Understanding degeneracy & redundancy using variational free energy

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Paper

Motivation

Degeneracy refers to a structure-function mapping in which a system can recruit from multiple structures to achieve functional plasticity. Systematic differentiation of these structures might provide insights into how cognitive functions recover following neurological damage. Since each structure is sufficient, but not necessary, for a particular function; profound deficit is manifest when all degenerate structures are damaged. Conversely, redundancy should be regarded as a distinct but related concept. Here, we provide a **computational account of degeneracy and redundancy**, in terms of variational Bayes.

Degeneracy & redundancy

Figure 1:Redundancy (a) and Degeneracy (b) during cup lifting

Redundancy refers to different structures that perform the same function. Under a functional setting, it implies an inefficient use of a system's degrees of freedom to realise a function e.g., it would be redundant to use both hands to 'lift a cup', when one hand is sufficient (Fig 1a).
Degeneracy refers to when many structures can yield one function e.g., either the left or right hand could be used to 'lift a cup' (Fig 1b). This provides a degenerate structure-function relationship.

If one degenerate structure is damaged, the function can be retained by the remaining structures.

Free energy, redundancy & degeneracy

Our definitions of degeneracy and redundancy rest upon the idea that the functional imperative for any sentient system is to minimise variational free energy [1]. Central to this is the notion of an internal (generative) model that generates predictions of sensory consequences of plausible causes. The essence of degeneracy lies in the many-to-one mapping between causal structures in this generative model and the observable outcomes.

Free energy & degeneracy To make this explicit, we decompose the variational free energy [2]:

$$\mathcal{F} = \underbrace{\mathbb{E}_Q[-\log P(s,o)]}_{Energy} - \underbrace{\mathbb{E}_Q[-Q(s)]}_{Entropy}$$

Here, entropy is the uncertainty of approximate posterior beliefs. We associate: degeneracy = entropy.

Because the essence of degeneracy lies in the 'many to one' mapping between causal structures in the (generative) model and the sensory data. This means that degeneracy is measured by the number of distinct causes that could produce the same outcome. Mathematically, high degeneracy implies that the posterior probability or belief about causes will in the context of a degenerate mapping between causes and consequences have a large entropy. This is precisely the entropy term above.

Free energy & redundancy For this, we again decompose the variational free energy to [1]:

$$\mathcal{F} = \underbrace{D_{KL}[Q(s)||P(s)]}_{Complexity} - \underbrace{\mathbb{E}_Q[P(o|s)]}_{Accuracy}$$

Here, complexity scores the degree to which posterior beliefs have to move away from prior beliefs to explain the data at hand. We associate: redundancy = complexity.

Briefly, complexity is the difference between posterior and prior beliefs. Alternatively, they are the degrees of freedom that are used to provide an accurate account of the data. Therefore, large divergences from prior beliefs to posterior beliefs would incur a greater complexity cost, i.e., have a larger redundancy.

Group Type	Duplicate Specification	Free Energy	Redundancy	Degeneracy
Control	Y	14.827	14.826	2.090
Control	N	13.106	13.055	1.540
Intrinsic (B) Lesions	Y	15.598	15.597	2.973
Intrinsic (B) Lesions	N	13.878	13.822	2.428
Extrinsic (A) Lesions	Y	29.955	26.186	2.393
Extrinsic (A) Lesions	N	29.786	26.042	2.132
Intrinsic (A) & Extrinsic (B) Lesions	Y	36.309	32.005	5.950
Intrinsic (A) & Extrinsic (B) Lesions	N	35.580	31.232	5.566

Figure 2:Average redundancy and degeneracy for each simulation type in natural units (across 50 subjects).

Simulations

To test this, we looked at how structural learning and in-silico lesions influence degeneracy and redundancy using a word repetition paradigm. This is a prescient task since it highlights the usefulness of our approach for quantifying which combinations of structural damage (as measured by degeneracy) are necessary to disrupt functional outcomes; i.e. ability to repeat words. We specified a (generic) generative model and belief updating scheme described in [1], for repeating words. See [2] for exact specifications of the generative model here but note the inclusion of a duplicate level of hidden states within the model. This structural duplication, is important for evaluating redundancy minimisation, using a formulation of structural learning.
Using this, we report two types of simulations:

- Omitting sensory information and disconnecting duplicate model parameters. We expect this to minimise redundancy .
- Progressively increasing lesioned connections via rendering them statistically imprecise. We hypothesised that increased lesion load would have a non-linear effect on degeneracy.

Results

The model with multiple lesions had highest degeneracy due to extremely uncertain posterior expectations about causes in the context of degenerate mappings. Conversely, with single lesions degeneracy measurement was reduced by 50%. This is in-line with our definition: degenerate mappings between causes and consequences, would lead to large entropy.
Behavioural responses (accuracy) for the simulated subjects [2] illustrated that the cumulative effect of successive lesions was super-additive, such that the model was unable to repeat the word correctly.

Conclusion

We demonstrated that degeneracy and redundancy can be associated, and measured quantitatively, using appropriate decompositions of free energy. This computational approach provides a framework to evaluate degeneracy, and potential recovery pathways, in damaged brains, and make predictions about recovery outcomes.

[1] Friston, et al. (2017). Active inference: a process theory, Neural Computation [2] Sajid et al, (2020). Degeneracy and Redundancy in Active Inference, Cerebral Cortex.