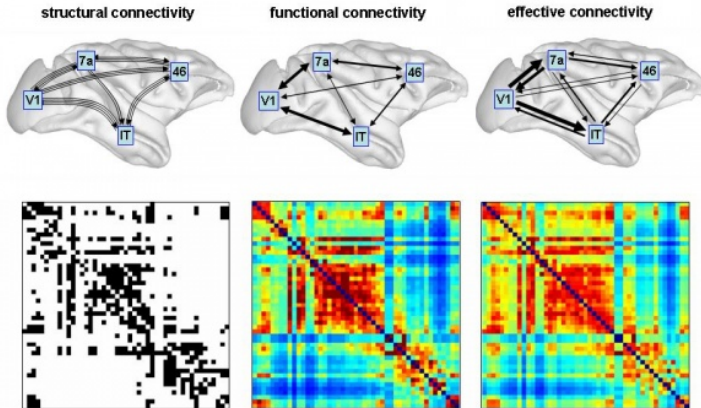


Causal Modeling

NEUR 608

October 19th 2018

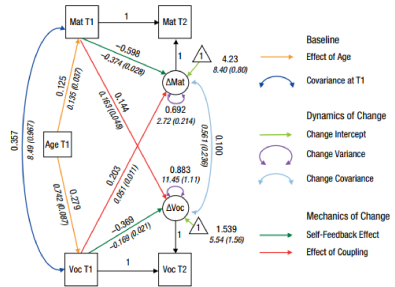
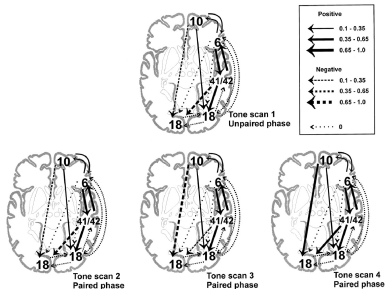
Connectivity



Inferring effective connectivity

- 1 physical influence
 - 2 temporal precedence
- structural equation modeling (SEM)
 - dynamic causal modeling (DCM)
 - “Granger causal” methods

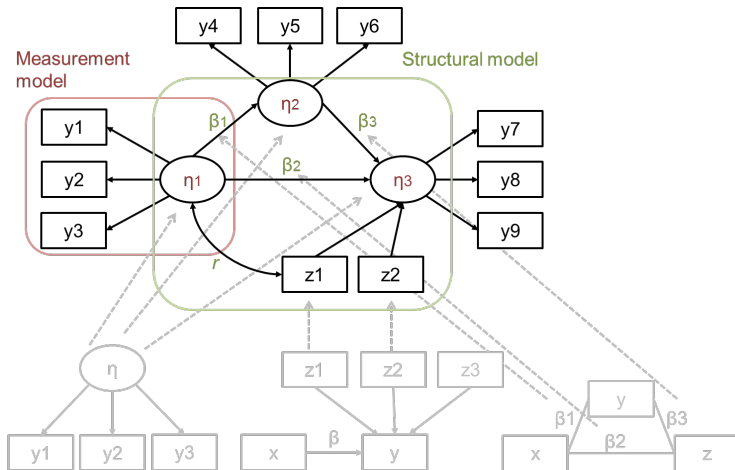
Structural equation modeling



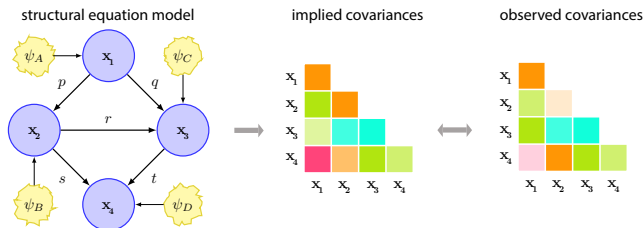
McIntosh et al. (1998) *J Neurophysiol*

Kievit et al. (2017) *Psych Sci*

Structural equation modeling



Structural equation modeling



structural equations:

$$x_1 = \psi_{x_1}$$

$$x_2 = px_1 + \psi_{x_2}$$

$$x_3 = qx_1 + rx_2 + \psi_{x_3}$$

$$x_4 = sx_2 + tx_3 + \psi_{x_4}$$

pairwise correlations:

$$R_{x_1, x_2} = p$$

$$R_{x_1, x_3} = q + pr$$

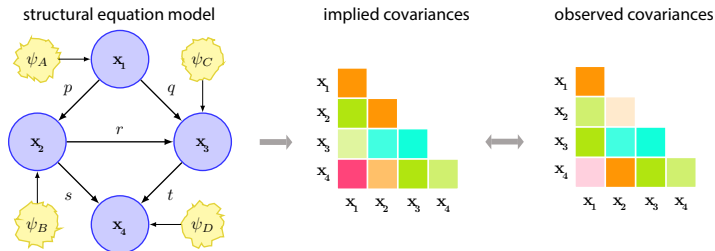
$$R_{x_1, x_4} = ps + prt + qt$$

$$R_{x_2, x_3} = r + pq$$

$$R_{x_2, x_4} = s + rt + pqt$$

$$R_{x_3, x_4} = t + sr + qps$$

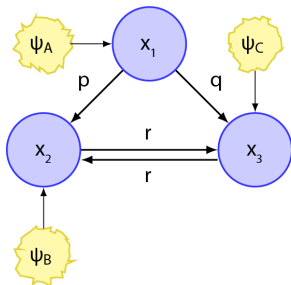
Fitting structural models



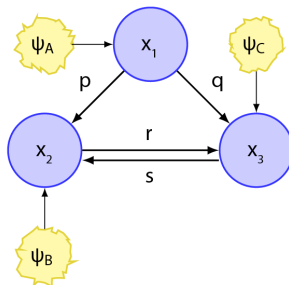
- compare implied and observed covariance matrices
- fewer parameters than variances/covariances
- fit indices: chi-sq, BIC/AIC, CFI, RMS, etc.
- non-significant chi-sq implies not-poor fit

Example 1: compare models

symmetric influence

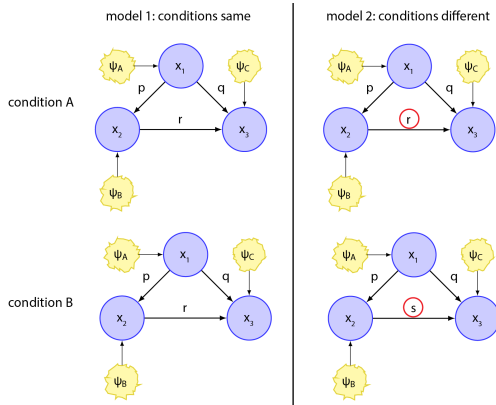


asymmetric influence



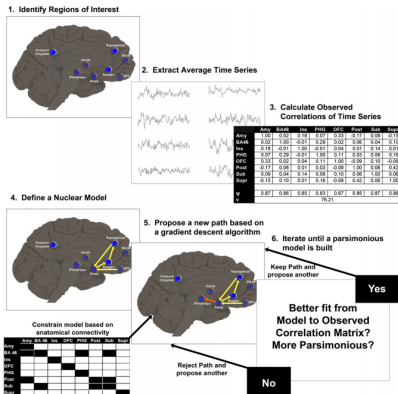
- is influence from x_1 to x_2 symmetric or asymmetric?
- difference test = $\chi^2_{sym} - \chi^2_{asym}$, $df = df(sym) - df(asym)$
- null hypothesis: no significant difference in fit

Example 2: compare groups/conditions



- are parameters different across groups/conditions?
- evaluate using difference test
- bootstrap to get confidence intervals on path coefficients

Example 3: relativistic models



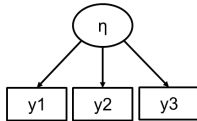
- modification index: improvement in fit if the path coefficient were unconstrained

Bullmore et al. (2000) *NeuroImage*

Stein et al. (2007) *NeuroImage*

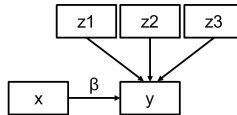
SEM as a general modeling framework

Factor Analysis



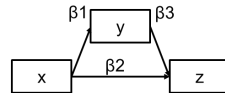
Estimate a construct (η) underlying values on various related indicator variables ($y_1 - y_3$)

Multiple Regression



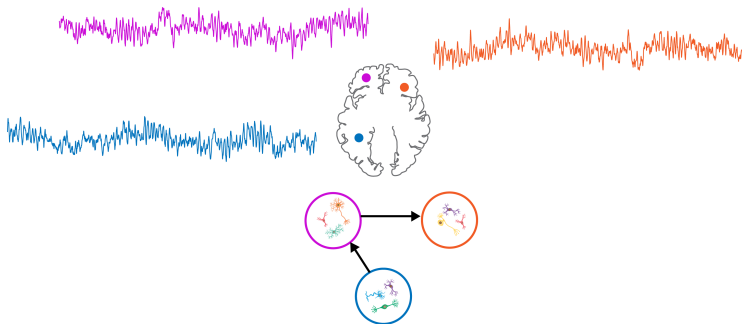
Estimate $x \rightarrow y$ (parameter β) while controlling for confounders z_1, z_2, z_3 that are related to x and to y

Path Analysis



Estimate relations (parameters $\beta_1, \beta_2, \beta_3$) between various constructs (x, y, z) at the same time

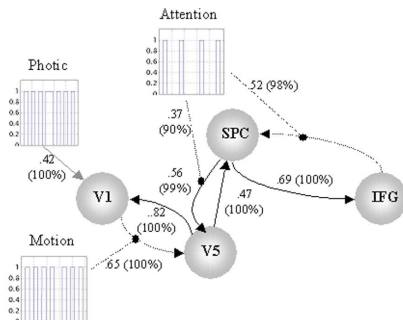
Dynamic Causal Models



- SEM estimates causality at level of observations
- DCM estimates causality at level of neural interactions
- observed: BOLD, EEG, MEG
- unobserved: neural states and interactions

Dynamic Causal Models

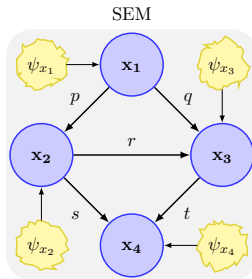
a



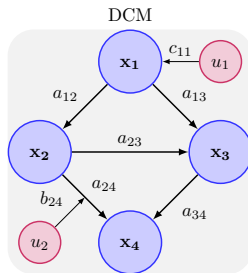
- neuronal model
- forward model
- Bayesian model inversion

Friston et al. (2003) *NeuroImage*

Neuronal model



$$\mathbf{X} = \beta \mathbf{X} + \psi$$

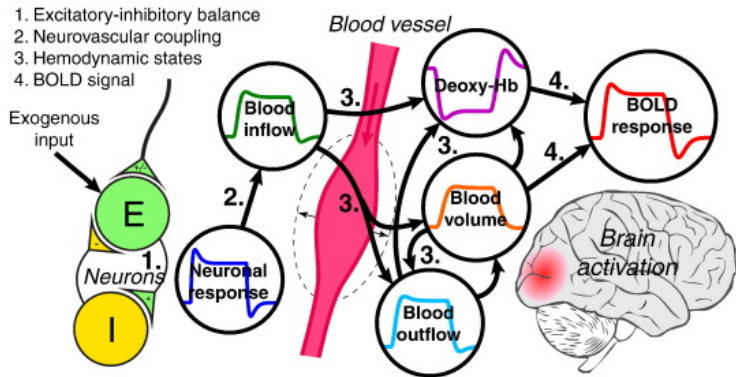


$$\frac{\partial \mathbf{X}}{\partial t} = \mathbf{A} \mathbf{X} + \mathbf{u} \mathbf{B} \mathbf{X} + \mathbf{C} \mathbf{u}$$

$$\text{SEM: } \begin{pmatrix} \dot{x}_1 \\ \dot{x}_2 \\ \dot{x}_3 \\ \dot{x}_4 \end{pmatrix} = \begin{pmatrix} 0 & 0 & 0 & 0 \\ p & 0 & 0 & 0 \\ q & r & 0 & 0 \\ 0 & s & t & 0 \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \\ x_3 \\ x_4 \end{pmatrix} + \begin{pmatrix} \psi_{x_1} \\ \psi_{x_2} \\ \psi_{x_3} \\ \psi_{x_4} \end{pmatrix}$$

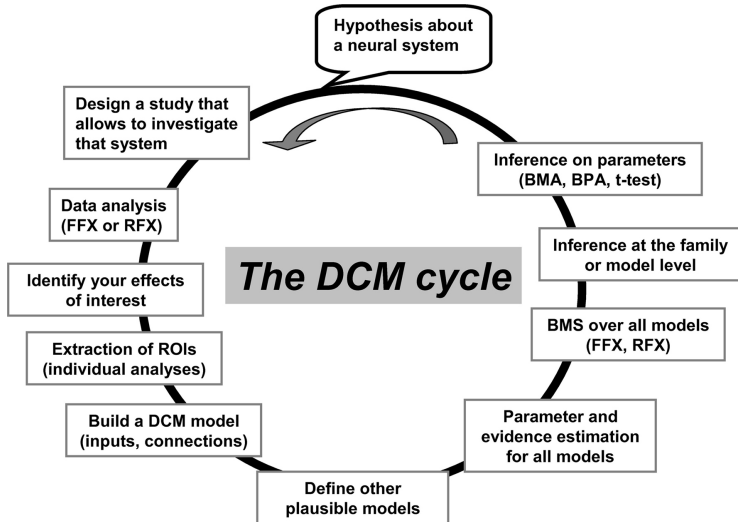
$$\text{DCM: } \begin{pmatrix} \dot{x}_1 \\ \dot{x}_2 \\ \dot{x}_3 \\ \dot{x}_4 \end{pmatrix} = \left(\begin{pmatrix} 0 & a_{12} & a_{13} & 0 \\ 0 & 0 & a_{23} & 0 \\ 0 & 0 & 0 & a_{34} \\ 0 & 0 & 0 & 0 \end{pmatrix} + u_2 \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & b_{24} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \right) \begin{pmatrix} x_1 \\ x_2 \\ x_3 \\ x_4 \end{pmatrix} + \begin{pmatrix} c_{11} & 0 \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{pmatrix} \begin{pmatrix} u_1 \\ u_2 \end{pmatrix}$$

Forward model



Havlicek et al. (2015) *NeuroImage*

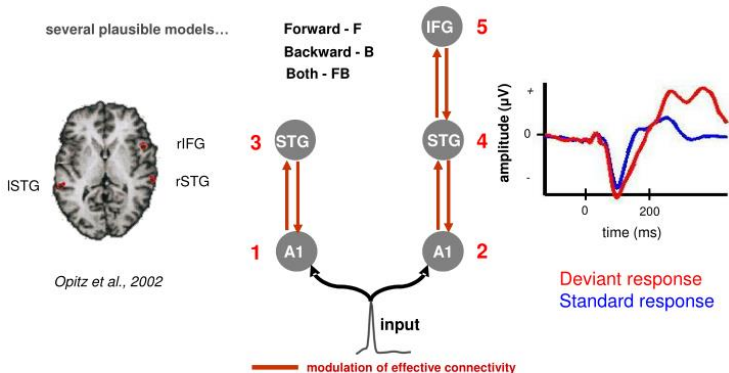
Model inversion



Example: mismatch negativity

DCM specification

What set of areas and interconnections caused the MMN?



Granger causal models

$$\begin{bmatrix} \gamma_1 \\ \gamma_2 \\ \gamma_3 \\ \vdots \\ \gamma_p \end{bmatrix} = \begin{bmatrix} \gamma_0 & \gamma_{-1} & \gamma_{-2} & \dots \\ \gamma_1 & \gamma_0 & \gamma_{-1} & \dots \\ \gamma_2 & \gamma_1 & \gamma_0 & \dots \\ \vdots & \vdots & \vdots & \ddots \\ \gamma_{p-1} & \gamma_{p-2} & \gamma_{p-3} & \dots \end{bmatrix} \begin{bmatrix} \varphi_1 \\ \varphi_2 \\ \varphi_3 \\ \vdots \\ \varphi_p \end{bmatrix}$$

- γ_i “causes” γ_j if past of i predicts future of γ_j
- if effects are linear, problem can be formulated as a multivariate vector autoregressive model (MVAR)
- current value of γ_i is a linear combination of the previous m values of all other γ_j

Granger (1969) *Econometrica*

Goebel et al. (2003) *Magn Reson Imag*

Extensions

- frequency domain; proportion of total power in x_j that can be attributed to x_i (“spectral Granger causality” / “directed transfer function”)
- mutual information; proportion of uncertainty in x_j that is reduced by knowledge of x_i (“transfer entropy”)
- nonparametric inference via surrogate models

Kaminski et al. (2001) *Biol Cybern*

Schreiber (2000) *Phys Rev E*

Strengths and limitations

- confirmatory
- mechanisms
- novel questions
- robust against some violations of assumptions
- hypotheses \approx assumptions
- causal inference vs. model comparison
- size limit (SEM, DCM)
- parameter estimation

Constructive criticism



Chris Holdgraf

@choldgraf

Follow



Has anybody ever read a paper that used Dynamic Causal Modeling and had any idea what the hell the authors were talking about?

2:15 PM - 16 Sep 2017

13 Retweets 56 Likes



9



13



56



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Tal Yarkoni @talyarkoni · Sep 16



Replying to @choldgraf

I think it starts to make sense if you ask yourself what wishful thinking would look like in mathematical form



4



3



34

