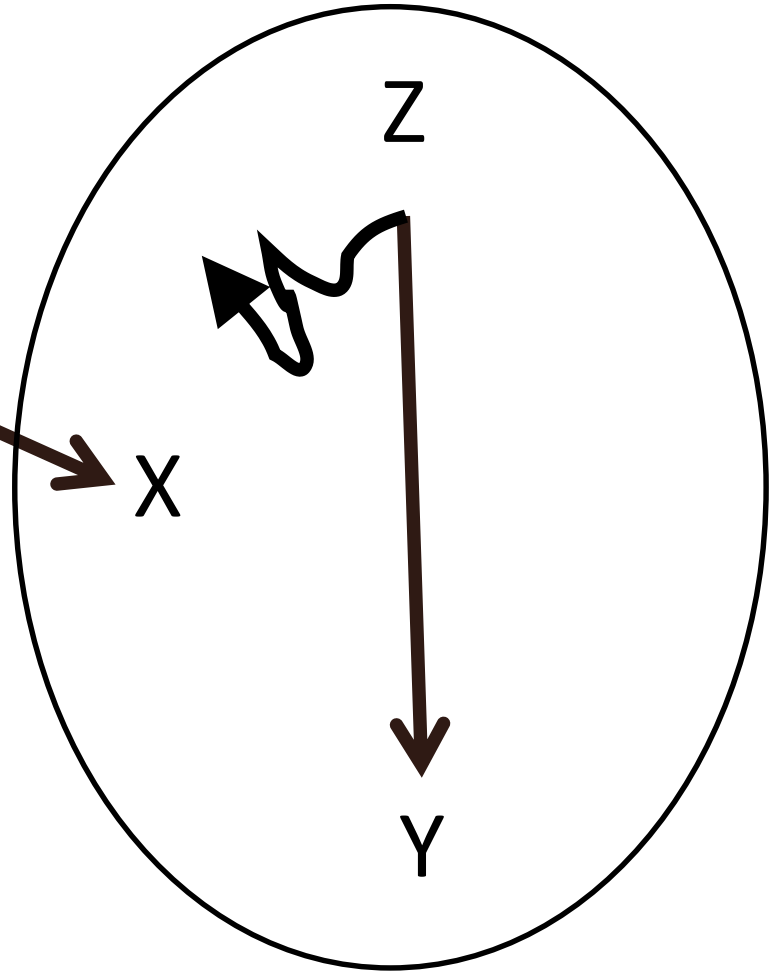
Relativity of causation to a variable set

Intervention



- where X is a control variable for Y

Pressures in two directions:

1. To make the set of variables sufficiently rich. For example, we do not want to have an unrepresented common cause of any pair of variables in our variable set.

2. To keep the set of variables sufficiently lean. For example, one basic demand is that the variables should be independent. Each variable should stand for, in Hume's phrase, an 'independent existent'.

Contrast Schaffner and Tabb on ‘robust patterns’ (Dennett):

> we should take a ‘scientific attitude’ that focuses on ‘predictive utility’ (p. 353)
>
> ‘being a pattern depends on being recognized, an intentional act dependent on the perceptual capacities of the observer’ (p. 354)
>
> ‘robustness’ (Wimsatt) ‘different theoretical approaches confirming the existence of the same phenomenon’ (p. 352)

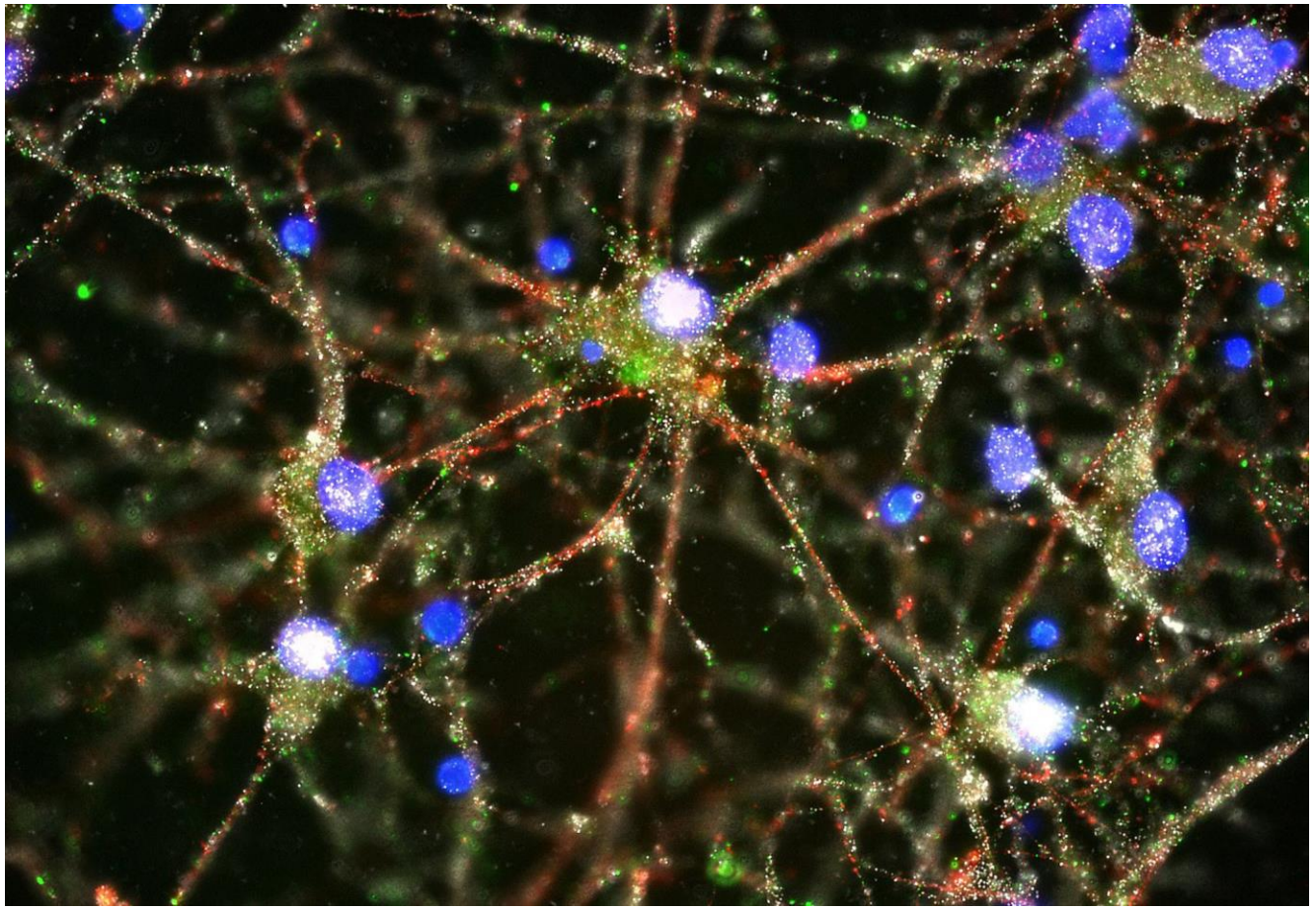
Topics

1. Control Panels for Disorders✓
2. The Vision Science Model: Marr's 3 Levels
3. The Mind-Body Problem

High levels of C4-A generate excessive synaptic pruning.

We don’t just want to toss in overactive C4 genes as one among many causes of schizophrenia

We want to know the cognitive significance of synaptic pruning, we want to interpret it mentalistically



Sekar et.al. 2016

‘Focusing on fearful face–viewing events, patients with anxiety and those with MDD both differed in amygdala responses from healthy participants and from each other during passive viewing. However, both MDD and anxiety groups, relative to healthy participants, exhibited similar signs of amygdala hyperactivation to fearful faces when subjectively experienced fear was rated.’

(Beesdo et. al. 2009)

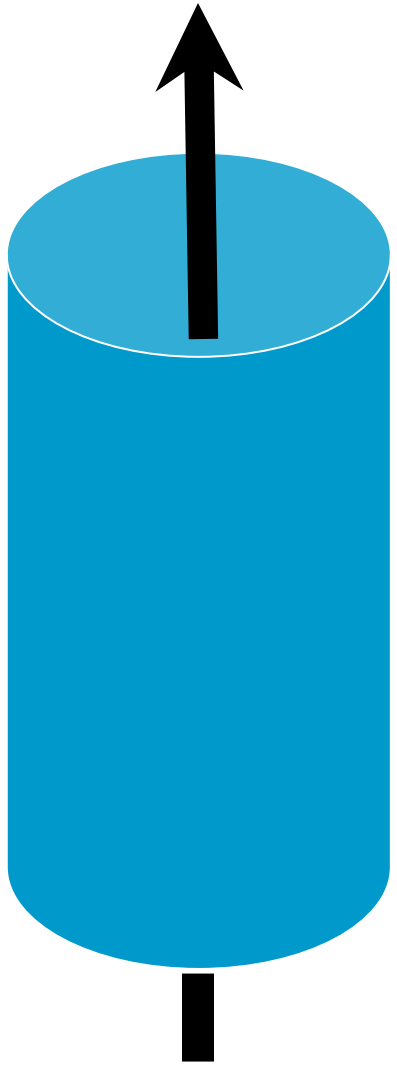
Again, we want to know the mentalistic interpretation of the amygdalar activity

Marr’s 3 levels:

1. Computation
2. Algorithm
3. Implementation

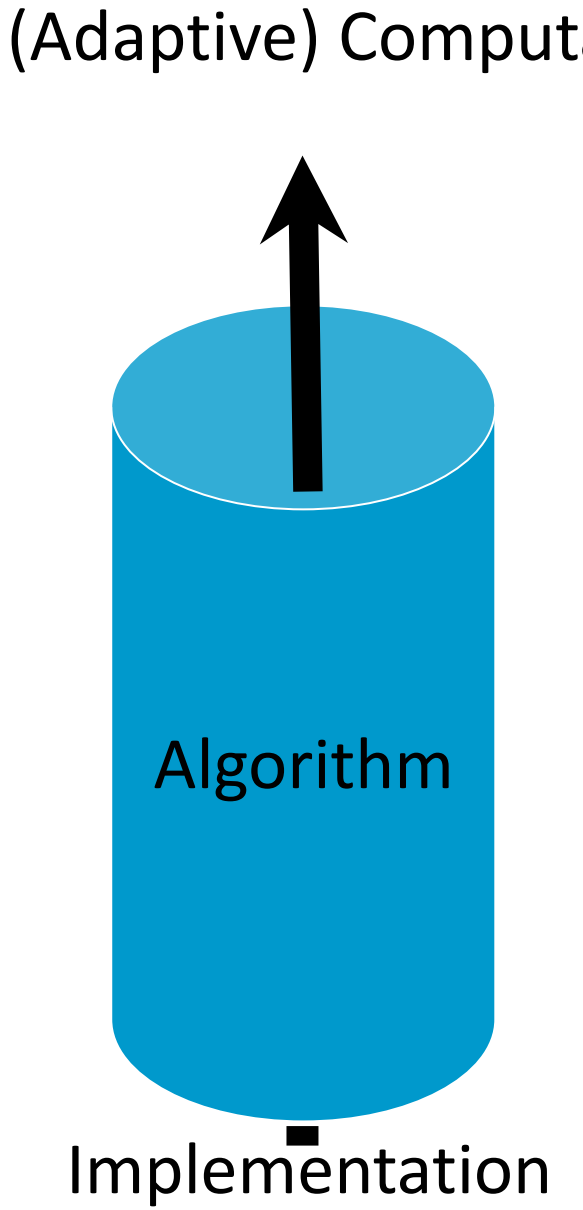
Modularity:

- Domain-specificity
- Encapsulation



Multi-level explanations:

There are different levels of explanation, and the variables at one level are *not* independent of the variables at another level.



Does e.g. humiliation have just one adaptive point?

General-purpose thinking, mood and motivation is not domain-specific (contrast visual systems).

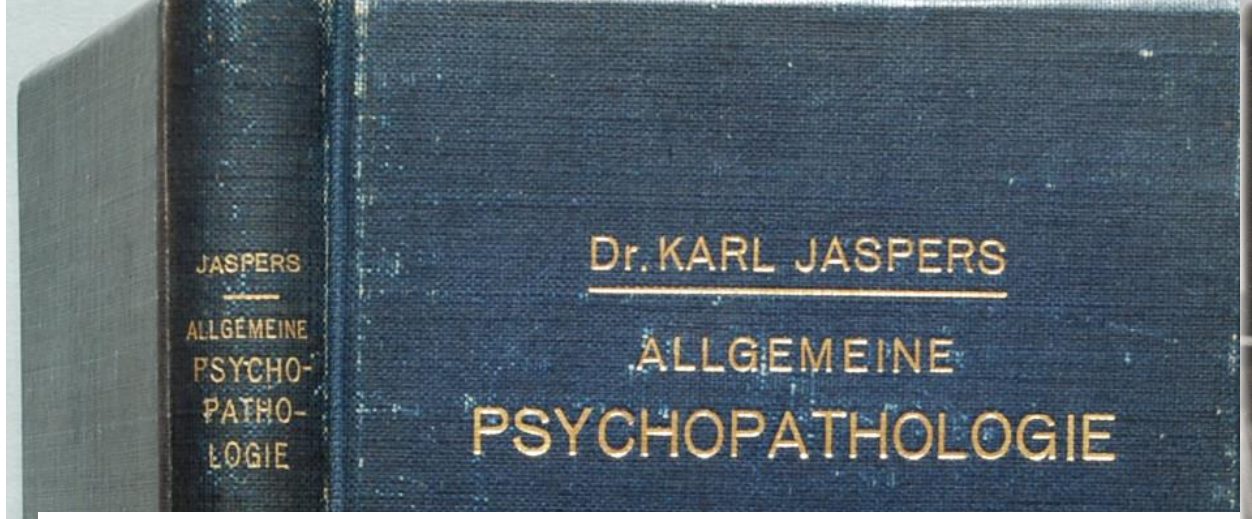
There may be endlessly many functions it serves.

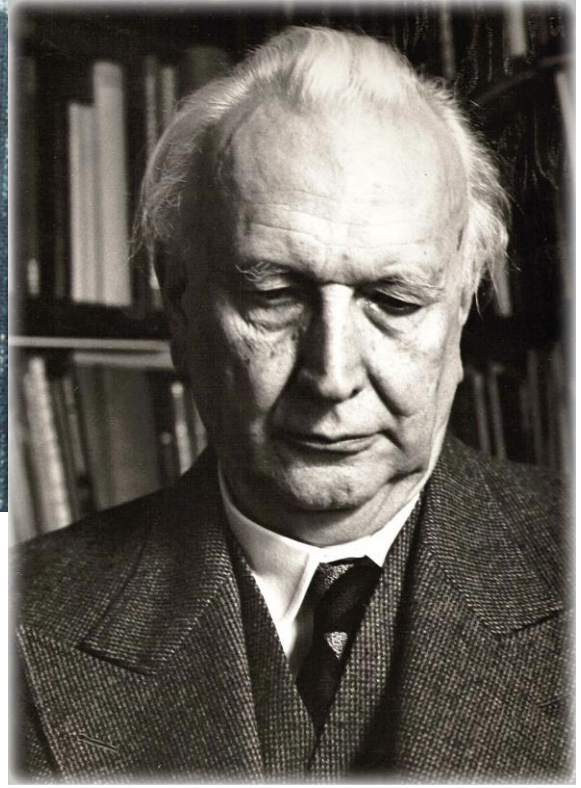
The Vision Science Model

1. ‘Circuits’ in psychiatry: Maybe no adaptive point that can be understood independently of the subjective life (e.g. ‘regulation of mood’)
2. In vision science, the whole representationalist analysis can proceed without any assumption of experience or consciousness on the part of the perceiver. It’s then a problem where, if at all, consciousness fits in. But the science itself doesn’t need to address this question.
3. Not obvious that psychiatry can take this approach. E.g. primary vs. secondary delusions, cognitive neuroscience models of disorders where at a certain point mental causation takes over.

‘At present we are completely unequipped to think about the subjective character of experience without relying on **imagination** - without taking up the point of view of the experiential subject.’

(Nagle)





1. ‘We sink ourselves into the psychic situation and *understand genetically by empathy* how one psychic event emerges from another.

2. We find by repeated experience that a number of phenomena are regularly linked together, and on this basis we *explain causally.’*

Un-understandability

‘Something is going on. Do tell me what on earth is going on?’ …. Patients feel uncanny and that there is something suspicious afoot. Everything gets a new meaning …. Something seems in the air which the patient cannot account for, a distrustful, uncomfortable, uncanny tension …. Patients obviously suffer terribly under it and to reach some definite idea at least is like being being relieved from some enormous burden

(Jaspers 1963)

‘dopamine mediates the conversion of the neural representation of an external stimulus from a neutral and cold bit of information into an attractive or aversive entity. In particular, the mesolimbic dopamine system is seen as a critical component in the ‘attribution of salience’, a process whereby events and thought come to grab attention, drive action, and influence goal-directed behavior because of their association with reward or punishment.’

(Kapur 2003)

Neuroscience driving an imaginative understanding of the patient

But at the psychological level, what kind of system is ‘the mesolimbic dopamine system’? Is it a ‘module’ in the sense of cognitive science? Domain-specific? Encapsulated? Is it *computational* at all?

Similarly for, e.g., the fear-anxiety circuit?

The Vision Science Model

Causal mechanisms are understood in advance of the ascription of representational content; it's what underpins the ascription of representational content

But there is a 'phenomenological' tradition in psychiatry which seems to demand that we find causal significance for the subjective life; the subjective life makes a difference

And famously, we can understand vision comprehensively in terms of representation and causal structure without knowing anything about the subjective experience of the perceiver.

Topics

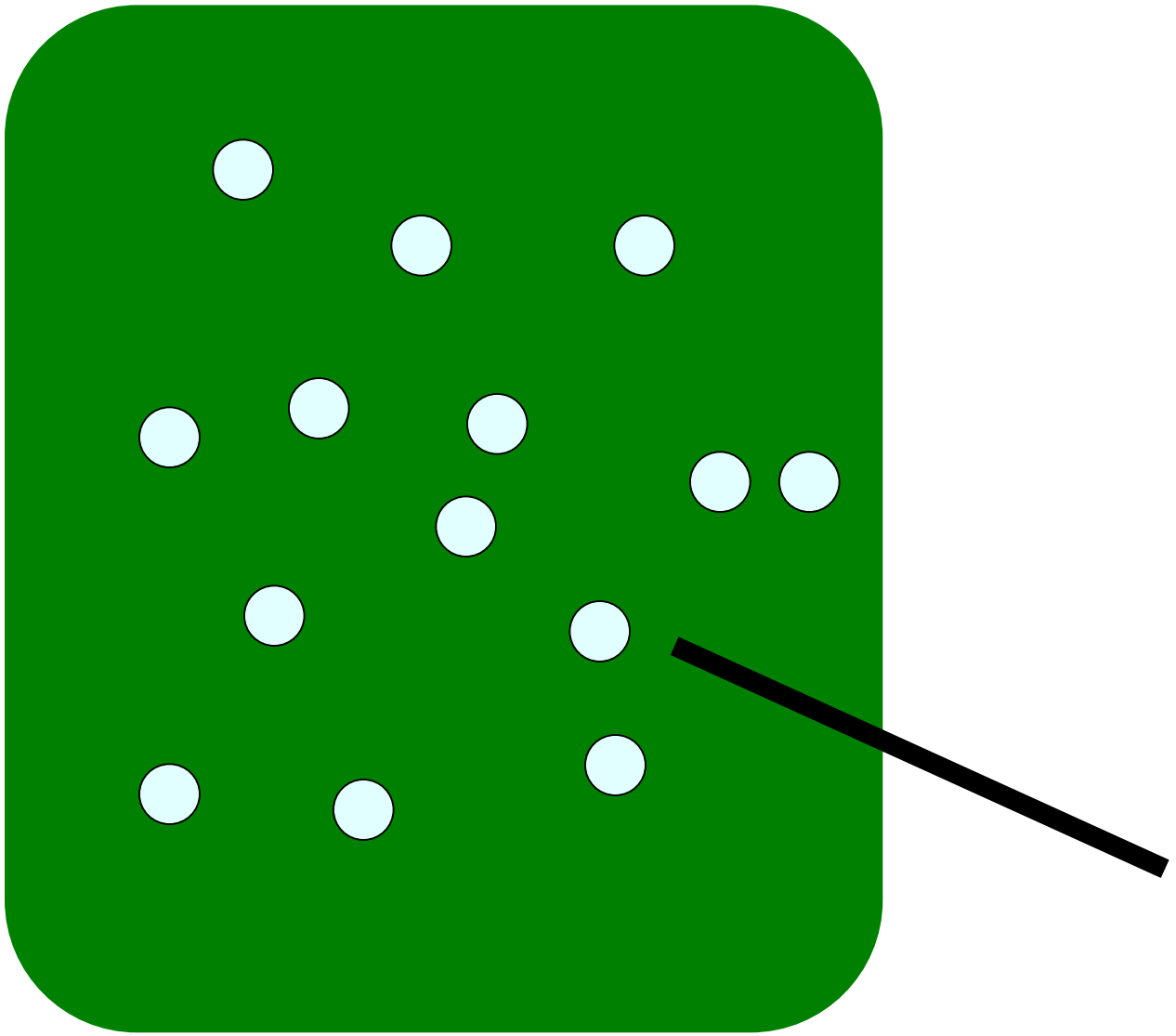
1. Control Panels for Disorders✓
2. The Vision Science Model: Marr's 3 Levels✓
3. The Mind-Body Problem

The billiard table

movements of balls governed by strict deterministic laws; table is relatively frictionless, so balls can roll for months

a fixed initial configuration of balls

an initial cue shot, with a particular force and direction

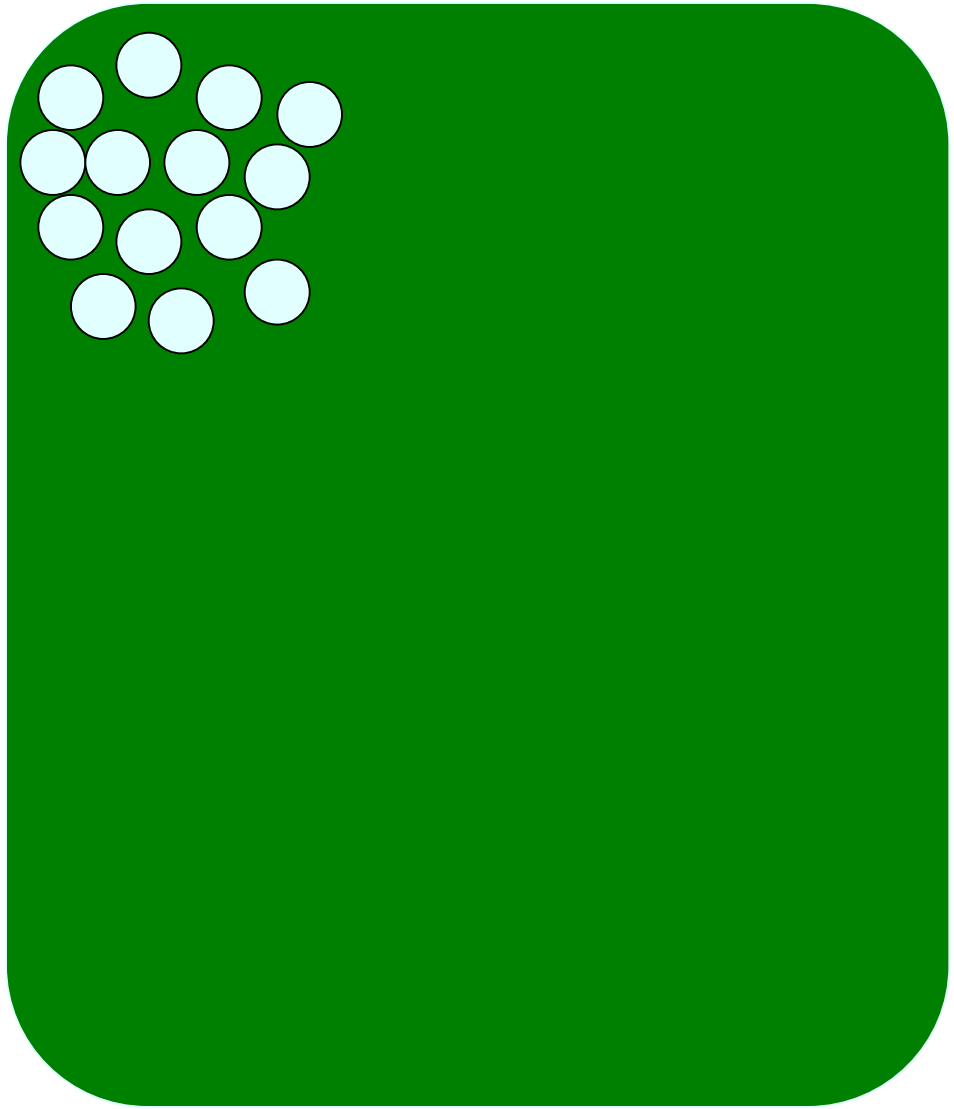


One Monday at 4.15pm all the balls have, momentarily, clustered in the top-left-hand corner

This happens on a series of Mondays at 4.15pm

Why does that happen? Why do the balls cluster at that place at that time, rather than clustering at some other place at some other time?

What is the cause of Clustering (p, t)?



One scenario

all the balls have iron cores, and there is a moveable electromagnet under the table that is switched on from time to time

here there is a control variable for the outcome

suppose there is no electromagnet

no control variable for that outcome space

no systematic connection between force and direction of initial cue shot and time and place of later clustering

Why clustering (p, t)?

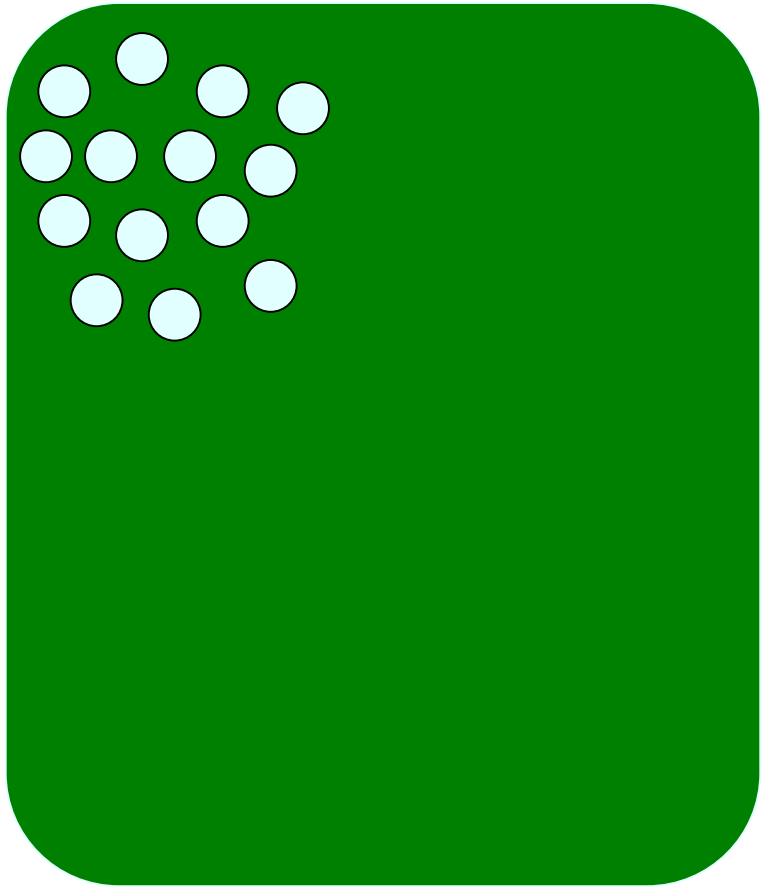
variable is well-defined, but question is ill-posed

consider some systematic way of specifying any of the totality of configurations possible at any one time

Can ask ‘Why (c, t)?’

(System chaotic, so still may not be any way in practice of specifying a control variable)

Suppose it’s us



Suppose we arrange to meet every Monday at 4.15, at some particular place

Suppose Martian physicists observe us

Physical dynamics of our movements no more difficult for Martians than movements of billiard balls for us

They see we cluster sometimes

They say, ‘Why Clustering (p,t)?’ is ill-posed

There is a psychological control variable for our congregating at the place and time we do

- our agreeing to meet at that place and time.
But there may be no physical control variable

Or does the second law of thermodynamics imply that high-level causal structure must always br grounded in physical control variables?

I Antecedent Validators
A. *Familial aggregation and/or co-aggregation (i.e., family, twin or adoption studies)
B. Socio-Demographic and Cultural Factors
C. Environmental Risk Factors
D. Prior Psychiatric History
II Concurrent Validators
A. Cognitive, emotional, temperament, and personality correlates (unrelated to the diagnostic criteria).
B. Biological Markers, e.g., molecular genetics, neural substrates
C. Patterns of Comorbidity
[Note - while categories A and B would most typically be assessed after illness onset, they also could be assessed prior to illness onset as pre-morbid characteristics]
III Predictive Validators
A. *Diagnostic Stability
B. *Course of Illness
C. *Response to Treatment

Topics

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2. Marr’s Three Levels in Psychiatry✓
3. The Mind-Body Problem✓