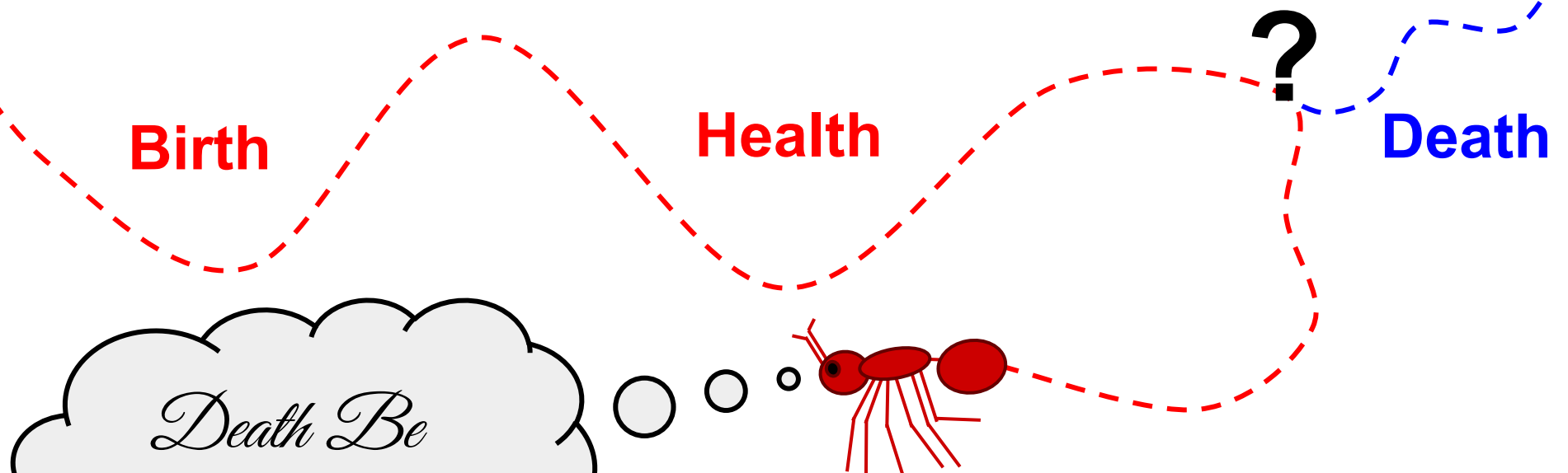


Of Ants & Aging



Birth

Health

?

Death

*Death Be
Not Proud!*

April 22, 2023
Daniel A. Friedman



Epistemic status & context

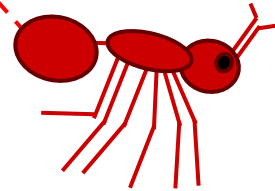
- I am aiming to present some rapid connected reflections on Ants & Aging, which when interpreted from the expert perspectives at this conference, might be useful – though I am not an expert or currently working in aging research.
- In terms of genre, what is presented here is meant more as whimsical and promptive, rather than exhaustive.

Topics Today (Questions that were listed on the conference website)

- Is aging the result of a passive accumulation of damage or an active self-destruction program? That is, do we age for the same reason cars wear out, or for the same reason we go through puberty?
 - **WHY does aging happen in Ants & Humans?**
- Do all mammals age by the same mechanisms?
 - **What are similarities & differences in “why aging happens” across even broader phylogenetic scope?**
- Does the biological clock that governs the timing of wisdom teeth budding affect the rate of aging?
 - **I don't know.**
- What progress has been made in reversing aging?
 - **I don't know about progress in reversing aging in humans.**
 - **Today we can ask about what type or extent of “progress has been made in reversing aging” in ants.**

Roadmap

- **Motivations & Definitions**
 - What are Ants & Aging, and why study them?
- **Why does biological Aging occur?**
 - Using Tinbergen's 4 Questions to ask Why aging happens
- **Discussions**
 - Maps, Territories, and Decisions.
- **Questions & Answer**



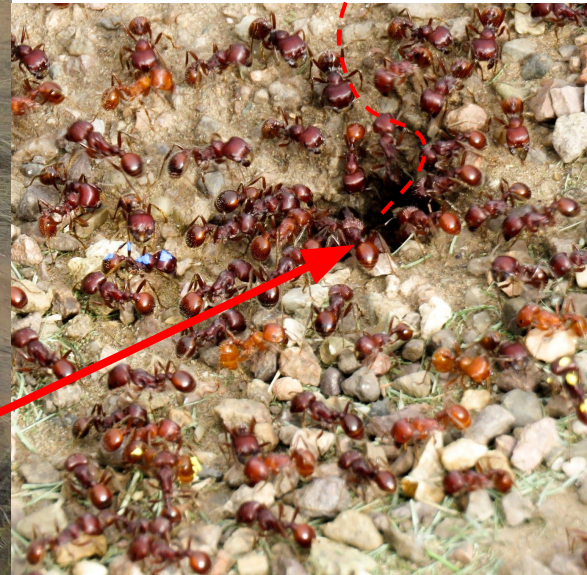
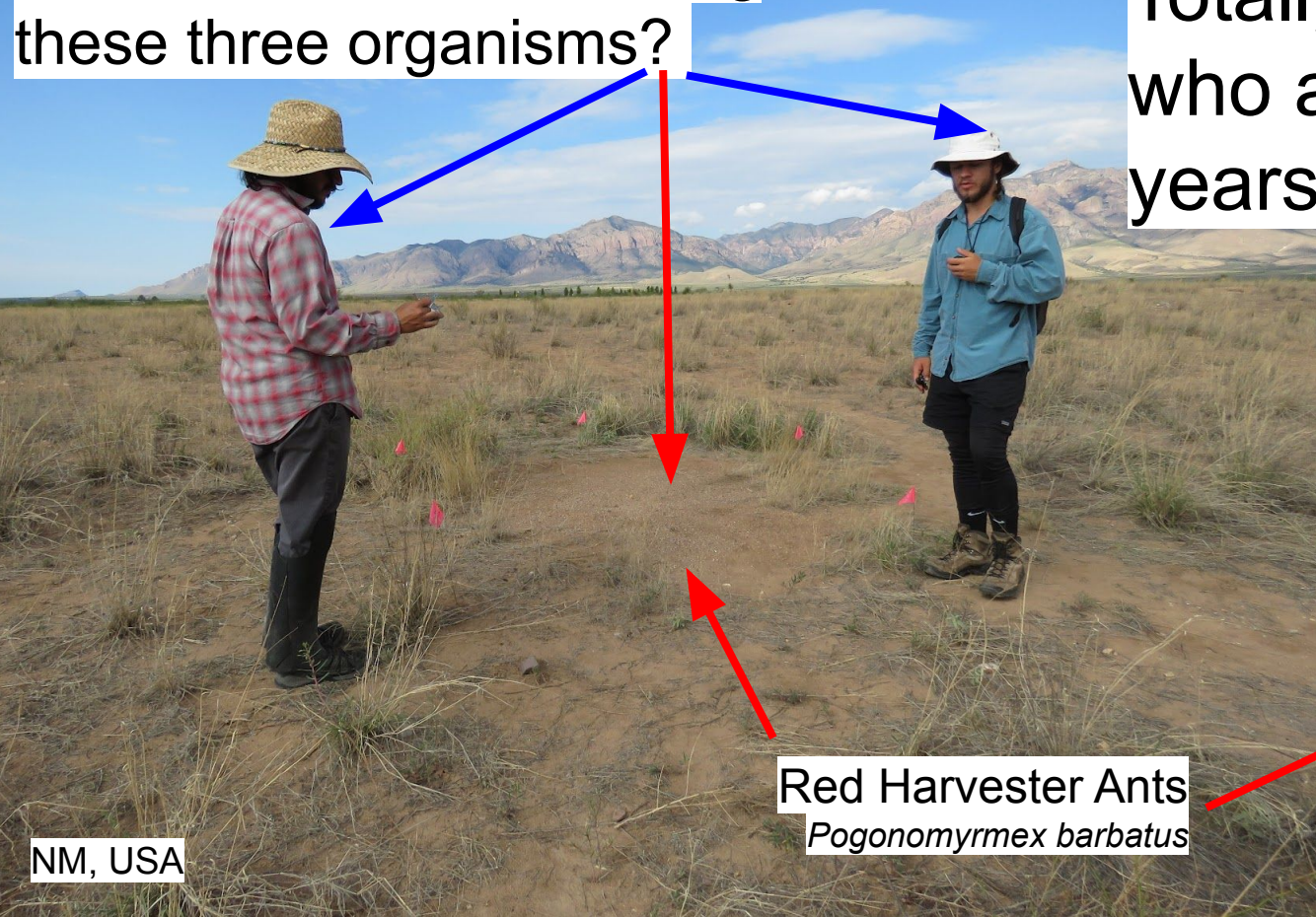
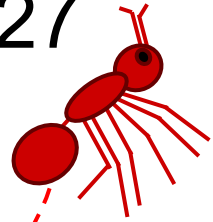
Motivations & Definitions

Question:

What is in common among these three organisms?

Answer:

Totally fit 6-foot guys who are about 27 years old (!!!)



Red Harvester Ants
Pogonomyrmex barbatus

NM, USA

30+ year field study of Red Harvester Ants by Professor Deborah Gordon at Stanford

Red and Blue are inferred parentage relationships at the field site

Colonies of *P. barbatus* have a single long-lived queen
Queen stores sperm from single mating & lay eggs life-long
Sterile nestmates (workers) turn over annually or faster

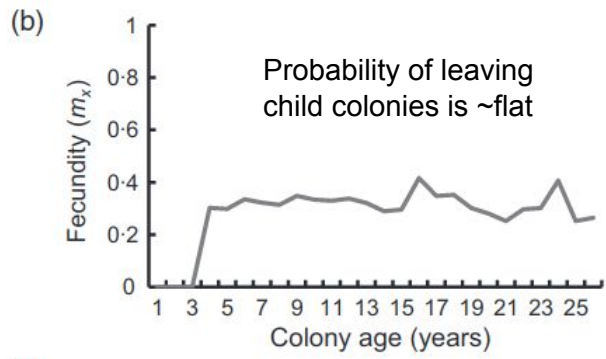
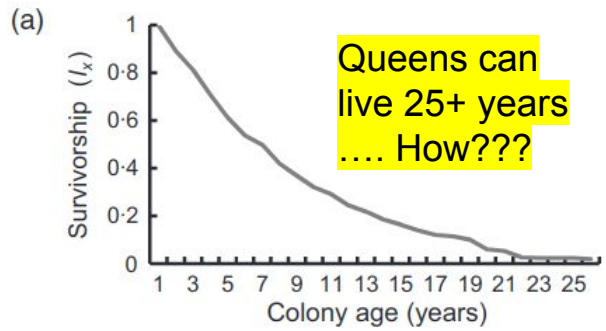
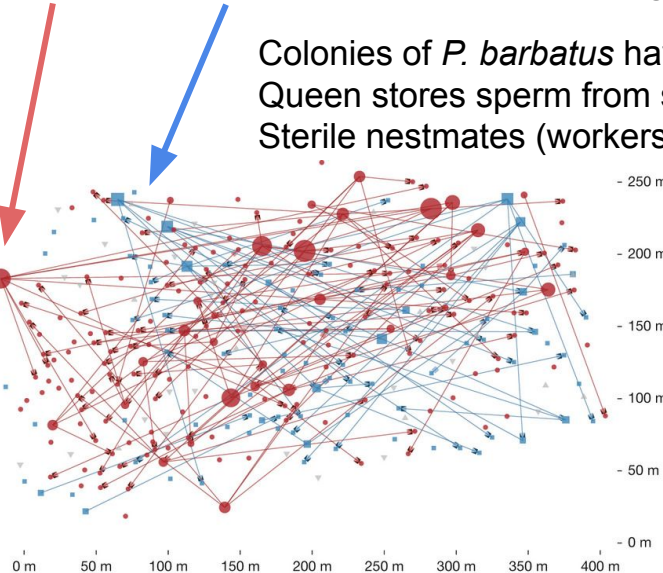
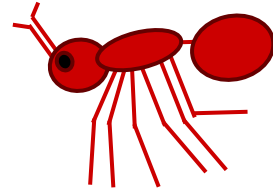


Fig. 4. (a) Survivorship as a function of colony age. (b) Fecundity as a function of colony age.

Definitions

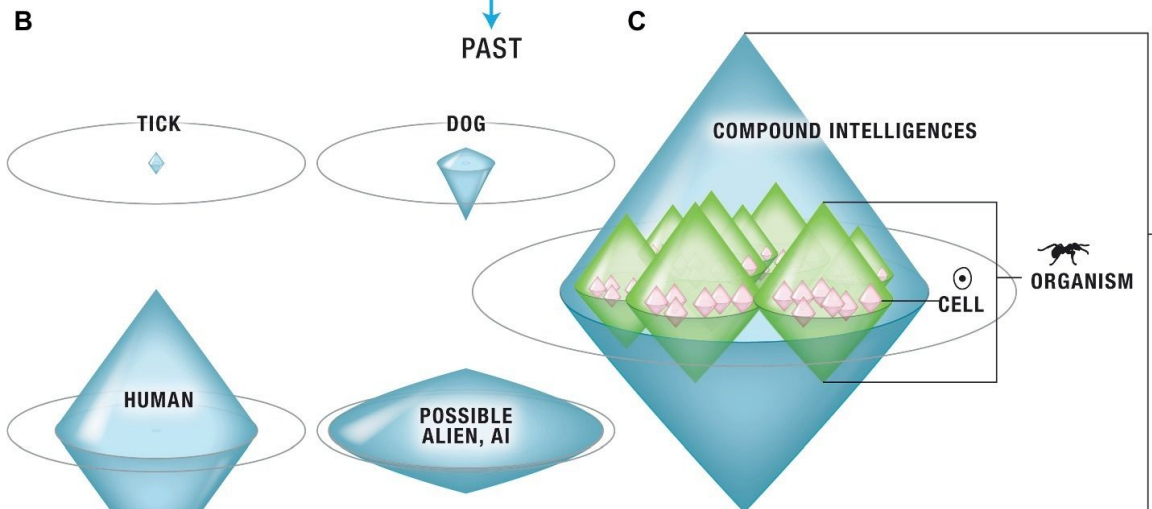
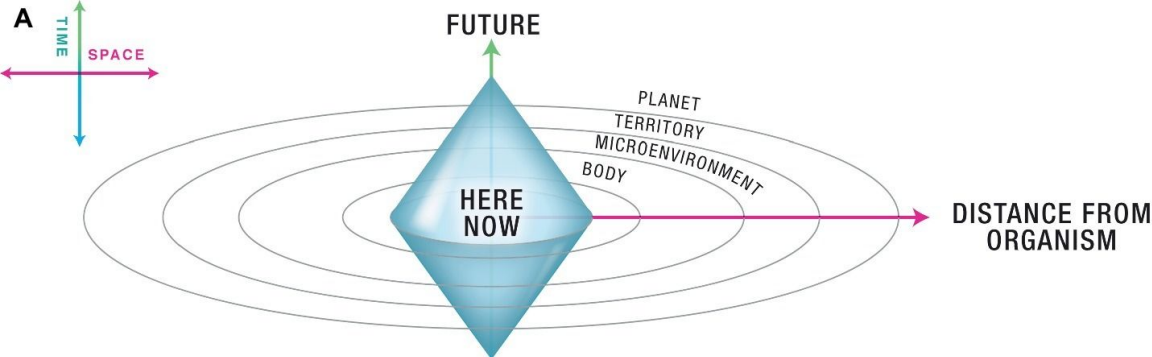
Ants

- Diverse and global group of with ~15,000 known species
- **Nestmate**
 - 6-legged insect body (might be sterile as in “worker” or reproductive as in “queen” or “male”).
- **Colony**
 - Aggregate of nestmates; how ants live in all cases (obligate eusociality)



Age ≠ Aging

- **Age**
 - An amount of time (*chronos*): the clock time elapsed during the lifespan of an individual
- **Aging**
 - A process and embodied outcome occurring through time (*kairos*), associated with a variety of specific phenotypes in a species-specific fashion, such that “more aged” is associated with increased risk of various specific diseases and/or thermodynamic dissolution (death). This definition of “aging” is clearly distinct from age, and in no ways is prescriptive or normative.
 - [Is ageing a disease?](#), The Lancet Healthy Longevity, July 2022
 - But what, on the face of it, looks like an academic dispute around linguistic precision masks a much greater inherent ideological conflict between longevity science and geriatric medicine, which are uneasy bedfellows in the emerging discipline of so-called longevity medicine. The conflict is centred around one crucial question: can ageing be regarded as a disease?
 - [Hayflick 2007](#) “Biological aging is no longer an unsolved problem”



What is Ant?
What is Human?

What is Aging?

HYPOTHESIS AND THEORY article
Front. Psychol., 13 December 2019
Sec. Theoretical and Philosophical Psychology
Volume 10 - 2019 |
<https://doi.org/10.3389/fpsyg.2019.02688>

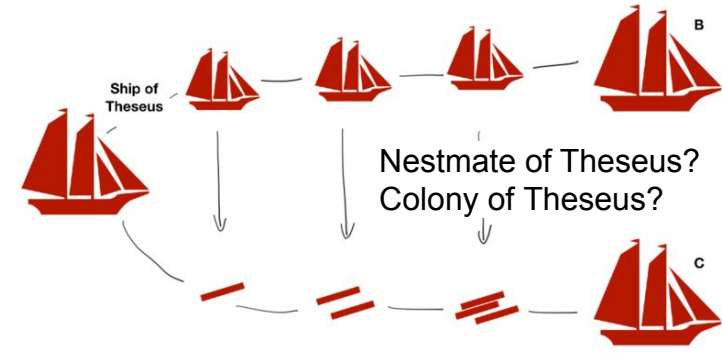
This article is part of the Research Topic
Epistemic Feelings: Phenomenology, Implementation, and
Role in Cognition
[View all 6 Articles >](#)

The Computational Boundary of a "Self": Developmental Bioelectricity Drives Multicellularity and Scale-Free Cognition

Michael Levin^{1,2*}

Levin 2019

FIGURE 2. Arbitrary cognitive "Individuals" can be classified according to their computational boundary.
(A) Each living system has a delimited "area of concern" – a region of space-time, with the organism at its center, within which its cognitive apparatus functions to take measurements and act.
(B) The size and shape of this cognitive boundary defines the sophistication of the agent and determines the scale of its goal directedness.....
(C) In this scheme, Individuals can overlap – the same biophysical system can support a number of coexisting, coupled Selves with different cognitive borders. A coordinated swarm of animals, the individual animals themselves, their organs, their cells, and even the metabolic and transcriptional networks inside the cells each have their own cognitive horizon.



**Why does biological
Aging occur?**



Provide the top 80 hypotheses for why biological aging occurs, just as a simple list.



GPT-4

1. Genetic mutations
2. Telomere shortening
3. Free radicals/oxidative stress
4. Mitochondrial dysfunction
5. Cellular senescence
6. Inflammation
7. Autophagy decline
8. Hormonal changes
9. Epigenetic alterations
10. DNA damage
11. Protein aggregation
12. Glycation
13. Caloric intake
14. Insulin/IGF-1 signaling
15. Sirtuins
16. Stem cell exhaustion
17. Microbiome changes
18. Extracellular matrix stiffening
19. Cellular nutrient sensing
20. Immune system decline
21. Altered intercellular communication
22. Cellular garbage accumulation
23. Loss of proteostasis
24. Deregulation of circadian rhythms
25. Nuclear pore dysfunction
26. Senescence-associated secretory phenotype (SASP)
27. Cellular metabolism dysregulation
28. Deregulation of RNA processing
29. Defective DNA repair pathways
30. Neuronal loss and dysfunction
31. Glial cell dysfunction
32. Lipofuscin accumulation
33. Defective protein folding
34. Chromosomal instability
35. Immunosenescence
36. Thymic involution
37. Somatic mutation accumulation
38. Impaired stem cell niche
39. Endocrine dysregulation
40. Replicative stress
41. Impaired cellular communication
42. Extracellular vesicle signaling changes
43. Unfolded protein response decline
44. Lysosomal dysfunction
45. Peroxisomal dysfunction
46. Mitophagy decline
47. Ribosome biogenesis decline
48. Cellular polarity loss
49. Defective calcium signaling
50. Changes in ion channel activity
51. Altered gene expression patterns
52. Histone modification changes
53. Nuclear envelope alterations
54. Cellular redox imbalance
55. Impaired cellular respiration
56. Alterations in energy metabolism
57. Dysregulation of apoptosis
58. Cellular stress resistance decline
59. Altered signaling pathways
60. Changes in cell adhesion
61. Genomic instability
62. Transposable element activation
63. Impaired hormone secretion
64. Oxidative stress in the endoplasmic reticulum
65. Altered lipid metabolism
66. Impaired angiogenesis
67. Deregulation of autocrine and paracrine signaling
68. Telomere uncapping
69. Oxidative stress in the Golgi apparatus
70. Altered post-translational modifications
71. Deregulation of cell cycle checkpoints
72. Altered proteasome function
73. Endoplasmic reticulum stress
74. Glycosylation changes
75. Dysregulation of microRNA
76. Altered DNA replication dynamics
77. Impaired endocytosis and exocytosis
78. Changes in cellular architecture
79. Altered membrane fluidity
80. Impaired cellular localization and trafficking

Why does Aging happen in humans and ants?

Aristotle's Four Causes

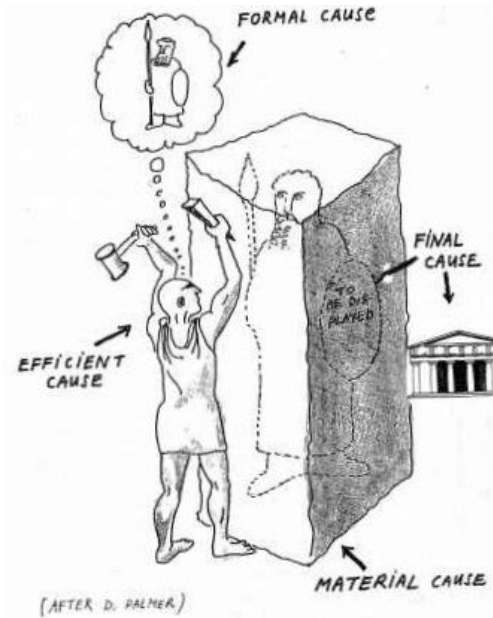


Fig. 1 Tinbergen's four question for the study of behavior. a The traditional view of Tinbergen's four questions has partitioned research into proximate or ultimate levels of analysis and historical and contemporary timescales of influence.

	Proximate	Ultimate
Contemporary	Causation	Current Utility
Historical	Ontogeny	Evolution

Review | [Published: 21 November 2018](#)

Synthesis of Tinbergen's four questions and the future of sociogenomics

Kapheim 2018

[Karen M. Kapheim](#) ✉

[Behavioral Ecology and Sociobiology](#) 73, Article number: 186 (2019) | [Cite this article](#)

2880 Accesses | 13 Citations | 16 Altmetric | [Metrics](#)

Example: Why is that house decayed?

Next we will use Tinbergen's 4 Questions to explore Aging.



	Proximate	Ultimate
Contemporary	Causation	Current Utility
Historical	Ontogeny	Evolution

How the material is now

The house is in a decayed state **because** it has broken windows & walls

How it functions now

The house is decayed **because** leaks water from the roof and wind blows through the windows

How it came to be

It used to be in good condition, then decayed **because** over time the windows & walls broke.

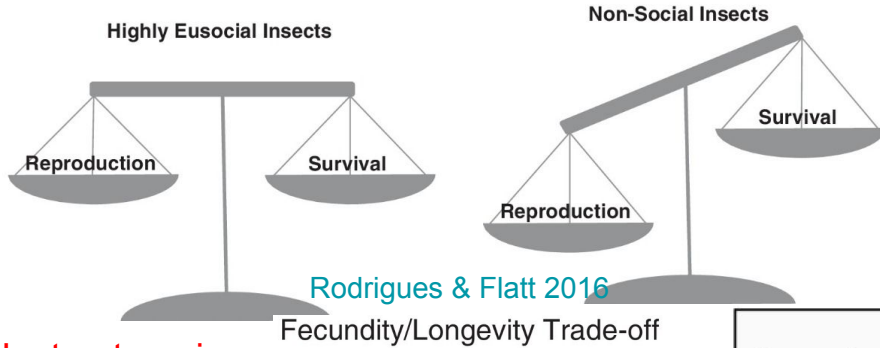
How the instance came to be through deep time

Because generations of similar houses have been made before, out of materials that are planned to last decades not centuries

Ants

Why does aging Develop?

Human



Where do mammals, and humans, fit on these scales?

Aging is a lifelong process.

Trajectories are complex:

- [Nguyen et al. 2021](#) “In this study we investigated the association between multimorbidity and different patterns of healthy ageing trajectories among 130880 individuals in a global sample.

Multimorbidity appeared to increase the likelihood of having poorer healthy ageing trajectories, but the extent to which healthy ageing trajectories were projected to decline depended on the specific patterns of multimorbidity.”

Various influence can shift the balance

- “Infection and inflammation in somatic maintenance, growth and longevity” [Kopp & Medzhitov 2009](#) “infectious pathogens in a given niche.... shifts this balance in favor of somatic maintenance at the expense of reproduction and growth.”

Nestmate aging

- Temporal polyethism
- [Giraldo & Traniello 2014](#) – “*Worker senescence and the sociobiology of aging in ants*” – “We present seven hypotheses concerning how selection could favor extended worker lifespan....”

Colony aging

- Some behavioral effects associated with colony age beyond worker age ([Barton et al. 2002](#))
- **Some Ant species have colonies that don't age!** Immortal colonies may have asexual reproduction, or intra-nest sexual reproduction

	Proximate	Ultimate
Contemporary		
Historical	Ontogeny	

Ants

What is the embodied mechanism of aging?

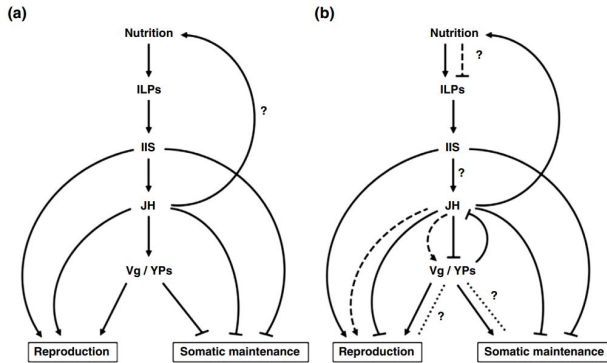
Human

- Rodrigues & Flatt 2016 —“Remarkably, however, **queens of highly eusocial social insects exhibit both enormous reproductive output and longevity, thus escaping the trade-off.** Here we argue — based on recent evidence — that the **proximate** reason for why eusocial insects can decouple this trade-off is that they have evolved a different ‘wiring’ of the IIS-JH-Vg/YP circuit.”
- insulin-like/IGF-1 signaling (IIS).

This is a large area, here we will just focus on Insulin signaling as an example

Provide the top 80 hypotheses for why biological aging occurs, just as a simple list.

1. Genetic mutations	21. Altered intercellular communication	42. Extracellular vesicle signaling changes	63. Impaired hormone secretion
2. Telomere shortening	22. Cellular garbage accumulation	43. Unfolded protein response decline	64. Oxidative stress in the endoplasmic reticulum
3. Free radicals/oxidative stress	23. Loss of proteolysis	44. Lysosomal dysfunction	65. Altered lipid metabolism
4. Mitochondrial dysfunction	24. Derangement of circadian rhythm	45. Peroxisomal dysfunction	66. Impaired angiogenesis
5. Cellular senescence	25. Nuclear pore dysfunction	46. Mitophagy decline	67. Derangement of autocrine and paracrine signaling
6. Inflammation	26. Senescence-associated secretory phenotype (SASP)	47. Boneone biosynthesis decline	68. Telomere uncapping
7. Autophagy decline	27. Cellular metabolism dysregulation	48. Cellular polarity loss	69. Oxidative stress in the Golgi apparatus
8. Hormonal changes	28. Derangement of RNA processing	49. Defective calcium signaling	70. Altered post-translational modifications
9. Epigenetic alterations	29. Defective DNA repair pathways	50. Changes in ion channel activity	71. Derangement of cell cycle checkpoints
10. DNA damage	30. Neuronal loss and dysfunction	51. Altered gene expression patterns	72. Altered proteasome function
11. Protein aggregation	31. Glial cell dysfunction	52. Histone modification changes	73. Endoplasmic reticulum stress
12. Glycation	32. Lipofuscin accumulation	53. Nuclear envelope alterations	74. Glycosylation changes
13. Caloric intake	33. Defective protein folding	54. Cellular redox imbalance	75. Dysregulation of microRNA
14. Insulin/IGF-1 signaling	34. Chromosomal instability	55. Impaired cellular respiration	76. Altered DNA replication dynamics
15. Sirtuins	35. Immunosenescence	56. Alterations in energy metabolism	77. Impaired endocytosis and exocytosis
16. Stem cell exhaustion	36. Thymic involution	57. Dysregulation of autophagy	78. Changes in cellular architecture
17. Microbiome changes	37. Somatic mutation accumulation	58. Cellular stress resistance decline	79. Altered membrane fluidity
18. Extracellular matrix stiffening	38. Impaired stem cell niche	59. Altered signaling pathways	80. Impaired cellular localization and trafficking
19. Cellular nutrient sensing	39. Substrate deprivation	60. Changes in cell adhesion	
20. Immune system decline	40. Replicative stress	61. Genomic instability	
	41. Impaired cellular communication	62. Transposable element activation	



	Proximate	Ultimate
Contemporary	Causation	
Historical		

- “Insulin and aging” ([Kurauti et al. 2021](#)) Most of the age-related diseases have been associated with impairment of action of an important hormone, namely insulin.....improving insulin action may be an important strategy to have a healthier and longer life.
- [Akintola & van Heemst 2015](#) “Insulin, Aging, and the Brain: Mechanisms and Implications”
- Sleep, Fasting, Exercise, Temperature variation, Cognitive Security Practices, Oxygen....

“Insulin signaling in the long-lived reproductive caste of ants”, Yan et al. 2022

To explore the relation between reproduction and longevity, we compared gene expression during caste switching. Insulin expression is increased in the gamergate brain that correlates with increased lipid synthesis and production of vitellogenin in the fat body, both transported to the egg. This results from activation of the mitogen-activated protein kinase (MAPK) branch of the insulin signaling pathway. By contrast, the production in the gamergate developing ovary of anti-insulin Imp-L2 leads to decreased signaling of the AKT/forhead box O (FOXO) branch in the fat body, which is consistent with their extended longevity.

Ants

What is the evolutionary basis or account of aging?

Human

Shared evolutionary history – Complex area!

Next slides will give a distillation of PaperStream #001.0
<https://www.youtube.com/watch?v=le-c-kkJemA> (1:19:25 video)

Of the many Evolutionary phenomena/perspectives to consider, here we will just discuss Kin Selection (KS) & Multi-Level Selection (MLS)

The Relation between Kin and Multilevel Selection: An Approach Using Causal Graphs

<https://www.journals.uchicago.edu/doi/abs/10.1093/bjps/axu047>

Samir Okasha
2016



PaperStream #001.0

Daniel A Friedman

Goals

1. Learn about Kin & Multilevel selection
2. Prepare for upcoming Lab meeting
3. Initiate a new stream series

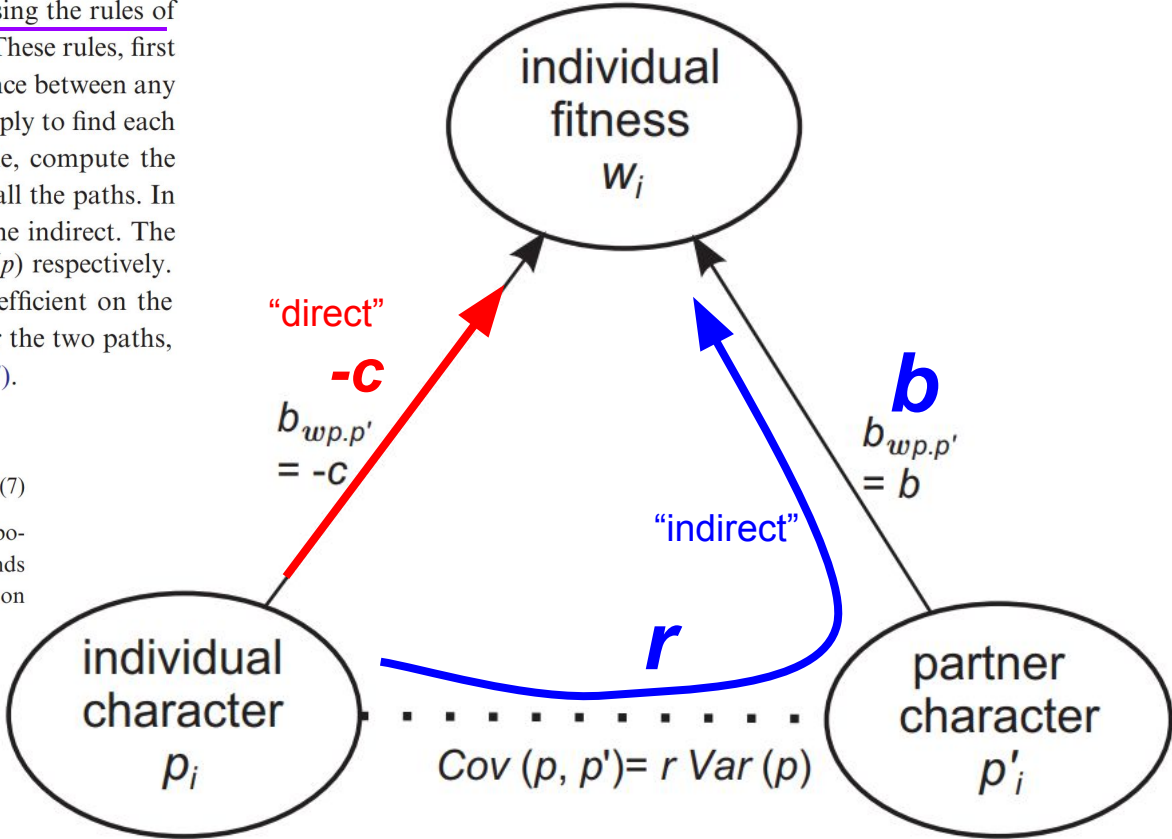
	Proximate	Ultimate
Contemporary		
Historical		Evolution

The value of $Cov(w_i, p_i)$ can be read directly off Figure 2 using the rules of path analysis, yielding the KS decomposition (Equation (7)). These rules, first laid out by Sewall Wright, tell us how to compute the covariance between any two variables on an acyclic causal graph. The basic idea is simply to find each distinct path from the dependent to the independent variable, compute the product of the path coefficients on each path, then sum over all the paths. In Figure 2, there are two paths from w_i to p_i , one direct and one indirect. The products of the coefficients on each path are $-c$ and $(rb)Var(p)$ respectively. (However, since the variables are unstandardized, the $-c$ coefficient on the direct path needs to be multiplied by $Var(p)$).¹⁹ Summing over the two paths, we can thus deduce the value of $Cov(w_i, p_i)$, as in Equation (7).

$$\bar{w}\Delta\bar{p} = \underbrace{\overbrace{(-c)Var(p)}^{\text{direct effect}}}_{-c} + \underbrace{\overbrace{rbVar(p)}^{\text{indirect effect}}}_{rb} . \tag{7}$$

Equation (7) partitions the total change into direct and indirect components, capturing the idea that the overall allele frequency change depends on the direct effect of the allele on its bearers and on the indirect effect on

Kin Selection (KS)



$$\bar{w}\Delta\bar{p} = Cov(w_i, p_i) = (-c)Var(p) + (rb)Var(p)$$

Figure 2. Case where KS is causally adequate.

Suppose we modify Figure 5 by adding causal arrows from p'_i to w_i and from p_i to w'_i , yielding Figure 6. We no longer have a non-social trait, since an individual's fitness is now causally influenced by social partners. However, in Figure 6 the MLS approach is still causally inadequate. For there is still no causal arrow between group p -value, P_i , and group fitness, W_i —they are joint effects of a common cause. So the $Cov(W_i, P_i)$ term of Equation (3) still does not reflect a causal influence of P_i on W_i . Conversely, the KS approach is causally adequate—for both the b and c terms of Equation (7) reflect direct causal influences, as the figure shows. This shows that the presence of fitness-affecting interactions between social partners is not sufficient for the MLS approach to be causally adequate; the group-level covariance may still be a non-causal side effect.

- What biological cases does Figure 6 apply to? The key features are the presence of social interactions between individuals within groups, but the absence of a causal influence of group p -value, P_i , on either individual or group fitness.
- This means that the causal explanation of any individual's fitness can be given in terms that refer only to individual-level properties, that is, p_i and p'_i . This will be true if there is no group-level functional organization, and the groups exhibit no emergent properties of their own, that is, the only group properties are aggregations of individual properties, such as P_i . Many mammalian social groups, for example, buffalo herds and baboon troops, arguably satisfy these conditions.
- Extensive social interactions take place within such groups, but the groups are not functionally integrated in the way that eusocial insect colonies are, for example.

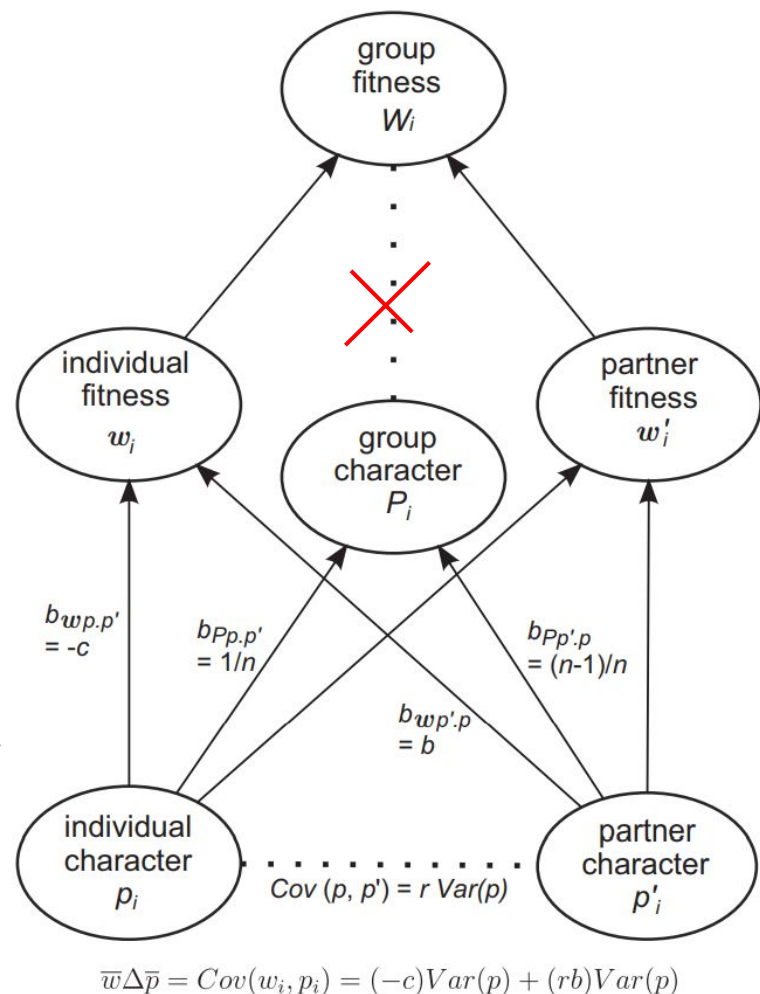


Figure 6. Social trait case where KS is causally adequate, MLS not.

5.4 Cases where MLS is causally adequate

To depict a case where MLS is causally adequate, let us frameshift downwards again as in Section 4.2, so the individuals are alleles and the groups are diploid organisms of size $n = 2$. As before, we consider a single locus with two alleles, A and B , and thus three genotypes, AA , AB , and BB . We define $p_i = 1$ if the i^{th} allele is an A , 0 otherwise; so \bar{p} is the population-wide frequency of the A allele. Assume, first, that meiosis is fair—that is, within-group selection is absent—but there are fitness differences between genotypes. This scenario, which I call ‘genotypic selection with fair meiosis’ is depicted in Figure 8 below.

Note that in Figure 8, a causal arrow leads from group fitness, W_i , to individual fitness, w_i (as in Figure 4b above). This is because it is a group (that is, diploid organism) that has a fitness value in the first instance, that is, it contributes a certain number of successful gametes to the next generation, depending on its genotype. This gametic output is shared equally among group members (that is, the two alleles), owing to the fairness of meiosis, so the fitness of an individual allele (w_i) is simply equal to the fitness of its group (W_i), which is itself causally determined by the group’s p -value (P_i), which in turn is determined by individual p -value (p_i) and partner p -value (p'_i).

There are two key features of Figure 8. First, group fitness, W_i , is primary and individual fitness, w_i , derivative. The fitness of any individual (i.e. allele) is determined by the fitness, or total gametic output, of the group (that is, diploid organism) to which it belongs. So an arrow runs from W_i to w_i , not vice versa.²⁴ Second, group fitness (W_i) is caused by group p -value (P_i), and thus

1. Group fitness (W) is primary
2. Group fitness (W) is caused by group P , and thus only indirectly on p and p'

only indirectly by p_i and p'_i . The reason for this is clear. The full causal explanation of why an organism has a particular fitness value (W_i) would refer to its phenotype, which depends in a complex way on its genotype (P_i), both of which are whole-organism properties. There is no reason to hold that properties of alleles—that is, p and p' —are doing the ‘real’ causal work, nor therefore that the link between P_i and W_i is a mere non-causal correlation.

Figure 8 depicts a situation in which MLS is causally adequate. Within-group selection is absent, so the overall evolutionary change, $\Delta\bar{p}$, is proportional to $Cov(W_i, P_i)$, that is, the covariance between group p -value and group fitness, which is by definition equal to $b_{WP} \cdot Var(P_i)$. Since there is a direct causal influence of P_i on W_i , the strength of which is measured by b_{WP} , it follows that the MLS decomposition is causally adequate.

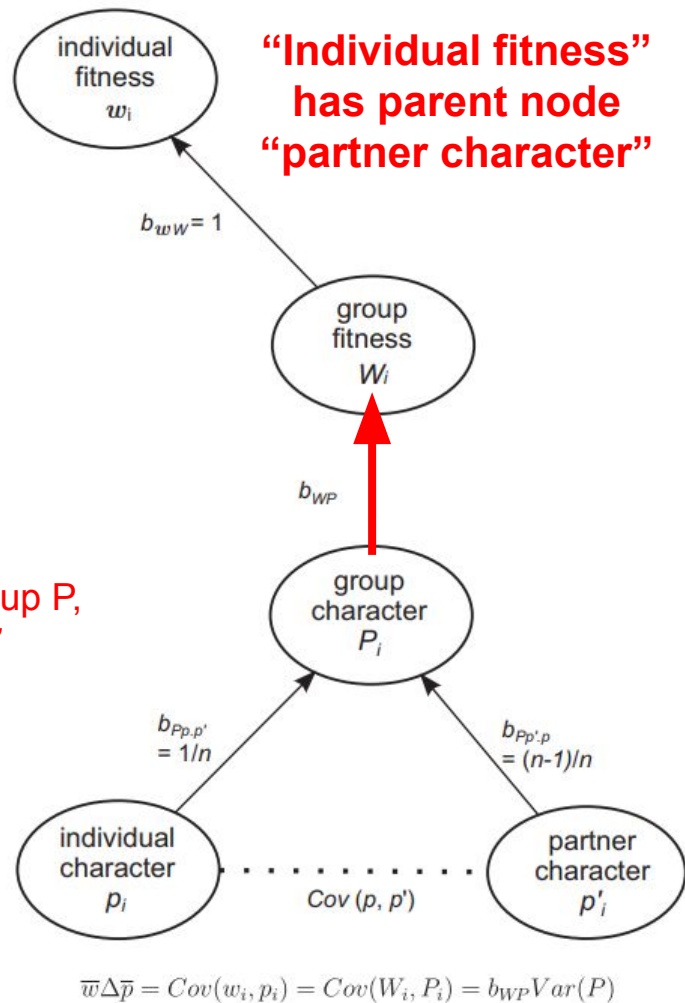


Figure 8. Genotypic selection with fair meiosis.

7 Conclusion

- The opposition between the kin and multilevel selection approaches to social evolution has long polarized evolutionary biologists.
- In recent discussions, theorists have increasingly come to regard the two approaches as equivalent, on the grounds that a correct expression for allele frequency change in structured populations can be written using either approach.
- **However, this establishes only that KS and MLS are formally equivalent, not that they constitute equally adequate causal descriptions of the evolutionary process.**
 - Connection with Active Inference, e.g. *“How particular is the physics of the free energy principle?”* Aguilera et al. 2022
- The bulk of this article has been concerned with fleshing out, in a precise way, the meaning of causal adequacy, and using it to determine when the KS and MLS decompositions of the total evolutionary change count as causally adequate. **The theory presented here does not pretend to be complete. Even if the theory is broadly on the right lines, a number of important issues remain.**
 - One is to get clearer about the implicit metaphysical commitments involved in the multilevel causal graphs above, which contain both individual and group variables.
 - Another is to address the epistemological problem of how we can tell which causal graph is correct in any given case.
 - Finally, further work to map actual empirical cases onto the abstract framework developed here would be useful; the cases discussed in the evolutionary transitions literature are an obvious starting point. These all represent potential avenues for future work.
- [Lastly], the need for the above theory stems from a striking disjunction between the scientific explanations that evolutionary biologists aim to give, which are causal, and the formal models of the evolutionary process that they develop, in which causal concepts do not feature explicitly. **This disjunction is not unique to evolutionary biology, less still to social evolution theory, but is a quite general problem; it stems from the fact that a mathematical framework adequate for describing causal relations has only been developed fairly recently (Pearl [2000]; Spirtes et al. [2001]).**
- Importing this framework into biology, and philosophy of biology, is an important task for the future. Pioneering first steps were taken by Shipley ([2000]), but much remains to be done.

Discussion

Map 1

Ants

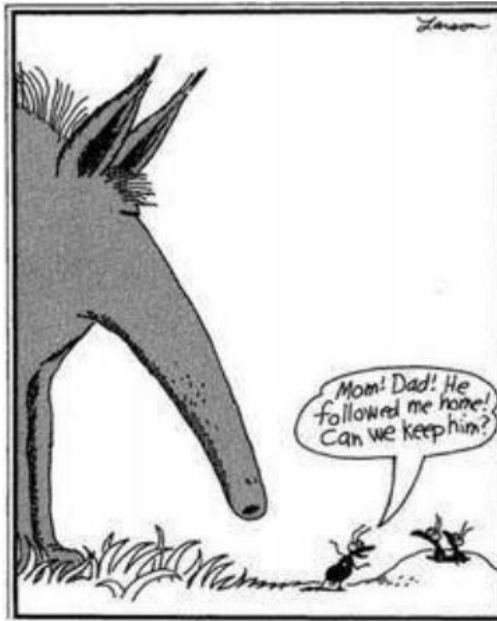
Human

Population

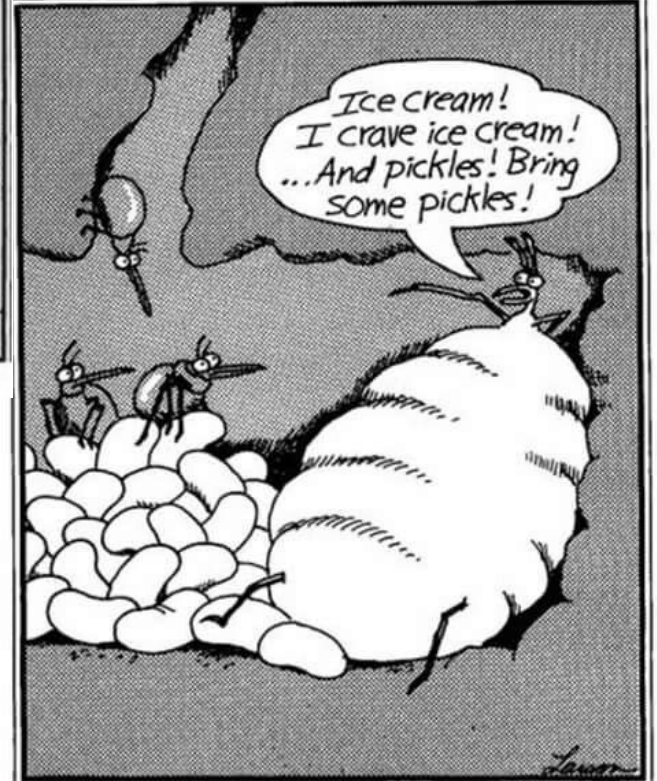
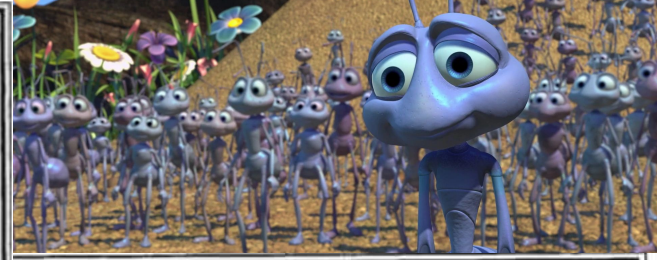
Colony ——— Society

Nestmate ——— Person

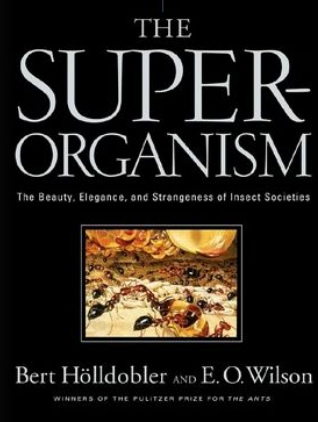
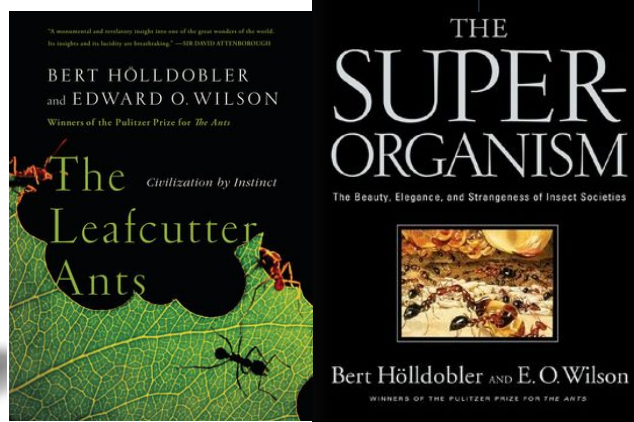
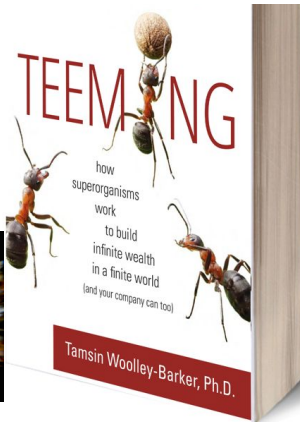
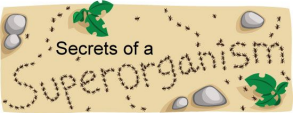
Organ ——— Organ



Childhood Innocence



The termite queen in her egg chamber



Map 2

Ants

Human

Population

Colony

Nestmate

Organ

Society

Person

Organ

Chapter Eight - The Molecular and Evolutionary Genetic Implications of Being Truly Social for the Social Insects [Linksvayer 2015](#)

[Timothy A. Linksvayer](#)  

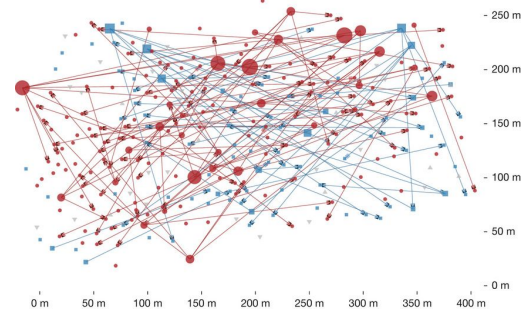
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A variational synthesis of evolutionary and developmental dynamics

[Friston et al. 2023](#)

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THE ANT-COLONY AS AN ORGANISM'

WILLIAM MORTON WHEELER

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[Review](#) > [Q Rev Biol.](#) 2012 Dec;87(4):325-41. doi: 10.1086/668166.

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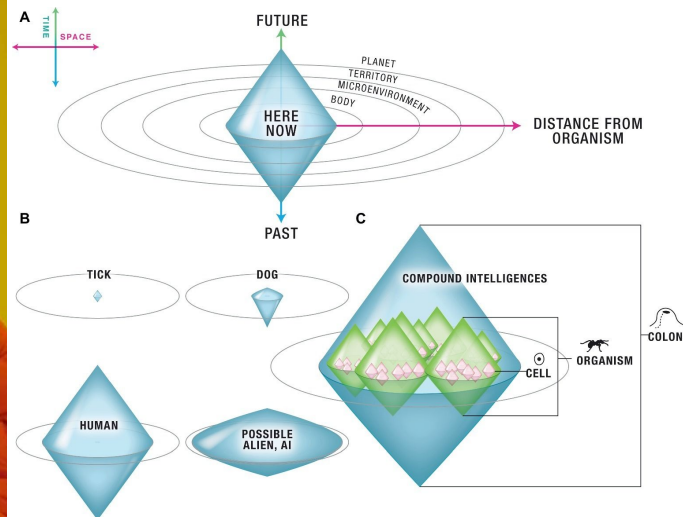
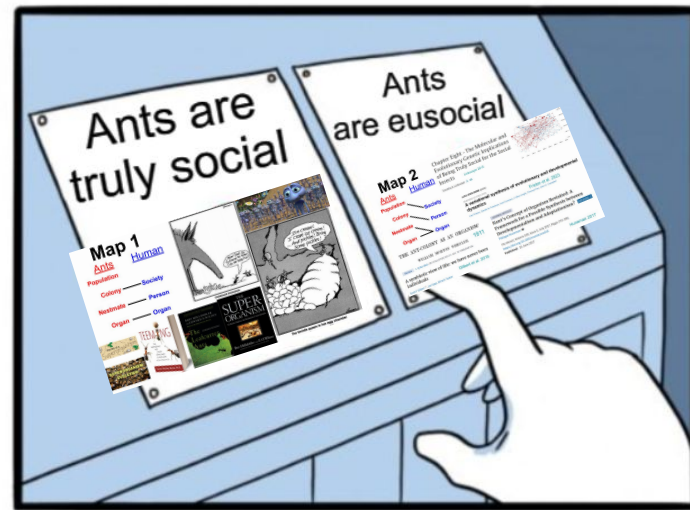
The Molecular and Evolutionary Genetic Implications of Being Truly Social for the Social Insects

Timothy A. Linksvayer¹

Individual versus collective cognition in social insects

Ofer Feinerman¹, Amos Korman²

Eusociality (from Greek εὖ eu "good" and social), the highest level of organization of sociality, is defined by the following characteristics.....



Map 1

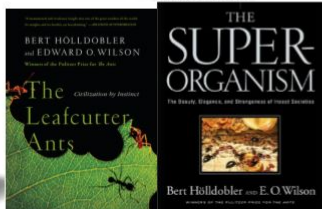
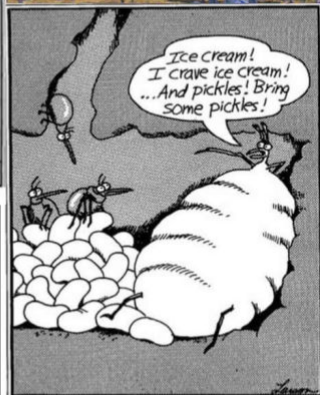
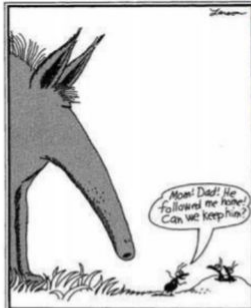
Ants **Human**

Population

Colony — **Society**

Nestmate — **Person**

Organ — **Organ**



Map 2

Ants **Human**

Population

Colony — **Society**

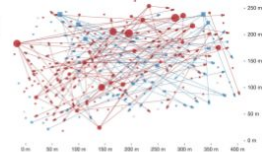
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Pivotal mental states

Brouwer et al. 2020

[Ari Brouwer](#) and [Robin Lester Carhart-Harris](#) [View all authors and affiliations](#)

[Volume 35, Issue 4](#) | <https://doi.org/10.1177/0269881120959637>

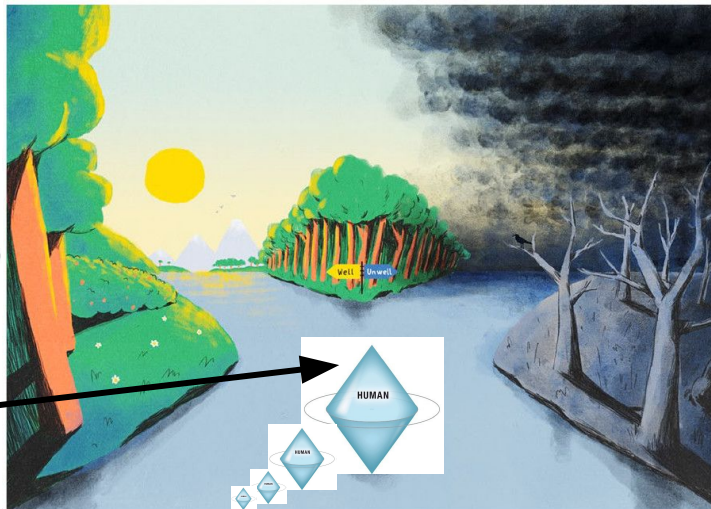
Context over a range of timescales

Past

Recent

Now

Overlapping Contexts that lead up to the Pivotal cognitive moment.



Inference

What is Aging?

Why does Aging occur?

Context over a range of timescales

Now

Recent

Past

What do we do?

Why do we do it?

Action

**Thank you for the
Attention & I will
look forward to
continuing the
Discussion**

