Available online at www.sciencedirect.com

ScienceDirect

Current Opinion in Behavioral Sciences

Bridging social evolution theory and emerging empirical approaches to social behavior

Erol Akçay[1,3], Timothy A Linksvayer[1,3] and Jeremy Van Cleve[2,3]

Spurred on by technological advances, the last several years have seen an explosion of studies of behavioral, genomic, and neurophysiological mechanisms of social behaviors. Yet these empirical studies and the vast amount of data they produce are typically disconnected from well-established social evolution theory. We argue that unlocking the transformative potential of the emerging empirical approaches to social behavior requires new kinds of theoretical approaches that integrate proximate behavioral, genomic, and neurophysiological mechanisms with evolutionary dynamics. We review recent efforts in this direction that show how proximate mechanisms are important for evolutionary dynamics. However, we argue that these frameworks are still too distant from empirical systems to interface with emerging datasets. As an example of improved approaches that can be developed, we focus on the evolution of social gene regulatory networks, and discuss how integrating dynamics of gene regulatory networks with social evolution theory can result in rigorous hypotheses that are testable with sociogenomic data.

Addresses
[1] Department of Biology, University of Pennsylvania, Philadelphia, PA, United States
[2] Department of Biology, University of Kentucky, Lexington, KY, United States

Corresponding author: Akçay, Erol (eakcay@sas.upenn.edu)
[3] These authors contributed equally.

Current Opinion in Behavioral Sciences 2015, **6**:59–64

This review comes from a themed issue on **Integrative study of animal behavior**

Edited by **Dustin Rubenstein** and **Hans Hofmann**

http://dx.doi.org/10.1016/j.cobeha.2015.09.002

Introduction

Increasingly, new technologies are transforming the types and quantities of empirical data available to researchers studying social behavior. For example, sequencing technologies enable rapid probing of the links between gene expression and behavior [1,2,3•], while automated tracking and behavioral data collection ('reality mining' [4]) technologies afford us a look at the dynamics of social behaviors at an unprecedented resolution. Additional new techniques, such as RNAi, CRISPR, and optogenetics, allow experimental manipulation of genetic and neural circuits at increasingly fine scales and are starting to be used for interrogating mechanisms of sociality [5,6].

While major lines of research in the study of social behavior (e.g., behavioral ecology) have long been closely tied to and motivated by social evolution theory (e.g., inclusive fitness theory [7,8] and game theory [9]), these theoretical frameworks have generally not been brought to bear on the new kinds of behavioral and '-omic' data now widely available. This gap between social evolution theory and these new data is primarily a product of the explicit exclusion of genetic, physiological, cognitive, and neural mechanisms from most social evolution models. Because of this gap, it has always been difficult to integrate mechanistic studies of behavior with social evolutionary theory. Yet, the emergence of 'big data' approaches that quantify complex phenomena such as social dynamics or genome-wide patterns of gene expression makes this disconnect even more salient because of a lack of strong quantitative evolutionary hypotheses to be tested with these new data. In the context of '-omics', this situation has recently caused some to express various degrees of skepticism [10•,11•] as to whether these new data will prove transformative or simply distracting from major evolutionary questions.

We contend that the emerging empirical approaches can be transformative, but realizing their full potential will require further development of social evolution theory to explicitly address proximate genetic, physiological, and cognitive mechanisms. We argue this view by outlining how classic social evolution theory has often avoided grappling with these proximate mechanisms in order to produce simple evolutionary predictions and interpretations of behavior. While simple, this classic theory often cannot not investigate the proximate mechanism itself as a legitimate evolving phenotype [12] and thus cannot use genomic or neurophysiological data to test predictions concerning these mechanisms. For instance, despite much interest in dissecting gene regulatory networks underlying social behaviors [2,3•,13], there exists no formal evolutionary theory of how social selection should shape them.

The most progress on investigating the evolution of proximate mechanisms has occurred in theoretical work on flexible behaviors like reciprocity [14–16]. When models do not explicitly account for proximate mechanisms underlying flexible behaviors, an inherent indeterminacy

results where many different behaviors may be equally evolutionarily stable. We review existing theoretical approaches that show how models that include details of the proximate mechanism such as neural processing of material and social rewards can resolve this indeterminacy and yield a clearer prediction for which behaviors evolve. Nevertheless, we argue that these theories still do not deal directly enough with behavioral and neural mechanisms in order to interface with current large-scale and high-resolution behavioral data.

On the genomics side, the absence of formal frameworks that connect directly with proximate mechanisms has meant that most empirical research that leverages sociogenomic and other high resolution data has largely proceeded based on verbal models (with a few exceptions, for example, population genetic models such as [17,18]). As a case study, we highlight the study of the molecular mechanisms and gene regulatory networks underlying social behavior in social insects, which have largely been interpreted using ideas from evolutionary developmental biology, such as the concept of 'genetic toolkits' [19,20], which have relatively little direct connection to formal social evolution theory. In the final section of the paper, we use the gene regulatory network example to sketch how a new approach based on evolutionary systems biology could deeply integrate proximate mechanisms with social evolution theory.

Social evolution theory: the phenotypic gambit

Modern theory of social evolution took off in the 1960s with the theoretical work of Hamilton [7], and Price [21] and Maynard Smith and Price [9] followed shortly afterwards. Hamilton's great contribution was to formalize the notion that when the fitness of a focal individual is determined not just by its own genotype, but also by that of its social partners, the net selection pressure on a trait is determined in part by correlations between the genotypes of interacting individuals, which are often measured by genetic relatedness. Hamilton showed that behaviors such as costly helping will evolve if their 'inclusive fitness effect' is positive, which is encapsulated in his celebrated eponymous rule, $rb - c > 0$. The inclusive fitness effect is defined as the sum of the change in the focal individual's own fitness and that of social partners' that can be ascribed to the focal individual's genotype. Hamilton proposed that inclusive fitness is the quantity maximized by natural selection, which has gained widespread acceptance among behavioral ecologists [22].

The inclusive fitness maximization perspective is intrinsically linked with the 'phenotypic gambit' [23], which holds it is both reasonable and profitable to study the evolution of complex behaviors as if they have a simple additive genetic basis and are unconstrained by the availability of beneficial mutations. The phenotypic gambit caused much controversy between researchers using optimality as a working hypothesis and those that relied on more detailed population and quantitative genetic models [24,25]. The controversy ultimately produced a precise understanding of the relationship between optimality models and population genetic perspectives [26,27,28•,29•] where optimality models are special cases (often approximations) of population genetics models. Moreover, the phenotypic gambit has been very successful in generating adaptive hypotheses and interpretations of complex social behaviors that are otherwise difficult with traditional population genetic models.

However, by focusing solely on phenotypes and their adaptive value, the phenotypic gambit has effectively suppressed the role of proximate mechanisms and genomic architecture in evolutionary theory of social behaviors. By 'genomic architecture' of social traits, we specifically mean the structure and function of gene regulatory networks underlying social trait expression and how segregating variation within these networks maps to variation in social traits. Not surprisingly, this has meant that theory contributed little to progress on questions about evolutionary genetic and genomic patterns for social traits (though see [17,18,30,31], for efforts in this direction). Crucially, social evolution theory does not currently make specific predictions about the details of the molecular and evolutionary genomic architecture of social traits. While population and quantitative genetics generally studies the genetic basis of trait variation as we discuss in the next section, it has not usually explicitly considered the gene regulatory networks that underlie behaviors studied in social evolution (except until recently, for example, [2,3•,13,32•]). This has left unanswered questions such as how gene regulatory network structure evolves when those networks drive social phenotypes. Addressing these questions is key to a comprehensive understanding of the evolution of social phenotypes that spans genes to social groups.

Social interactions and flexible behaviors

Theoretical work on the evolution of flexible behaviors, particularly those like reciprocity that are involved in cooperation, is an important case study for why proximate mechanisms are crucial for evolutionary theories of social behavior. The most common approach to studying flexible behaviors is to use iterated social interactions, or 'games', where a single game is played repeatedly and individuals are endowed with sets of rules that prescribe their behavior in response to past interactions [33]. Although very popular, this approach is hampered by the fact that infinitely many such rules can be devised, which makes evolutionary stable rules indeterminate. Although recent work has put some bounds on the indeterminacy [34], it cannot be resolved completely without addressing the proximate genetic, physiological, or cognitive mechanisms, which could in

principle empirically constrain the set of allowable rules.

Another approach to modeling the evolution of flexible behaviors, called 'interacting phenotypes' or 'indirect genetic effects' (IGE) theory [35–37], extends traditional quantitative genetics to the case of social interactions. IGE theory measures the phenotype of a focal individual in terms of its own genes and those of its social partners (the so-called IGEs). Similar to other models of reciprocity, IGEs have effects on social traits similar to relatedness and can change the direction and rate of social evolution [37]. Furthermore, because IGE theory is based on quantitative genetics, it is eminently suitable for quantifying genetic constraints and feedbacks between social traits [38,39] and for discovering associated quantitative-trait loci (QTLs) and single-nucleotide polymorphisms (SNPs) [40].

One important limitation of conventional IGE theory is that it regards social effects themselves as fixed, rather than as evolving together with the proximate mechanism of behavior. The question of how flexible behaviors evolve as a result of evolving proximate cognitive mechanisms has been addressed by a series of models Akçay, Van Cleve, and colleagues [16,41,42]. In these models both the social behavior and the degree of flexibility, or responsiveness, emerge from evolving preference functions that represent the output of neural circuits weighing the reward values of different options. The problem of evolutionary indeterminacy remains, since an infinity of different preference functions are possible, but it can be dealt with by using behavioral and neuroeconomics experiments to empirically constrain how reward values are processed in the brain [43].

Thus, the IGE framework is well-suited for quantifying behavioral flexibility and genetic constraints, and the responsiveness/preference function framework is appropriate for modeling cognitive and/or neurophysiological mechanisms in an evolutionary context. Neither framework alone, however, fully integrates both genetic and neurophysiological constraints and mechanisms in a manner readily applicable to data. Combining these frameworks is an immediate goal of future work integrating proximate mechanisms with social evolution theory. Further improvements can come from detailed behavioral observations of how animals respond to each other. For example, Johnstone *et al.* [44•] quantified the mechanism of parental responses (in this case, alternating visits) in great tits using nest recordings, and used the empirically determined behavioral model in the evolutionary analysis. As automated behavioral monitoring becomes more widespread, such studies will become more feasible, along with the opportunity to integrate with data on the genetic variation underlying behavioral responses.

Gene regulatory networks for social traits: the case of social insects

Social insects have long been established models for testing a range of predictions of social evolution theory (e.g., [45,46]), such as queen-worker conflict over the sex ratio. More recently, social insect researchers have rapidly embraced sequencing technologies, resulting in a veritable flood of new transcriptomic and genomic data (e.g., [47,48•]), and leading to social insect groups being among the most well-studied animal groups in terms of genomic and transcriptomic resources.

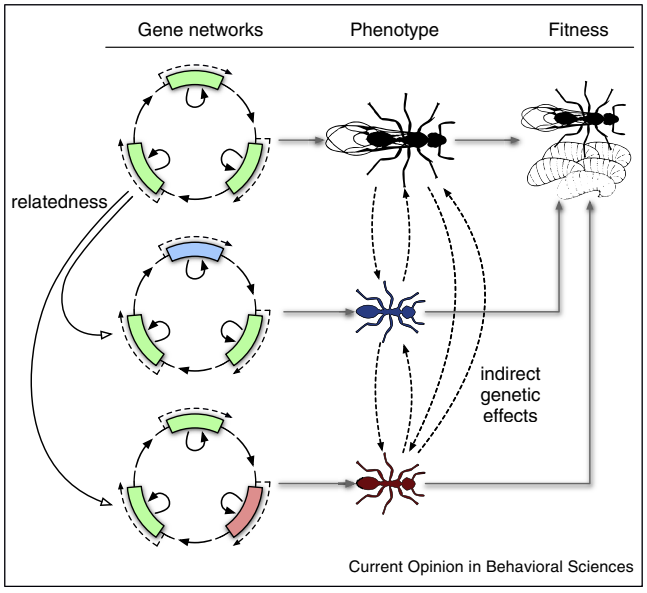
Without a strong and explicit connection between sociogenomic data and social evolution theory, questions in the field of sociogenomics have rarely been explicitly motivated by the predictions from social evolution theory. Instead, sociogenomic studies have often been motivated by conceptual frameworks borrowed from other fields. In particular, the 'evo-devo' approach [49] has inspired the search for 'genetic toolkits' for social behavior, highly conserved genes and gene networks underlying the evolution of diverse social behavior [1,19,20]. While widely accepted and intuitively appealing, precisely defining toolkits for development, let alone social behavior, has proven difficult, leading some authors to question the utility of the framework for hypothesis testing [50]. Moreover, other researchers emphasize the importance of rapidly evolving, or novel genes underlying the evolution of social behavior [32•,51–53]. In reality, both conserved and novel genes presumably play important roles in the evolution of social behavior, with novel components likely added to conserved, core network components over the course of social evolution [3•]. Yet the mere existence of such sets of genes is relatively uninformative about the unique features of gene regulatory networks that are actually involved in social processes such as social signal production and response. These types of gene regulatory networks are the ones that are likely to reflect unique social evolutionary processes.

Social evolutionary systems biology

Thus, several critical questions about the genetic architecture of social behavior remain unexplored by evolutionary theory, such as: how do the unique features of social systems affect the contribution of conserved versus novel genes and gene pathways for the evolution of social traits? Can we expect differences in the evolutionary genomic underpinnings of traits relating to social behavior relative to other traits? Does selection through direct vs. indirect fitness effects differentially shape the genomic architecture of social traits?

We propose that a promising approach to answer questions such as these and generate testable hypotheses for emerging sociogenomic data is to integrate models of gene regulatory networks (GRNs) underlying social traits with social evolution models. As diagrammed in

Figure 1



The social evolutionary systems biology approach and the role of indirect genetic effects. Individuals living in a social group may share genes that are identical by descent due to some kind of population structure where the extent (probability) of gene sharing is given by a relatedness coefficient (denoted by open headed arrows). Genes within individuals form a gene regulatory network where expression from genes in the network is determined by the sum of the regulatory interactions. Expression from genes in the network shapes individual behaviors (phenotypes). Social behaviors result from the interaction of multiple individuals where the behavior of each individual is directly affected by the behavior of its social partners and their underlying GRNs. These social interactions are sometimes called 'indirect genetic effects' [15,35,37] and are denoted by dashed arrows. Fitness (e.g., offspring of a queen ant) is the product of the social behaviors of all individuals in the social interaction. Thus, fitness is affected by the degree of relatedness in the population, the structure and function of the GRNs shaping behavior, and the behavioral interactions among individuals. Finally, genetic variation may exist (different colored genes in the networks), and this variation may result in phenotypic variation (different colored individual ants) and in fitness variation in the population. Heritable variation in fitness results in natural selection on behavior in the social interaction and on the function of the GRNs.

Figure 1, this approach marries evolutionary systems biology, which has been developed mainly in non-social evolutionary settings, with social evolution theory, such as the IGE approach, that has been developed in the absence of models of gene regulation.

One of the main questions in evolutionary systems biology has been how GRNs can produce robust phenotypes (i.e., insensitive to perturbations) while still allowing enough heritable variation for natural selection to act on [54,55]. A general result from this work is that evolved GRNs can reconcile these two seemingly contradictory qualities thanks to large mutational networks with nearly neutral effects [56,57]. Moving to social settings reveals a limitation of this theory, which is that it regards the phenotype as a static property of the individual. When the phenotype is plastic, as it is in many social interactions, robustness and evolvability properties become more complicated, since selection for plasticity means phenotypes need to be malleable under some perturbations, and not under others. Likewise, the generation of a variety of phenotypes from a single genotype can either enhance or diminish evolvability [58]. Relatively little is known about how GRNs evolve to produce plastic phenotypes [59,60]. This gap in our knowledge is even more dire for social phenotypes, since GRNs for such phenotypes need to adapt and respond to both external abiotic environments and to social environments created by other GRNs. As we discuss above, social evolution theory predicts that interactions with relatives and the nature of behavioral flexibility and responsiveness are a critical to social evolution.

Therefore, to understand the evolution of GRNs for social phenotypes, we need to extend them to the social system level [61]. As an example of a first step in this direction, recent social insect studies have sought to identify features of GRNs (e.g., network connectivity) underlying social behavior that have been shaped by social evolution [3•,13,32•]. Recent work also suggests that changes in specific regulatory pathways, such as carbohydrate metabolism, might be associated with the expression and evolution of particular social behaviors [62–64]. A complementary approach to looking at whole GRNs is therefore to focus on the evolution and expression of specific pathways underlying social behavior. We predict that these different types of approaches will yield broad insights into both evolutionary patterns of social gene regulation as well as insights into when and how cooperation and sociality are adaptive.

In conclusion, we believe that realizing the potential of new data-generation technologies for social behaviors will depend on further developing social evolution theory to make meaningful statements about proximate mechanisms. We do not advocate a wholesale rejection of the 'phenotypic gambit' or optimality approaches. Rather, we contend that recognizing proximate mechanisms and genomic architecture as legitimate social phenotypes and applying existing evolutionary theory at that level will yield important insights into the evolution of social life.

Conflict of interest statement

Nothing declared.

Acknowledgements

This work was in part supported by a National Academies Keck Futures Initiatives grant to JVC, TAL and EA. EA was funded by NSF award EF-1137894, and TAL by NSF award IOS-1452520.

References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. Robinson GE, Fernald RD, Clayton DF: **Genes and social behavior**. *Science* 2008, **322**:896-900.

2. Chandrasekaran S, Ament SA, Eddy JA, Rodriguez-Zas SL, Schatz BR, Price ND, Robinson GE: **Behavior-specific changes in transcriptional modules lead to distinct and predictable neurogenomic states**. *Proc Natl Acad Sci U S A* 2011, **108**:18020-18025.

3. • Mikheyev AS, Linksvayer TA: **Genes associated with ant social behavior show distinct transcriptional and evolutionary patterns**. *eLife* 2015, **4**:e04775.

This empirical study asked how structural features of gene regulatory networks associated with ant social behavior influence molecular evolution. The results highlight the roles of both conserved and novel genes associated with social behavior, and also how gene network connectivity can affect molecular evolution.

4. Krause J, Krause S, Arlinghaus R, Psorakis I, Roberts S, Rutz C: **Reality mining of animal social systems**. *Trends Ecol Evol* 2013, **28**:541-551.

5. Rittschof CC, Robinson GE: **Genomics: moving behavioural ecology beyond the phenotypic gambit**. *Anim Behav* 2014, **92**:263-270.

6. Taborsky M, Hofmann HA, Beery AK, Blumstein DT, Hayes LD, Lacey EA, Martins EP, Phelps SM, Solomon NG, Rubenstein DR: **Taxon matters: promoting integrative studies of social behavior: NESCent Working Group on Integrative Models of Vertebrate Sociality: evolution, mechanisms, and emergent properties**. *Trends Neurosci* 2015, **38**:189-191.

7. Hamilton WD: **The genetical evolution of social behaviour. I**. *J Theor Biol* 1964, **7**:1-16.

8. Hamilton WD: **The genetical evolution of social behaviour. II**. *J Theor Biol* 1964, **7**:17-52.

9. Maynard Smith J, Price GR: **The logic of animal conflict**. *Nature* 1973, **246**:15-18.

10. • Travisano M, Shaw RG: **Lost in the map**. *Evolution* 2013, **67**: 305-314.

A skeptical discussion of the value of detailed studies of the genotype–phenotype map. The authors contend that even if the map is worked out in great detail, these studies add little predictive power of the course of evolution, and an approach based on proximate biological processes will be more fruitful.

11. • Zuk M, Balenger SL: **Behavioral ecology and genomics: new directions, or just a more detailed map?** *Behav Ecol* 2014, **25**:1277-1282.

A nuanced take on whether genomics will revolutionize the evolutionary study of behavior. The authors argue that genomics can transform the field, but warn against the danger of getting fixated on 'finding the gene'.

12. Springer SA, Crespi BJ, Swanson WJ: **Beyond the phenotypic gambit: molecular behavioural ecology and the evolution of genetic architecture**. *Mol Ecol* 2011, **20**:2240-2257.

13. Molodtsova D, Harpur BA, Kent CF, Seevananthan K, Zayed A: **Pleiotropy constrains the evolution of protein but not regulatory sequences in a transcription regulatory network influencing complex social behaviours**. *Front Genet* 2014, **5**:431.

14. McNamara JM, Gasson C, Houston AI: **Incorporating rules for responding into evolutionary games**. *Nature* 1999, **401**:368-371.

15. Wolf JB, Brodie IED, Moore AJ: **Interacting phenotypes and the evolutionary process. II. Selection resulting from social interactions**. *Am Nat* 1999, **153**:254-266.

16. Akçay E, Van Cleve J, Feldman MW, Roughgarden J: **A theory for the evolution of other-regard integrating proximate and ultimate perspectives**. *Proc Natl Acad Sci U S A* 2009, **106**:19061-19066.

17. Linksvayer TA, Wade MJ: **Genes with social effects are expected to harbor more sequence variation within and between species**. *Evolution* 2009, **63**:1685-1696.

18. Van Dyken JD, Wade MJ: **Origins of altruism diversity II: Runaway coevolution of altruistic strategies via ''reciprocal niche construction''**. *Evolution* 2012, **66**:2498-2513.

19. Toth AL, Robinson GE: **Evo-devo and the evolution of social behavior**. *Trends Genet* 2007, **23**:334-341.

20. Berens AJ, Hunt JH, Toth AL: **Comparative transcriptomics of convergent evolution: different genes but conserved pathways underlie caste phenotypes across lineages of eusocial insects**. *Mol Biol Evol* 2015, **32**:690-703.

21. Price GR: **Selection and Covariance**. *Nature* 1970, **227**:520-521.

22. Davies NB, Krebs JR, West SA: *An Introduction to Behavioural Ecology*. edn 4. Oxford: Wiley-Blackwell; 2012.

23. Grafen A: **Natural selection, kin selection and group selection**. In *Behavioural Ecology: An Evolutionary Approach,* edn 2. Edited by Krebs JR, Davies NB.Oxford: Blackwell Scientific Publications; 1984:62-84. [chapter 3].

24. Cavalli-Sforza LL, Feldman MW: **Darwinian selection and 'altruism'**. *Theor Popul Biol* 1978, **14**:268-280.

25. Karlin S, Matessi C: **The eleventh R. A. Fisher memorial lecture: kin selection and altruism**. *Proc R Soc B* 1983, **219**:327-353.

26. Uyenoyama M, Feldman MW: **Theories of kin and group selection: a population genetics perspective**. *Theor Popul Biol* 1980, **17**:380-414.

27. Rousset F: *Genetic Structure and Selection in Subdivided Populations*. Princeton: Princeton University Press; 2004.

28. • Van Cleve J: **Social evolution and genetic interactions in the short and long term**. *Theor Popul Biol* 2015, **103**:2-26.

A review of the connections between W.D. Hamilton's classic work on inclusive fitness and more recent efforts to ground his results in population genetic theory for the dynamics of selection and genetic drift in spatially structured populations.

29. • Lehmann L, Alger I, Weibull J: **Does evolution lead to maximizing behavior?** *Evolution* 2015, **69**:1858-1873.

A theoretical paper addressing with great generality a crucial question relating to the phenotypic gambit: when is selection guaranteed to maximize inclusive fitness? They find inclusive fitness is not guaranteed to be maximized in general, but it is when selection is weak and/or, interactions are additive.

30. Van Dyken JD, Linksvayer TA, Wade MJ: **Kin selection-mutation balance: a model for the origin, maintenance, and consequences of social cheating**. *Am Nat* 2011, **177**:288-300.

31. Hall DW, Yi SV, Goodisman MAD: **Kin selection, genomics and caste-antagonistic pleiotropy**. *Biol Lett* 2013, **9**:20130309.

32. • Jasper WC, Linksvayer TA, Atallah J, Friedman D, Chiu JC, Johnson BR: **Large-scale coding sequence change underlies the evolution of postdevelopmental novelty in honey bees**. *Mol Biol Evol* 2015, **32**:334-346.

This study compared the transcriptomic profiles of tissues involved in honey bee social communication to tissues with more conserved function. Tissues involved in social communication had profiles dominated by a small number of very highly expressed, rapidly evolving, and loosely connected genes, demonstrating that rapidly evolving, 'novel' genes play important roles in social evolution.

33. Axelrod R, Hamilton WD: **The evolution of cooperation**. *Science* 1981, **211**:1390-1396.

34. Stewart AJ, Plotkin JB: **From extortion to generosity, evolution in the iterated prisoner's dilemma**. *Proc Natl Acad Sci U S A* 2013, **110**:15348-15353.

35. Moore AJ, Brodie ED III, Wolf JB: **Interacting phenotypes and the evolutionary process: I. Direct and indirect genetic effects of social interactions**. *Evolution* 1997, **51**:1352-1362.

36. Moore AJ, Haynes KF, Preziosi RF, Moore PJ: **The evolution of interacting phenotypes: genetics and evolution of social dominance**. *Am Nat* 2002, **160**:S186-S197.

37. McGlothlin JW, Moore AJ, Wolf JB, Brodie ED: **Interacting phenotypes and the evolutionary process. III. Social evolution**. *Evolution* 2010, **64**:2558-2574.

38. Wolf JB: **Genetic architecture and evolutionary constraint when the environment contains genes**. *Proc Natl Acad Sci U S A* 2003, **100**:4655-4660.

39. Bleakley BH, Brodie ED: **Indirect genetic effects influence antipredator behavior in guppies: estimates of the coefficient of interaction psi and the inheritance of reciprocity**. *Evolution* 2009, **63**:1796-1806.

40. Biscarini F, Bovenhuis H, van der Poel J, Rodenburg TB, Jungerius AP, van Arendonk JAM: **Across-line SNP association study for direct and associative effects on feather damage in laying hens**. *Behav Genet* 2010, **40**:715-727.

41. Akçay E, Van Cleve J: **Behavioral responses in structured populations pave the way to group optimality**. *Am Nat* 2012, **179**:257-269.

42. Van Cleve J, Akçay E: **Pathways to social evolution: Reciprocity, relatedness, and synergy**. *Evolution* 2014, **68**:2245-2258.

43. Schultz o: **Neuronal reward and decision signals: from theories to data**. *Physiol Rev* 2015, **95**:853-951.

44. • Johnstone RA, Manica A, Fayet AL, Stoddard MC, Rodriguez-Gironés MA, Hinde CA: **Reciprocity and conditional cooperation between great tit parents**. *Behav Ecol* 2014, **25**:216-222.

A good example of how behavioral modeling in an evolutionary context can be used to connect to detailed observational data. The authors quantify conditional nest visit probabilities of great tit parents during parental care, and show that this simple behavioral mechanism can resolve much of the evolutionary conflict over biparental care.

45. Crozier RH, Pamilo P: *Evolution of Social Insect Colonies: Sex Allocation and Kin Selection*. Oxford: Oxford University Press; 1996.

46. Queller DC, Strassmann JE: **Kin selection and social insects**. *Bioscience* 1998:165-175.

47. Yan H, Simola DF, Bonasio R, Liebig J, Berger SL, Reinberg D: **Eusocial insects as emerging models for behavioural epigenetics**. *Nat Rev Genet* 2014, **15**:677-688.

48. • Kapheim KM, Pan H, Li C, Salzberg SL, Puiu D, Magoc T, Robertson HM, Hudson ME, Venkat A, Fischman BJ, Hernandez A, Yandell M, Ence D, Holt C, Yocum GD, Kemp WP, Bosch J, Waterhouse RM, Zdobnov EM, Stolle E, Kraus FB, Helbing S, Moritz RFA, Glastad KM, Hunt BG, Goodisman MAD, Hauser F, Grimmelikhuijzen CJP, Pinheiro DG, Nunes FMF, Soares MPM, Tanaka ED, Sim oes ZLP, Hartfelder K, Evans JD, Barribeau SM, Johnson RM, Massey JH, Southey BR, Hasselmann M, Hamacher D, Biewer M, Kent CF, Zayed A, Blatti C, Sinha S, Johnston JS, Hanrahan SJ, Kocher SD, Wang J, Robinson GE, Zhang G: **Genomic signatures of evolutionary transitions from solitary to group living**. *Science* 2015, **348**:1139-1143.

Comparing the genome sequences of 10 bee species, this study found a robust tendency for more complex gene regulatory interactions with increased social complexity. The gene families that increased in complexity however were lineage specific, with little overlap in rapidly evolving genes between separate complex eusocial lineages.

49. Carroll SB, Grenier JK, Weatherbee SD: *From DNA to Diversity: Molecular Genetics and the Evolution of Animal Design*. edn 2. Malden, MA: Blackwell Publishing; 2005.

50. Wilkins A: **'The genetic tool-kit': the life-history of an important metaphor**. In *Advances in Evolutionary Developmental Biology.* Edited by Streelman J. Hoboken, NJ: John Wiley & Sons, Inc.; 2013.

51. Johnson BR, Linksvayer TA: **Deconstructing the superorganism: social physiology, groundplans, and sociogenomics**. *Q Rev Biol* 2010, **85**:57-79.

52. Feldmeyer B, Elsner D, Foitzik S: **Gene expression patterns associated with caste and reproductive status in ants: worker-specific genes are more derived than queen-specific ones**. *Mol Ecol* 2014, **23**:151-161.

53. Sumner S: **The importance of genomic novelty in social evolution**. *Mol Ecol* 2014, **23**:26-28.

54. Alon U: *An Introduction to Systems Biology: Design Principles of Biological Circuits*. CRC Press; 2006.

55. Aldana M, Balleza E, Kauffman S, Resendiz O: **Robustness and evolvability in genetic regulatory networks**. *J Theor Biol* 2007, **245**:433-448.

56. Wagner A: **Does evolutionary plasticity evolve?** *Evolution* 1996, **50**:1008-1023.

57. Ciliberti S, Martin OC, Wagner A: **Robustness can evolve gradually in complex regulatory gene networks with varying topology**. *PLoS Comput Biol* 2007, **3**:e15.

58. Ancel LW: **Undermining the Baldwin expediting effect: Does phenotypic plasticity accelerate evolution?** *Theor Popul Biol* 2000, **58**:307-319.

59. Draghi JA, Whitlock MC: **Phenotypic plasticity facilitates mutational variance, genetic variance, and evolvability along the major axis of environmental variation**. *Evolution* 2012, **66**:2891-2902.

60. Kaneko K: **Phenotypic plasticity and robustness: evolutionary stability theory, gene expression dynamics model, and laboratory experiments**. In *Evolutionary Systems Biology.* Edited by Soyer OS. Springer; 2012:249-278.

61. Linksvayer TA, Fewell JH, Gadau J, Laubichler MD: **Developmental evolution in social insects: Regulatory networks from genes to societies**. *J Exp Zool (Mol Dev Evol)* 2012, **318**:159-169.

62. Woodard SH, Fischman BJ, Venkat A, Hudson ME, Varala K, Cameron SA, Clark AG, Robinson GE: **Genes involved in convergent evolution of eusociality in bees**. *Proc Natl Acad Sci U S A* 2011, **108**:7472-7477.

63. Chandrasekaran S, Rittschof C, Djukovic D, Gu H, Raftery D, Price N, Robinson G: **Aggression is associated with aerobic glycolysis in the honey bee brain**. *Genes Brain Behav* 2015, **1**:158-166.

64. Rittschof CC, Grozinger CM, Robinson GE: **The energetic basis of behavior: bridging behavioral ecology and neuroscience**. *Curr Opin Behav Sci* 2015, **6**:19-27.