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## Research



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## Evolutionary biology

# Genes underlying altruism

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William D. Hamilton postulated the existence of 'genes underlying altruism', under the rubric of inclusive fitness theory, a half-century ago. Such genes are now poised for discovery. In this article, we develop a set of intuitive criteria for the recognition and analysis of genes for altruism and describe the first candidate genes affecting altruism from social insects and humans. We also provide evidence from a human population for genetically based trade-offs, underlain by oxytocin-system polymorphisms, between alleles for altruism and alleles for non-social cognition. Such trade-offs between self-oriented and altruistic behaviour may influence the evolution of phenotypic diversity across all social animals.

## 1. Introduction

Can genes mediate the expression of altruism? Hamilton began his career and made his most profound impacts on evolutionary biology by considering this controversial question in light of Darwin's conundrum: how selection could favour individually costly behaviours, such as those exemplified by self sacrifice and sterility in social insects.

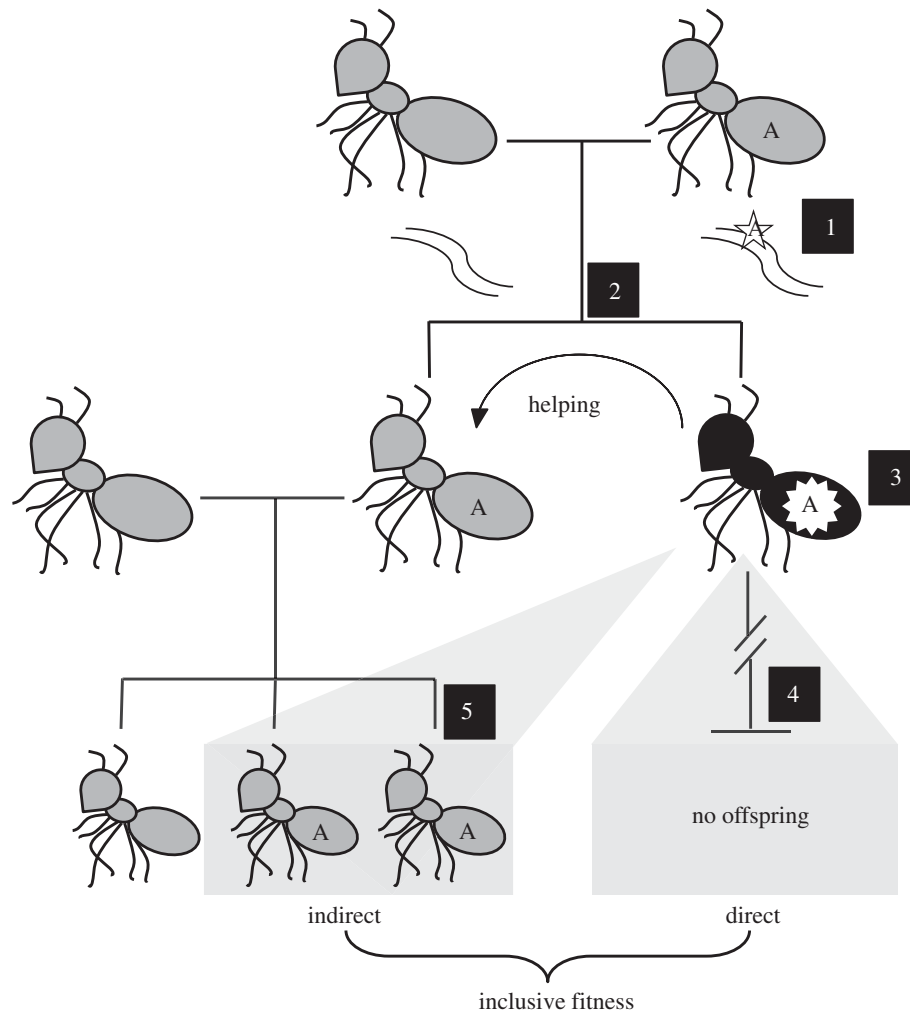
Hamilton showed how altruism could evolve if 'altruism genes' had the effect of augmenting the fitness of other carriers, even at some expense to the altruist's own direct fitness [1,2]. Hamilton did so by demonstrating in population genetic terms how an individual's fitness could be partitioned into direct (descendant kin) and indirect (collateral kin) components—the latter arising through social interaction—and then summed to total 'inclusive fitness' (figure 1). Despite the widespread use of Hamilton's gene-centric theory to explain evolved aspects of social life, a chasm remains between our conceptual understanding of social evolution and our knowledge of the specific genes and alleles that underlie it, and how they code for altruistic behaviour.

We attempt to bridge this gap between genetic theory and genes. First, we develop a set of criteria to characterize genes underlying altruism, in comparison, for example, to genes affecting cooperation, selfishness or spite. Second, we identify and describe the current best examples of genes and alleles for altruism and suggest approaches to identify the molecular mechanisms that underpin Hamilton's theory. Finally, we present data relating two candidate genes for human altruism (OXTR and CD38), which mediate levels of plasma oxytocin, to their phenotypic effects on social and non-social cognition and behaviour.

## 2. Recognizing genes for altruism

We first describe a set of testable hypotheses that, in whole or in part, describe the evolution, expression and expected characteristics of genes for altruism.

(i) Genes underlying altruism should satisfy Hamilton's rule of  $rb > c$ , where  $r$  is relatedness of actor to recipient,  $b$  is benefits to the recipient and  $c$  is costs to the actor [3]. Altruism exists, and to the extent that this type of behaviour has evolved, we expect genetic variation to underlie it. In this sense, there



**Figure 1.** The origin, effects and transmission of a hypothetical allele for altruism. A germ-line mutation for reproductive altruism arises (1) and is transmitted to descendant kin (2). In the focal individual, the gene is turned 'ON' (3) and, as a consequence, the first-generation altruist directs help towards reproducing relatives who are also likely to carry, but do not express, the gene. If the altruistic effect is strong (e.g. self sacrifice or sterility) then the altruist has no direct fitness (4). But even in this extreme case, copies of Hamilton's 'gene for altruism' can gain representation in the next generation through indirect transmission (5).

must be genes 'for' altruism (genes showing allelic variation that is statistically associated with variation in altruistic behaviour) that are potentially detectable. From social insects, to microorganisms, social birds and mammals and humans, there are examples of reproductive altruism that ostensibly evolve through socially mediated selection at the level of the gene [2]. In this regard, genes for altruism are alleles that underlie Hamilton's  $rb > c$  decisions that result in fitness benefits to collateral kin and fitness costs to self.

(ii) Genes underlying altruism should be environmentally sensitive. If genes for altruism are to evolve, then at least some carriers must reproduce. This inference implies that genes underlying altruism should be conditionally expressed as a function of their social environment. Specifically, these genes should be environmentally switched 'on' in altruistic helpers, yet remain unexpressed and switched 'off' in reproductive recipients (figure 1). In this context, genes differentially expressed between reproductive (selfish) and non-reproductive (altruistic) castes, or between reproductive and non-reproductive individuals within castes or other groups, can be considered as genes for altruism to the extent that they mediate determination of caste, drive the divergence of reproductive and helping behaviour or maintain such differences. Alternatively, the evolution of genetically based altruistic behaviour may proceed from initially environmentally cued phenotypic diversity in

helping versus reproduction, which serves as a substrate for the evolution of conditionally expressed altruistic alleles that consolidate divergence between incipient castes [4]. Under both scenarios, the evolutionary change is ultimately genetic.

(iii) Genes underlying altruism should increase in number and complexity with social-behavioural sophistication. A mutation that predisposes its carrier towards altruism—for example, a mutation for being smaller, or less reproductive or dispersive, or a better forager, defender or carer—could involve a single locus. If selection favours the initial gene-based division in labour, then selection at the gene, individual and colony levels may amplify and embellish this division through disruptive selection that favours more and more specialized or efficient divisions in labour, involving larger numbers of downstream and interacting genes. The genetic architecture of altruistic castes should therefore involve few loci initially, but more loci as caste roles become more pronounced, specialized and interdependent. Comparative genomic analyses between weakly and strongly social species will provide a powerful test of this idea and help to determine if functional diversity arises from new genes, new gene regulation or both [5].

(iv) Genes underlying altruism should coevolve with, or depend on, the previous evolution of genes for kin recognition. Hamilton's 1964 papers [1] stressed the importance of

**Table 1.** Select examples of candidate genes underlying altruism in insects and humans.

gene	evidence	refs
Queen – worker caste differences		
uncharacterized loci	heritable variation for queen versus worker caste differentiation in various species of ants and bees	[10,12]
<i>Worker</i>	allelic segregation at potentially just one X-linked locus affects royal versus worker caste differentiation in multiple species of subterranean termites	[13]
<i>Neofem2</i>	expression of <i>Neofem2</i> in drywood termite queens is required for reproductive suppression of workers	[14]
Worker – worker interactions		
<i>OvA1 – OvA4</i>	quantitative trait loci that influence worker sterility and selfish reproduction in the European honeybee	[15]
<i>Sting-1</i>	quantitative trait locus that influences selfless colony defence in Africanized worker honeybees	[16,17]
<i>Thelytoky</i>	allelic segregation at this locus influences development into queen-like versus altruistic worker castes in the Cape honeybee	[18]
Human social groups		
AVPR1a RS3 microsatellite repeat	amounts of money allocated to others in dictator game, and self-report measures of altruism	[19,20]
COMT rs4680	psychological-genetic test of altruistic donations to poor, anonymous children	[21]
DRD4, DRD5, IGF2 polymorphisms	self-report measures of selflessness, empathy and altruism	[22,23]
GABRB2 rs187269	self-report measures of altruism; gene is subject to imprinting	[24]
OXTR SNPs, rs1042778, rs2254298	amounts of money allocated to others in dictator game	[23,25]

relatedness and recognized that the evolution of altruism among relatives requires mechanisms for individuals to direct their help towards other carriers. The ability to recognize other carriers may come from identification of kin (or reliable correlates of kin) through genetic or shared environmental cues. Alternatively, the ability to recognize other carriers, regardless of kinship, may be coupled with genes for altruism, if altruism is caused by ‘greenbeard’ genes (single genes that programme a signal, its recognition in others and altruistic responses to it), as in some social insects [6]. Greenbeard genes thus show the potential for coevolution of recognition and kin-directed benefits. In humans, social learning appears to underpin kin recognition, but the altruism-promoting neuropeptide oxytocin has been shown to enhance the perception of kin relationships, at least for females [7].

(v) Genes for altruism may reside in regions of low recombination, exhibit co-expression and show modular genetic architecture. If transitions in social evolution involve increasing numbers of genes recruited to caste specific phenotypes, as described above, then these loci may eventually become linked into co-expressed and co-adapted gene complexes that collectively encode worker-like or queen-like traits. That linkage driven by epistasis among genes for socially correlated traits could result in ‘social chromosomes’ is intriguing [8], and an example of such social-genetic architecture effects from ants [9] resembles Y-borne gene clusters that encode sex-specific traits of mammals. A test for widespread linkage of caste-biased genes is possible from mapping these and other genes for altruism onto regions of relatively low or high recombination on single genomes.

(vi) Genes underlying altruism should be at least partially additive. The evolution of altruism requires heritable variation, and we therefore expect genes for this and other

evolved social traits to have significant additive effects that are responsive to kin-mediated selection. Data from termites and social Hymenoptera suggest that the size and nature of genetic effects on caste include a mix of major and minor genes, and potential roles for both additive and non-additive effects [10].

(vii) Genes underlying altruism should exhibit strong pleiotropy. Pleiotropy (multiple phenotypic effects of alleles) should be fundamental to the evolution of altruism, given that it involves combinations of costs and benefits that may be simultaneously physiological, morphological, reproductive and behavioural. For example, in honeybees (*Apis mellifera*), the yolk precursor protein vitellogenin is synthesized by the reproductive queen and invested directly into her developing offspring (eggs). Workers, by contrast, are functionally sterile but nonetheless synthesize this protein for a different reason: they incorporate it into royal jelly and feed it to the queen’s brood [11]. This example demonstrates how single genes can coevolve with sociality to perform multiple social functions—in this case, parental versus alloparental care. Moreover, genes like *vitellogenin* that function on ‘both sides’ of the division in reproductive labour exemplify how the evolution of altruism involves genes that modulate trade-offs between reproduction and helping.

How well do real genes match these criteria? A set of the first candidate genes underlying altruism in social insects and humans is presented in table 1. From these and other potential examples, kin selection (criterion i) and recognition (criterion iv) are strongly implicated, while gene-by-environment effects (criterion ii), response to selection (criterion vi), and pleiotropy (criterion vii) are either implied or directly reported. The remaining two criteria—namely, gene number (iii) and linkage (v) remain mostly untested. These examples also suggest that

there are three main selective arenas where the effects of genes underlying altruism are most commonly expressed: caste determination, worker reproduction and social groups of kin and non-kin.

### 3. Three arenas for altruism

#### (a) Caste determination

Queen–worker caste determination in eusocial insects represents a crucial decision point between lifetime relative selfishness and relative altruism. Such decisions have, for many years, been considered to be predominantly or entirely cued by environmental factors that influence the timing and proportion of queen versus worker production. Under this mostly environmental model, genes for altruism can be recognized as alleles that increase in frequency as helper caste differences evolve, and alleles that mediate environmental responses towards selfish versus altruistic development. Queen–worker caste determination as a more-or-less stable threshold trait should, under this framework, be underlain by some combination of environmental (including social) effects and gene  $\times$  environment interactions that affect the conditional production of one or another caste.

Despite expectations from theory of environmental caste determination, a notable suite of recent studies has uncovered instances where caste determination is subject to major genetic effects [10] (table 1). These findings suggest that genes for altruism may also include queen- and worker-biasing alleles of large effect. If so, how could such alleles be favoured or maintained?

First, selection at the colony level may favour genetic mechanisms that are relatively impervious to reproductive cheating by workers, especially in taxa where the costs of producing ‘excess’ reproductives are relatively low [26]. Another possibility would be essentially the opposite: queen–worker, worker–worker and male–female conflicts over caste determination may maintain variation through ongoing genetically based conflicts [10,27]. Under these scenarios, strong selection for alleles that bias caste towards reproductives would generate major gene effects that engender colony-level costs, or individual-level deleterious effects associated with homozygosity of the selfish, reproductive allele, in both cases leading to more or less balanced maintenance by selection [28]. This conflict hypothesis is consistent with apparent or expected high colony-level costs of genetic caste determination in some ants, bees and termites, and with the unusual mechanisms involved in many cases of genetic caste determination, including hybridization effects (which may release selfish caste-drive alleles from repression) and asexuality (which ‘selfishly’ replicates the genomes of reproductives) [10,26]. To the extent that genetically based queen–worker caste determination systems are indeed controlled by genomic conflicts, worker-biasing alleles may be less altruistic than coerced or forced, to the relative detriment of their bearers.

#### (b) Worker reproduction

In the same way that selection should favour development as a reproductive compared with a worker, it should also promote reproduction by workers that is subject to suppression by queens, workers or both, and opposing selection due to colony-level costs [2]. The strength of such selective effects is

evidenced by the rarity of complete sterility in social insect workers and by the high frequencies of worker reproduction and within-colony conflicts over reproduction [29]. In this context, alleles ‘for’ not reproducing as a worker represent alleles for altruism, and these can be recognized, in part, as alternative allelic variants to alleles ‘for’ selfish worker reproduction. In honeybees, genes associated with selfish worker egg-laying have already been mapped [15].

Evidence for large-effect selfish worker alleles has accumulated for several social taxa (table 1), but the degree to which worker reproduction is mediated by genes of large or small effects, compared with environmental circumstances, remains unknown. Unlike the case of queen–worker divergence in reproduction, which is favoured by specialization that reduces the impact of life-historical, physiological and behavioural trade-offs, worker–worker divergence should be strongly constrained, behaviourally (by queens and other workers) and through frequency-dependent selection. Despite such differences, the same genes and mechanisms might underpin queen–worker and worker–worker divergence in reproduction, given that the processes involved are fundamentally the same, and genomes are shared between queens and workers [30].

#### (c) Social groups of kin and non-kin

A third major domain of genes underlying altruism is structured social groups that comprise mixtures of kin and non-kin—especially humans [31]. What are the genes for altruism in humans, and are they comparable with those in social insects in modulating balances between social and self-oriented abilities and behaviour?

Studies of altruism and sociality in humans have reported strong effects of oxytocin and vasopressin allelic variation, and experimental oxytocin administration, on a variety of altruistic social phenotypes [25,32]. These considerations suggest that genes affecting altruism in humans may be represented as the set of functional genetic variants that modulate oxytocin–vasopressin–dopaminergic neural and neuroendocrine systems (including AVPR1A, COMT, DRD4, DRD5, GABRB2 and OXTR in table 1), and thereby influence neurodevelopment, personality traits and expression of altruistic behaviours. In theory, alleles underlying such systems that enhance the expression of altruism have increased during human evolution, a hypothesis that has yet to be studied but could be evaluated through analyses of positive selection and the ancestral versus derived status of functionally characterized alleles. Do such alleles more or less specifically modulate behavioural-investment decisions regarding descendant and collateral kin, and non-relatives? Behavioural-genetic studies of human altruism have yet to incorporate effects of relatedness. By contrast, might such alleles mediate more fundamental and general cognitive-behavioural phenotypes and trade-offs, as postulated for the maintenance of polygenic personality traits [33]?

We evaluated the hypothesis that single nucleotide polymorphisms (SNPs) associated in a previous study with serum oxytocin levels [34] and altruistic behaviour in an experimental game [25] might mediate cognitive trade-offs, by genotyping three such polymorphisms (in the OXTR and CD38 genes) in a human population that we also scored for performance on a paradigmatic visual-spatial, non-social task: mental rotation (MR) of a three-dimensional object



**Table 2.** For two SNPs in the OXTR gene, individuals with genotypes associated with higher plasma oxytocin and more-altruistic behaviour in experimental games show worse performance on the MR test, a measure of visual-spatial ability. Details of the methods are provided in Leach *et al.* [35]. Patterns of similarity or difference in plasma oxytocin between the two 'relatively high oxytocin' genotypes of rs1042778 and rs3796863 have not been reported, so the details of these genotype–phenotype associations remain unknown.

OXTR rs1042778 genotype	GG	GT ( <i>T less altruistic</i> )	TT ( <i>lower OT</i> ) ( <i>T less altruistic</i> )	<i>p</i> (by ANOVA)
test group	MR score (s.d.) ( <i>N</i> )	MR score (s.d.) ( <i>N</i> )	MR score (s.d.) ( <i>N</i> )	
both sexes	9.79 (4.95) (182)	11.09 (5.06) (218)	11.05 (4.6) (88)	0.021*
males	11.66 (5.29) (65)	13.55 (4.66) (75)	13.41 (4.43) (37)	0.055
females	8.75 (4.44) (117)	9.80 (4.8) (143)	9.33 (3.95) (51)	0.18
OXTR rs2254298 genotype	CT	CC ( <i>lower OT, altruism</i> )	n.a.	
test group	MR score (s.d.) ( <i>N</i> )	MR score (s.d.) ( <i>N</i> )		
both sexes	9.71 (5.06) (93)	10.79 (4.93) (389)		0.058
males	12.84 (5.02) (32)	12.84 (4.92) (144)		0.99
females	8.07 (4.27) (61)	9.59 (4.54) (245)		0.018*
CD38 rs3796863 genotype	TT	TG	GG ( <i>lower OT</i> )	
test group	MR score (s.d.) ( <i>N</i> )	MR score (s.d.) ( <i>N</i> )	MR score (s.d.) ( <i>N</i> )	
both sexes	10.2 (5.4) (50)	10.43 (4.8) (199)	10.76 (5.0) (230)	0.68
males	12.8 (5.2) (17)	12.2 (4.8) (69)	13.23 (4.9) (86)	0.43
females	8.88 (5.0) (33)	9.48 (4.6) (130)	9.28 (4.4) (144)	0.78

(table 2). MR ability is negatively correlated with social-empathic interest and skills in this population [36]. For two of the SNPs, the genotypes linked with lower plasma oxytocin and less-altruistic behaviour were also associated with better performance in MR (table 2). These results are suggestive of genetically based trade-offs between social and non-social phenotypes in humans, such that alleles underlying altruism, at least considered as alleles for high plasma oxytocin and altruism in experimental games, trade-off with alleles for some non-social phenotypes.

## 4. Conclusions

Students of social evolution are now poised to discover and characterize the 'genes for altruism' postulated 50 years ago by Hamilton. In social insects, altruism genes direct the evolution of thresholds and trade-offs between reproducing contrasted with helping, and the evolution of the specialized selfish-

reproductive versus highly social and largely altruistic suites of queen–helper phenotypes that result. Among humans, genetically based altruism may also involve trade-offs, here between non-social, self-focused cognition and behaviour versus prosociality and altruism. As Hamilton [37, p. 205] opined

People divide roughly, it seems to me, into two kinds, or rather a continuum is stretched between two extremes. There are things people and people people.

Do alleles underlying self-orientation or selfishness, compared with prosociality and altruism, generate axes of behavioural and cognitive trade-offs across all social forms?

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## References

- Hamilton WD. 1964 The genetical evolution of social behaviour, I and II. *J. Theor. Biol.* **7**, 1–52. (doi:10.1016/0022-5193(64)90038-4)
- Bourke AFG. 2011 *Principles of social evolution*, p. 267. New York, NY: Oxford University Press.
- Hamilton WD. 1963 The evolution of altruistic behavior. *Am. Nat.* **97**, 354–356. (doi:10.1086/497114)
- Crozier RH. 1992 The genetic evolution of flexible strategies. *Am. Nat.* **139**, 218–223. (doi:10.1086/285322)
- Robinson GE, Ben-Shahar Y. 2002 Social behavior and comparative genomics: new genes or new gene regulation? *Genes Brain Behav.* **1**, 197–203. (doi:10.1034/j.1601-183X.2002.10401.x)
- Gardner A, West SA. 2010 Greenbeards. *Evolution* **64**, 25–38. (doi:10.1111/j.1558-5646.2009.00842.x)
- Fischer-Shofty M, Levkovitz Y, Shamay-Tsoory SG. 2013 Oxytocin facilitates accurate perception of competition in men and kinship in women. *Soc. Cogn. Affect. Neurosci.* **8**, 313–317. (doi:10.1093/scan/nsr100)
- Linksvayer TA, Busch JW, Smith CR. 2013 Social supergenes of superorganisms: do supergenes play important roles in social evolution? *BioEssays* **35**, 683–689. (doi:10.1002/bies.201300038)
- Wang J, Wurm Y, Nipitwattanaphon M, Riba-Grognuz O, Huang YC, Shoemaker D, Keller L. 2013 A Y-like social chromosome causes alternative colony organization in fire ants. *Nature* **493**, 664–668. (doi:10.1038/nature11832)
- Schwander T, Lo N, Beekman M, Oldroyd BP, Keller L. 2010 Nature versus nurture in social insect caste differentiation. *Trends Ecol. Evol.* **25**, 275–282. (doi:10.1016/j.tree.2009.12.001)
- Amdam GV, Norberg K, Hagen A, Omholt SW. 2003 Social exploitation of vitellogenin. *Proc. Natl Acad. Sci. USA* **100**, 1799–1802. (doi:10.1073/pnas.0333979100)
- Smith CR, Toth AL, Suarez AV, Robinson GE. 2008 Genetic and genomic analyses of the division of labour in insect societies. *Nat. Rev. Genet.* **9**, 735–748. (doi:10.1038/nrg2429)

13. Kitade O, Hoshi M, Odaira S, Asano A, Shimizu M, Hayashi Y, Lo N. 2011 Evidence for genetically influenced caste determination in phylogenetically diverse species of the termite genus *Reticulitermes*. *Biol. Lett.* **7**, 257–260. (doi:10.1098/rsbl.2010.0856)
14. Korb J, Weil T, Hoffmann K, Foster KR, Rehli M. 2009 A gene necessary for reproductive suppression in termites. *Science* **324**, 758. (doi:10.1126/science.1170660)
15. Oxley PR, Thompson GJ, Oldroyd BP. 2008 Four quantitative trait loci that influence worker sterility in the honeybee (*Apis mellifera*). *Genetics* **179**, 1337–1343. (doi:10.1534/genetics.108.087270)
16. Hunt GJ, Guzman-Novoa E, Fondrk MK, Page RE. 1998 Quantitative trait loci for honey bee stinging behavior and body size. *Genetics* **148**, 1203–1213.
17. Arechavala-Velasco ME, Hunt GJ, Emore C. 2003 Quantitative trait loci that influence the expression of guarding and stinging behaviors of individual honey bees. *Behav. Genet.* **33**, 357–364. (doi:10.1023/A:1023458827643)
18. Lattorff HMG, Moritz RFA, Crewe RM, Solignac M. 2007 Control of reproductive dominance by the thelytoky gene in honeybees. *Biol. Lett.* **3**, 292–295. (doi:10.1098/rsbl.2007.0083)
19. Knafo A *et al.* 2008 Individual differences in allocation of funds in the dictator game associated with length of the arginine vasopressin 1a receptor RS3 promoter region and correlation between RS3 length and hippocampal mRNA. *Genes Brain Behav.* **7**, 266–275. (doi:10.1111/j.1601-183X.2007.00341.x)
20. Avinun R, Israel S, Shalev I, Gritsenko I, Bornstein G, Ebstein RP, Knafo A. 2011 AVPR1A variant associated with preschoolers' lower altruistic behavior. *PLoS ONE* **6**, e25274. (doi:10.1371/journal.pone.0025274)
21. Reuter M, Frenzel C, Walter NT, Markett S, Montag C. 2011 Investigating the genetic basis of altruism: the role of the COMT Val158Met polymorphism. *Soc. Cogn. Affect. Neurosci.* **6**, 662–668. (doi:10.1093/scan/nsq083)
22. Bachner-Melman R, Gritsenko I, Nemanov L, Zohar AH, Dina C, Ebstein RP. 2005 Dopaminergic polymorphisms associated with self-report measures of human altruism: a fresh phenotype for the dopamine D4 receptor. *Mol. Psychiatr.* **10**, 333–335. (doi:10.1038/sj.mp.4001635)
23. Anacker K, Enge S, Reif A, Lesch KP, Strobel A. 2013 Dopamine D4 receptor gene variation impacts self-reported altruism. *Mol. Psychiatr.* **18**, 402–403. (doi:10.1038/mp.2012.49)
24. Tsang SY *et al.* 2013 Social cognitive role of schizophrenia candidate gene GABRB2. *PLoS ONE* **8**, e62322. (doi:10.1371/journal.pone.0062322)
25. Israel S *et al.* 2009 The oxytocin receptor (OXTR) contributes to prosocial fund allocations in the dictator game and the social value orientations task. *PLoS ONE* **4**, e5535. (doi:10.1371/journal.pone.0005535)
26. Lo N, Beekman M, Oldroyd BP. 2010 Caste in social insects: genetic influences over caste determination. In *Encyclopedia of animal behavior* (eds MD Breed, J Moore), pp. 254–260. Oxford, UK: Oxford University Press.
27. Schwander T, Humbert JY, Brent CS, Cahan SH, Chapuis L, Renai E, Keller L. 2008 Maternal effect on female caste determination in a social insect. *Curr. Biol.* **18**, 265–269. (doi:10.1016/j.cub.2008.01.024)
28. Libbrecht R, Schwander T, Keller L. 2011 Genetic components to caste allocation in a multiple-queen ant species. *Evolution* **65**, 2907–2915. (doi:10.1111/j.1558-5646.2011.01348.x)
29. Ratnieks FLW, Foster KR, Wenseleers T. 2006 Conflict resolution in insect societies. *Annu. Rev. Entomol.* **51**, 581–608. (doi:10.1146/annurev.ento.51.110104.151003)
30. Oldroyd BP, Thompson GJ. 2007 Behavioural genetics of the honey bee, *Apis mellifera*. In *Advances in insect physiology*, vol. 33 (ed. SJ Simpson), pp. 1–49. London, UK: Academic Press.
31. Haldane JBS. 1932 *The causes of evolution*. London, UK: Longmans Green.
32. De Dreu CKW. 2012 Oxytocin modulates cooperation within and competition between groups: an integrative review and research agenda. *Horm. Behav.* **61**, 419–428. (doi:10.1016/j.yhbeh.2011.12.009)
33. Nettle D. 2007 *Personality: what makes you the way you are*, p. 298. Oxford, UK: Oxford University Press.
34. Feldman R, Zagoory-Sharon O, Weisman O, Schneiderman I, Gordon I, Maoz R, Shalev I, Ebstein RP. 2012 Sensitive parenting is associated with plasma oxytocin and polymorphisms in the OXTR and CD38 genes. *Biol. Psychiatry* **72**, 175–181. (doi:10.1016/j.biopsych.2011.12.025)
35. Leach EL, Hurd PL, Crespi BJ. 2013 Schizotypy, cognitive performance, and genetic risk for schizophrenia in a non-clinical population. *Pers. Individ. Differ.* **55**, 334–338. (doi.org/10.1016/j.paid.2013.03.010)
36. Dinsdale NL, Hurd PL, Wakabayashi A, Elliot M, Crespi BJ. 2013 How are autism and schizotypy related? Evidence from a non-clinical population. *PLoS ONE* **8**, e63316. (doi:10.1371/journal.pone.0063316)
37. Hamilton WD. 1993 *Between Shoreham and Downe: seeking the key to natural beauty*. Inamori foundation Kyoto prize commemorative lecture. Oxford, UK: Oxford University Press. (Reprinted by Oxford University Press 2006.)