genes produced no phenotype however, suggesting functional redundancy between the JAZ proteins. This was further indicated by the dominant phenotype of a line with a carboxy-terminal deletion in JAZ1: the transgenic line was insensitive to jasmonate, and male sterile.

These results therefore provide the first convincing evidence for the protein targets of the SCF^{COI1} ubiquitin ligase: the JAZ proteins. This indicates that the likely function of the JAZ proteins is to repress jasmonate responses, but does not provide the mechanism. Chini *et al.* [4] cloned *JAI3,* an *Arabidopsis* gene defined by the dominant jasmonate-insensitive (but male fertile) mutant *jin3*, and found that it corresponds to *JAZ3*. *jai3* encodes a protein lacking the carboxy-terminal domain of JAZ3. They showed that in transgenic plants expressing *the JAI3–GFP* fusion, the GFP protein disappeared after treatment with jasmonate, in a COI1-dependent manner, and also that JAI3/JAZ3 bound to COI1 in *in vitro* pull-downs. Significantly, they also showed that the *jai3* mutation prevented the jasmonate-induced destruction of other JAZ proteins. The most likely explanation is that the carboxy-terminal part of JAI3/JAZ3 is required for dissociation from COI1, and therefore in this mutant the COI1 protein is not free to participate in the degradation of other JAZ proteins. The likely target of the JAZ was revealed to be MYC2. Pull-down assays and yeast two-hybrid assays indicated that JAI3/JAZ3 binds MYC2. Not only does this transcription factor activate transcription of jasmonate-responsive genes, it also activates transcription of the *JAZ* genes.

These results suggest a model for the jasmonate perception response pathway (Figure 1) which shows striking parallels to the auxin perception-response pathway [10]. In this model the negative feedback loop is predicted to attenuate the jasmonate signal. The model provides a very welcome advance, and makes several predictions. Presumably, transcription factors besides MYC2 also interact with JAZ proteins, in part because MYC2 activity alone does not account for all of the jasmonate functions. It is also possible, as the authors point out, that different jasmonates interact with different JAZ proteins in binding to COI1. This would permit the overlapping functions of the different JAZ proteins to be regulated independently, and could account for the diverse responses of plants to stress, and to jasmonates in particular.

References

1. Turner, J.G., Ellis, C., and Devoto, A. (2002). The jasmonate signal pathway. Plant Cell *14*, S153–S164.
2. Devoto, A., Ellis, C., Magusin, A., Chang, H.S., Chilcott, C., Zhu, T., and Turner, J.G. (2005). Expression profiling reveals COI1 to be a key regulator of genes involved in wound- and methyl jasmonate-induced secondary metabolism, defence, and hormone interactions. Plant Mol. Biol. *58*, 497–513.
3. Thines, B., Katsir, L., Melotto, M., Niu, Y., Mandaokar, A., Liu, G., Nomura, K., He, S.-H., Howe, G.A., and Browse, J. (2007). JAZ repressor proteins are targets of the SCF^{COI1} complex during jasmonate signalling. Nature *448*, 661–665.
4. Chini, A., Fonseca, S., Fernandez, g., Adie, B., Chico, J.M., Lorenzo, O., Garcia-Casado, G., Lopez-Vidriero, I., Lozano, F.M., Ponce, M.R., *et al*. (2007). The JAZ family of repressors is the missing link in jasmonate signalling. Nature *448*, 666–671.
5. Staswick, P.E., and Tiryaki, I. (2004). The oxylipin signal jasmonic acid is activated by an enzyme that conjugates it to isoleucine in Arabidopsis. Plant Cell *16*, 2117–2127.
6. Xie, D.X., Feys, B.F., James, S., Nieto-Rostro, M., and Turner, J.G. (1998). COI1: an Arabidopsis gene required for jasmonate-regulated defense and fertility. Science *280*, 1091–1094.
7. Devoto, A., Nieto-Rostro, M., Xie, D.X., Ellis, C., Harmston, R., Patrick, E., Davis, J., Sherratt, L., Coleman, M., and Turner, J.G. (2002). COI1 links jasmonate signalling and fertility to the SCF ubiquitin-ligase complex in Arabidopsis. Plant J. *32*, 457–466.
8. Bai, C., Sen, P., Hofmann, K., Ma, L., Goebl, M., Harper, J.W., and Elledge, S.J. (1996). SKP1 connects cell cycle regulators to the ubiquitin proteolysis machinery through a novel motif, the F-box. Cell *86*, 263–274.
9. Gagne, J.M., Downes, B.P., Shiu, S.H., Durski, A.M., and Vierstra, R.D. (2002). The F-box subunit of the SCF E3 complex is encoded by a diverse superfamily of genes in Arabidopsis. Proc. Natl. Acad. Sci. USA *99*, 11519–11524.
10. Dharmasiri, N., Dharmasiri, S., and Estelle, M. (2005). The F-box protein TIR1 is an auxin receptor. Nature *435*, 441–445.
11. Lorenzo, O., Chico, J.M., Sanchez-Serrano, J.J., and Solano, R. (2004). Jasmonate-insensitive1 encodes a MYC transcription factor essential to discriminate between different jasmonate-regulated defense responses in Arabidopsis. Plant Cell *16*, 1938–1950.

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Evolutionary Conflicts: Rapid Suppression of a Male-Killer

Conflicts between and within species can drive fast evolutionary change. A recent study has documented remarkably rapid counter-adaptations in the wild to an extreme sex-ratio distortion caused by a bacterial symbiont.

Oliver Y. Martin and Matthew J.G. Gage

A universal feature of most species that reproduce sexually is that the sex-ratio is balanced at 50:50. In 1930, Ronald Fisher [1] provided a theoretical explanation for this equilibrium in the case where the costs of producing sons or daughters are the same: when one sex is produced in greater numbers, then the other sex becomes rarer; if that sex is rare, individuals of that sex will also enjoy greater relative reproductive success; evolutionary selection will then act to increase the production of the more reproductively successful sex, causing the sex-ratio to swing back to 50:50. In a recent paper, Sylvain Charlat and colleagues [2] demonstrate remarkable speed in the evolution of the sex-ratio of natural populations of *Hypolimnas bolina* butterflies. Susceptibility to a sex-ratio distorting parasite had

driven populations to an extreme female bias (99:1), but counter-adaptation by the host, captured by Charlat *et al.* [2], led to the sex-ratio evolving back to unity in just five years.

Selfish genetic elements exist throughout vertebrates and invertebrates, and their evolutionary effects may be significant, particularly through distortion of the genetic architecture and systems of sex determination [3]. One sex-ratio distorter that has received much attention is the bacterium *Wolbachia*. This microbe infects many arthropods [4,5] and manipulates its hosts in ways likely to promote its own fitness. *Wolbachia* is maternally inherited, and has been shown to possess various mechanisms capable of achieving a shift in host sex-ratio towards females. Host-specific sterility, feminization of genetic males, induction of parthenogenesis or male-killing are all recognised routes by which *Wolbachia* achieves a female-biased sex-ratio in the host, and thereby increases its rate of self-propagation [4]. Beyond sex-determination, *Wolbachia* can also marginalise host populations that it has infected by inducing cytoplasmic incompatibility, where uninfected individuals are at a reproductive disadvantage and thus potentially leading to reproductive isolation between populations [3,4]. These effects can lead to further evolutionary responses in phenotypic traits of the host, such as changes in reproductive behaviour [6].

In the blue moon butterfly (*H. bolina*), which exists across the Indo-Pacific, some Polynesian populations are susceptible to a strain of *Wolbachia* (*w*Bol1) which causes male-killing in the embryos of its host. Before 2001, and as far back as 1923, records show that sex-ratios were extremely female-biased as a result of male-killing, with only 1% of the population being male [7]. Before 2001, therefore, these populations appeared to carry no resistance to the male-killing parasite [7]. However, records over the last few years on two Samoan islands of Upolu and Savii, revealed a remarkably rapid evolutionary change from 99% female in the population in 2001 [7], to a sex-ratio of unity by 2006 [2]. Given the interactions between host and parasite and sexual conflict in the *Wolbachia:Hypolimnas* system, it was predicted that fast evolutionary responses might be recorded [8]. But the study of natural evolutionary responses has shown that evolutionary conflict can fuel incredibly rapid changes in important reproductive traits in the wild. A more distant population (Sagone) appears to be in transition with a currently still female-biased ratio. The evolutionary response was not due to infection or suppression by a new *Wolbachia* strain, as sequence data obtained using the polymerase chain reaction (PCR) showed that all current butterflies carry the infection and that the strain remains the same as in 2001. The evolutionary response is consistent with the proliferation of a suppressor gene that nullifies the deficit in males caused by male-killing *Wolbachia.* Recent related work [8] on this system using crosses between butterfly populations that are resistant and susceptible to male-killing suggests that the suppressor gene is nuclear, resides at a single dominant locus and acts zygotically.

The relationship between host and parasite within the *Wolbachia* system is an excellent example of one rife with evolutionary conflict. Evolutionary conflicts can fuel evolutionary arms races, resulting in accelerated evolution as either player tries to 'out-evolve' the other. In fact, given that *Wolbachia* specifically influences reproductive traits, infected populations suffer selection from both inter-species conflict between the host and parasite, and also a shift in the within-species sexual conflict between males and females. Sexual conflict has been proposed to be responsible for rapid-evolving reproductive proteins [9] and reproductive isolation between populations [10]. Because *Wolbachia* influences male–female reproductive dynamics, such as operational sex-ratio and compatibility, the result has been a shift in how sexual selection and conflict act on male and female reproductive optima. For example, a population's operational sex-ratio is an important balance that leads to evolution of male–male competition, female choice and the dynamic of sexual conflict. If the sex-ratio is unusually skewed away from the Fisherian equilibrium and, for example, to a female majority, then this can lead to sex-role reversal, with evolution then selecting on males to become the 'choosy' sex, and females becoming competitive in actively soliciting matings, as in *Acraea* butterflies [11]. In *H. bolina* butterflies, a female-biased sex-ratio changes the female mating pattern, leading to prudent male ejaculate investment to match the increase in mating opportunities [12]. A similar change in strategic ejaculate allocation is known to have resulted from sex-ratio distortion by other agents too, for example in the amphipod *Gammarus duebeni* where males allocate fewer sperm to females infected with the microsporidian sex-ratio distorter *Nosema granulosis* [13].

The discovery by Charlat *et al.* [2] is of general significance because similar changes are likely to have occurred in the past, and be happening now in other systems. Suppressors could hinder the detection of infections if typical phenotypes (skewed sex-ratio, lack of males) are not apparent. Also, *Wolbachia* is not the only bacterial endosymbiont known to affect host reproduction. Arthropods are known to harbour other microbial symbionts such as *Rickettsia*, *Spiroplasma* and *Cardinium*, with similar consequences [4,5,14]. Considering such a large proportion of microbial species remain unknown, it also seems highly likely that more bacteria of this type will be discovered in future. Even multiple infections with different strains or species of endosymbiotic bacteria within a host occur [5]. In other populations of *Hypolimnas* across the South Pacific, *Wolbachia* strains instead cause cytoplasmic incompatibility, and not the

male-killing strain described in Upolu and Savii [15]. Populations infected with this second strain can resist invasion from the male-killer *w*Bol1, adding more complexity and yet another level of evolutionary conflict to the mix.

Compelling support for rapid evolution often stems from laboratory studies, yet here the research documents transitions in the wild without experimental interference. Charlat *et al.* [2] have caught evolution-in-action on islands that have really lived up to their 'natural laboratory' tag. Evidence indicating rapid host counter-adaptation after 10 generations, obtained without sustained targeted selection possible in experimental evolution studies, is impressive and shows what an effective agent of punctuated change conflict-based evolution can be. The data captured from the natural environment avoid criticisms of artificial selection, commonly levelled at lab experiments. Powerful evidence from field systems is understandably rare, because it requires being around at the right place, at the right time, *and* measuring the right trait(s) using the right tools. The issue of timing is particularly keen here, as the extreme female-biased sex-ratio seems to have persisted for at least 78 years — between 1923 and 2001 — before rapidly switching and approaching parity in 2005/2006 [2,7]. Dynamics over this period, and in the future, beg many further questions of this powerful system. It would be extremely informative to know how the sex ratio varied in the female-biased population before the suppressor joined the fray? How did the suppressor enter the population: via immigration or mutation? What are the exact mechanisms by which *w*Bol1 kills male embryos, and how does the suppressor nullify this effect? What are the costs, if any, of carrying the suppressor? When, if ever, will (*w*Bol1) change and out-evolve the suppressor? The continued effective combination of population and behavioural ecology with modern molecular genetic techniques, as well as some laboratory experimental control, should allow some of these important evolutionary questions to be answered.

References

1. Fisher, R.A. (1930). The Genetical Theory of Natural Selection (Oxford: Clarendon Press).
2. Charlat, S., Hornett, E.A., Fullard, J.H., Davies, N., Roderick, G.K., Wedell, N., and Hurst, G.D.D. (2007). Extraordinary flux in sex ratio. Science *317*, 214.
3. Hurst, G.D.D., and Werren, J.H. (2001). The role of selfish genetic elements in eukaryotic evolution. Nature Rev. Genet. *2*, 597–606.
4. Hurst, G.D.D., and Jiggins, F.M. (2000). Male-killing bacteria in insects: mechanisms, incidence and implications. Emerg. Infect. Dis. *6*, 329–336.
5. Goodacre, S.L., Martin, O.Y., Thomas, C.F.G., and Hewitt, G.M. (2006). *Wolbachia* and other endosymbiont infections in spiders. Mol. Ecol. *15*, 517–527.
6. Vala, F., Egas, M., Breeuwer, J.A.J., and Sabelis, M.W. (2004). *Wolbachia* affects oviposition and mating behaviour of its spider mite host. J. Evol. Biol. *17*, 692–700.
7. Dyson, E.A., and Hurst, G.D. (2004). Persistence of an extreme sex-ratio bias in a natural population. Proc. Natl. Acad. Sci. USA *101*, 6520–6523.
8. Hornett, E.A., Charlat, S., Duplouy, A.M.R., Davies, N., Roderick, G.K., Wedell, N., and Hurst, G.D.D. (2006). Evolution of male-killer suppression in a natural population. PLOS Biol. *4*, e283.
9. Swanson, W.J., and Vacquier, V.D. (2002). The rapid evolution of reproductive proteins. Nature Rev. Genet. *3*, 137–144.
10. Martin, O.Y., and Hosken, D.J. (2003). The evolution of reproductive isolation through sexual conflict. Nature *423*, 979–982.
11. Jiggins, F.M., Hurst, G.D.D., and Majerus, M.E.N. (2000). Sex-ratio-distorting Wolbachia causes sex-role reversal in its butterfly host. Proc. Roy. Soc. Lond. B *267*, 69–73.
12. Charlat, S., Reuter, M., Dyson, E.A., Hornett, E.A., Duplouy, A.M., Davies, N., Roderick, G.K., Wedell, N., and Hurst, G.D. (2007). Male killing bacteria trigger a cycle of increasing male fatigue and female promiscuity. Curr. Biol. *17*, 273–277.
13. Dunn, A.M., Andrews, T., Ingrey, H., Riley, J., and Wedell, N. (2006). Strategic sperm allocation under parasitic sex-ratio distortion. Biol. Lett. *2*, 78–80.
14. Zchori-Fein, E., and Perlman, S.J. (2004). Distribution of the bacterial symbiont *Cardinium* in arthropods. Mol. Ecol. *13*, 2009–2016.
15. Charlat, S., Engelstadter, J., Dyson, E.A., Hornett, E.A., Duplouy, A.M., Tortosa, P., Davies, N., Roderick, G.K., Wedell, N., and Hurst, G.D. (2006). Competing selfish genetic elements in the butterfly *Hypolimnas bolina*. Curr. Biol. *16*, 2453–2458.

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Autism: Not Interested or Not 'Tuned-in'?

Recent studies of perceptual adaptation to faces have revolutionised our understanding of neural mechanisms that support face recognition. A new study has applied this approach to autistic spectrum disorders, revealing severe deficits in such adaptation.

Greg Davis and Kate Plaisted

Human faces differ subtly along many different dimensions, yet the human brain is able to distinguish between them with a rapidity and precision unmatched by any artificial recognition system. Given their central role in human interaction, the brain's face-processing mechanisms have understandably been the subject of intense scrutiny, and functional imaging (fMRI) studies have located such mechanisms are present in regions of occipitotemporal cortex. However, progress in understanding *how* these mechanisms function has been frustratingly slow. Some theories suggest that the brain encodes many individual faces independently of each other, whereas other theories postulate that all faces are coded relative to an average or 'prototype' face. Indeed, it is only recently that simple, but ingenious studies of the way that face processing mechanisms alter their responses ('adapt') when exposed to new face