OXFORD GENETICS

GENETICS, 2025, 230(3), iyaf096
https://doi.org/10.1093/genetics/iyaf096
Advance Access Publication Date: 2 June 2025
The 2025 GSA Honors and Awards

The shape of discovery: a journey through genetics, coevolution, and a scientific life

Noah K. Whiteman *

Division of Genetics, Genomics, Evolution and Development, Department of Molecular and Cell Biology, Department of Integrative Biology, University of California, Berkeley, CA 94720, USA

*Corresponding author: Email: whiteman@berkeley.edu

The Genetics Society of America Medal honors an individual member of the Society for their contributions to the field of genetics at the mid-career level. Whiteman is recognized for his impactful contributions to the field through genetic and evolutionary studies on herbivorous insects, their host plants, and their natural enemies. Additionally, his use of genetic tools, including genome editing, has led to a better understanding of herbivore resistance in plants and plant defenses. His insightful leadership allowed for the retracing of the adaptive walk taken by the monarch butterfly's lineage over many years resulting from a study that used CRISPR-Cas9 to characterize candidate toxin resistance mutations from the monarch butterfly into Drosophila melanogaster.

Genetics was the first upper division natural sciences course I took in 1995 as a sophomore at Saint John's University in rural, central Minnesota. The campus was founded by Benedictine monks and featured both Bavarian-inspired brick architecture and modern concrete buildings designed by Bauhaus architect Marcel Breuer, who had fled Nazi Germany for Harvard. The Abbey Church was his masterpiece, with its stunning wall of stained glass held by 430 honeycomb frames of concrete that formed a rainbow mosaic, rising 150 feet into the air and stretching across the entire width of the building (the largest wall of stained glass in the world). As a

sheltered young man from rural northeastern Minnesota, I was so dazzled by the wall of color and light that it took my breath away when I first visited the campus—a moment of transcendent beauty that unwittingly set the course of my scientific life.

Three years later, hexagons of another sort—the paper cells of social wasp nests—phenocopies of those made by honeybees from wax—stared back at me as I worked on my Honors Thesis. I was investigating ecological character displacement in *Polistes fuscatus*, the northern paper wasp. My advisor, Jim Poff, had collected these wasps on a road trip from Minnesota to Louisiana, and I was measuring their body sizes to determine to what extent Minnesota populations expanded their niche through ecological release compared to southern populations that competed for prey with other *Polistes* species. Under my dissecting scope, I saw that the compound eyes of the wasps were also constructed of hexagons—individual facets called ommatidia, which first evolved in proto-crustaceans at least half a billion years ago.

Jim had drawn my thesis idea from a book entitled "Coevolution," which was edited by the evolutionary biologist Douglas Futuyma and evolutionary geneticist Montgomery (Monty) Slatkin (Futuyma and Slatkin 1983). Its chapters revealed what would become my passion: how interactions between species, like competition, can result in reciprocal evolutionary change. Those long hours in the ascetic Engel Science Center (also designed by Breuer) where I measured wasps in the dark Minnesota winter were deeply satisfying as countless 6-sided snowflakes fell onto the frozen surface of Lake Sagatagan down the hill.

Why hadn't I chosen genetics for my thesis topic? Though Genetics was my first upper division course, I struggled with the quantitative aspects due to math anxiety that persisted until graduate school. Despite this, Charles (Chuck) Rodell's course exposed me to both its conceptual and physical beauty. In the lab, we studied linkage and measured recombination rates using *Drosophila melanogaster* and even karyotyped our own chromosomes from blood samples. Despite my enthusiasm, I earned a B, which discouraged me from pursuing more quantitative courses. Instead, I gravitated toward organismal biology, which better dovetailed with my background as a naturalist.

In hindsight, that B was an unexpected gift. The challenge of quantitative problems was both anxiety-provoking and thought-provoking. Like a moth to a flame, genetics slowly drew me in because it was difficult yet intriguing. It eventually became a lodestar around which my research interests would orbit. As Chuck drew the hexagonal rings of purines and pyrimidines on the board, I glimpsed the Abbey Church's hexagons from the corner of my eye and saw how that shape reverberated across so many levels of biological organization: from the stained glass built by humans, to the nests built by wasps, to their compound eyes, and even to the nucleotides that made it all possible.

I received an MS degree in Entomology from the University of Missouri, Columbia and my project again focused on species interactions—this time on water beetles and the plants they were associated with, including herbivorous species like the beautiful, emerald green *Donacia* beetles whose larvae bored into the stems of water lilies. My advisor, Robert (Bob) Sites finally called out my math anxiety and because of him I confronted it and overcame it, receiving as in all of my statistics courses. Bob also taught me, with great patience and kindness, how to write scientifically.

I then moved to St. Louis to do my PhD in what was then one of the most burgeoning tropical biology programs anywhere owing to its connections with the Missouri Botanical Garden and Saint Louis Zoo. In my first year there, I continued to build my quantitative skills by focusing on analysis of empirical data and rigorous coursework (Robert [Bob] Ricklefs, a student of Robert MacArthur's, taught me Population and Community Ecology). But the truth is, I was lost, both intellectually and personally. I switched labs in my first year, came out of the closet, and began to think about what research I might do under my new advisor, Patricia (Patty) Parker, who was starting a disease ecology research program in Galapagos. I chose a topic centered on co-divergence of island populations of the Galápagos Hawk and its ectoparasites because I had seen a lecture from Dale Clayton on co-speciation between doves and their feather lice and Patty had been studying that hawk because of its unusual mating system of cooperative polyandry. In 2002, I found myself doing fieldwork on Espumilla Beach on Santiago Island—I was the same age as Charles Darwin when he'd landed there in 1835 during the voyage of the HMS Beagle. This project allowed me to marry my background in natural history with new training in population and evolutionary genetics. In this way, I discovered that the hawks and some of their ectoparasites had potentially been coevolving (Whiteman *et al.* 2006), co-diverging across the geographic islands (Whiteman *et al.* 2007), and that the hosts themselves could cause their ectoparasite populations to become genetically distinct (Koop *et al.* 2014). To remove the ectoparasites from the birds, I used pyrethrum with piperonyl butoxide and its 6-membered ring.

As my dissertation was waning, I wanted to learn experimental, molecular genetics in the context of co-adaptation between hosts and parasites, not just co-speciation. For personal reasons I wanted to be in the Boston area and was searching for potential mentors. So, after a Google search, I cold-emailed Professor Naomi Pierce at the Museum of Comparative Zoology (MCZ) and Professor Frederick Ausubel at Massachusetts General Hospital (MGH). They had a long-running collaboration studying how the plant innate immune system resisted bacteria on the 1 hand and herbivorous insects on the other (Cui *et al.* 2005). They were using as models the mustard plant *Arabidopsis thaliana*, the bacterium *Pseudomonas syringae*, and the caterpillar *Trichoplusia ni*. While the plant and bacterium were genomically characterized and genetically tractable, the caterpillar was not.

Naomi mentioned that there were leaf-mining flies that attacked *A. thaliana*, and because *D. melanogaster* was also a fly, perhaps we could develop a leaf-mining fly as a model herbivore by taking advantage of its evolutionary proximity to an actual model organism (Mitchell-Olds 2001). This casual suggestion would define my research program for years to come. After another Google search, I discovered a 1902 paper (Chittenden *et al.* 1902) reporting the fly *Scaptomyza flaveola* (actually *S. flava*) from *A. thaliana*. Flies in the genus *Scaptomyza* are evolutionarily nested deep in the *Drosophila* lineage and most closely related to the Hawaiian *Drosophlia*. I collected wild mustards in the park where I walked my dogs in Belmont and soon had flies emerging from mines from my cages in the MCZ Laboratories. The anticipation I felt when placing those flies with *A. thaliana* plants I'd grown was palpable—would they accept this lab-grown host? The next morning, I found spindle-shaped punctures all over the leaves, and 3 weeks later, new flies emerged—completing their life cycle on that plant as in 1902. That moment remains one of the most satisfying of my career.

In 2008 Chuck from Saint John's emailed me because he would be visiting Boston where I was a postdoctoral fellow at Harvard and wondered if I wanted to meet up. Over lunch, I mentioned the irony that I'd become a geneticist despite earning a B in his course. He smiled and said something along the lines of, "Well, I

got a B when I took Genetics in college!" That quip transformed how I viewed my struggles—rather than seeing my B through the lens of shame, I realized his masterful teaching grew from his own early difficulty with the subject. The lesson was that sometimes our greatest eventual strengths emerge from areas of initial weakness.

For about 1 year In Fred's lab at MGH, I sat through his research group meetings and really didn't understand much of what they were discussing. The jargon was a thicket of confusion for me. I was a bit discouraged and thought maybe I'd made the wrong move to attempt to gain a new skill set in a new field at that stage of my career. Then right around the 1 year mark, things started to click, as if I was in a foreign language immersion program, and I was brought into the fold of experimental, molecular genetics. I loved it. We used *A. thaliana* mutants and reporter lines to investigate mechanisms of plant defenses against *S. flava* (Whiteman *et al.* 2011), and reciprocally, how the fly responded transcriptionally to the presence or absence of these defense pathways (Whiteman *et al.* 2012), allowing me to learn both molecular genetics and functional genomics. It was early days in genomics and without a genome sequence to map to, we built a patchy transcriptome assembly of *S. flava* using error-prone 454 pyrosequencing and Illumina short-read sequencing and filled in the gaps using the related species *Drosophila grimshawi* to create a sort of chimeric platform for mapping read from our transcriptional profiling of larvae that were feeding on *A. thaliana* plants with or without indolic and aliphatic glucosinolates. It was deeply satisfying to use the genetics and genomics approaches to isolate variables in the context of species interactions.

In my first faculty position at the University of Arizona, my focus shifted to understanding the evolutionary genomic and physiological basis of herbivory and host plant specialization on the fly side, a question that had fascinated me since those undergraduate days reading about coevolution. Using new *S. flava* genomic tools we built, my first PhD students Benjamin (Ben) Goldman-Huertas and Andrew (Andy) Gloss and I, along with collaborators like Jonathan Gershenzon from the Max Planck Institute for Chemical Ecology, discovered it evolutionarily lost odorant receptor orthologs required by *D. melanogaster* (and many other drosophilids) to detect yeast but had evolved highly efficient glutathione S-transferase detoxification enzymes against mustard oils (Gloss *et al.* 2014; Goldman-Huertas *et al.* 2015).

In parallel in Arizona, Anurag Agrawal from Cornell and Susanne Dobler from Hamburg proposed a collaboration that would leverage my emerging skills in *Drosophila* genetics. They had been studying how insects like the monarch butterfly evolved resistance to cardiac glycosides (with steroid backbones of tripartite hexagons) in milkweeds and how the very same sites in the protein targeted by those toxins had re-evolved the same amino acid substitutions over and over (Dobler *et al.* 2012). I led the writing of a *News and Views* about these findings and because it was 2012 (Whiteman and Mooney 2012), the same year CRISPR emerged as a gene editing tool, we suggested that somebody should use it to retrace the evolution of resistance mutations in the *ATPA* gene encoding the Na[+]/k[+])-ATPase alpha subunit targeted by cardiac glycosides in whole organisms like *D. melanogaster* in order to test the functional effects of these mutations across levels of organization. Gene duplications of *ATPA* in some of these "milkweed village" species that included resistant and susceptible ATPA copies encoded in the same genome were later discovered that suggested an even more complex layer of parallel evolution (Zhen *et al.* 2012). I didn't think that it would be my laboratory that would do the experiment we suggested, but after Anurag and Susanne approached me, I agreed. After failing for 3 years, postdoctoral fellows Simon "Niels" Groen and Marianthi Karageorgi successfully engineered mutant flies carrying various mutations in the *ATPA* gene after I moved to Berkeley (Karageorgi *et al.* 2019). The day we confirmed our triple mutant *D. melanogaster* line ("monarch flies") that recapitulated the monarch butterfly's ATPA genotype for those sites, after so many failed attempts, the lab erupted in celebration. Notably, Niels had been an undergraduate intern I advised from the Netherlands when I was at Harvard.

This project shed light on several genetic phenomena. One was how strongly evolution can be constrained by pleiotropy. We found that the adaptive walk taken by the monarch butterfly's *ATPA* gene was defined by a particular order of appearance of 3 amino acid changing substitutions in the binding pocket of the Na[+]/k[+])-ATPase alpha subunit (known as the sodium-potassium pump) at positions 111, 119, and 122. The most potent resistance mutation to a histidine at 122 may have fixed later in the adaptive walk taken by the monarch butterfly possibly due to its high costs (seizures), while an earlier mutation to a serine at position 119 that resulted in some resistance somehow also ameliorated the duration of the seizures (see also Taverner *et al.* 2019). Some of the double mutants even resulted in positive sign epistasis for resistance. In the end, experiments on whole animals allowed us to discern why this evolutionary path repeated itself so many times across different insect lineages that fed on milkweeds or foxgloves. This pattern extended to the warning coloration that evolved independently in many of these distantly related insects. How "*trans*-acting" dietary toxins from 1 species could drive adaptation across levels of organization in another still fills me with wonder. The other lesson was that although one could view the resistance phenotype as simply deriving from one large effect locus, this view would be superficial, because we discovered that complex intramolecular interactions drove coevolution among amino acid residues of Na[+]/K[+])-ATPase alpha subunit to give rise to the adaptive phenotypes we observed. Coevolution between plants and insects was in part driving coevolution between amino acids of a single protein too.

Over beers 1 evening, Niels and I discussed the next steps for the "milkweed village" project. I mentioned that Black-headed Grosbeaks (which, by coincidence, were visiting the bird feeder in my Oakland yard this week) that overwintered in the same Oyamel Fir forests as monarch butterflies in Mexico were physiologically resistant to cardiac glycosides. The late Lincoln Brower and Linda Fink had observed these birds consuming millions of monarchs whole (save the wings) (Fink and Brower 1981). Niels dug into this and discovered that someone had sequenced the grosbeak's genome, and remarkably, 2 of its 4 copies of *ATPA* encoded amino acid substitutions conferring cardiac glycoside resistance—depending on the copy, the same substitutions were the same ones found in the oleander aphid, a milkweed bug, the common crow butterfly, and rodents, which were all resistant to cardiac glycosides (Groen and Whiteman 2021). This illuminated how some toxins act as keystone molecules that can trigger cascades of convergent molecular and phenotypic evolution across 3 trophic levels, from plant to herbivore to predator.

With Jia Huang and his team in Hangzhou, along with PhD student Diler Haji in my lab, we also studied convergent evolution of resistance to plant-derived terpenoid neurotoxins targeting $GABA_A$ receptors across herbivorous insects, including aphids, and the insect predators that consumed those herbivores, the ladybird beetles (Guo *et al.* 2023). Using CRISPR-edited *D. melanogaster* with mutations in the *Resistance to dieldrin* (*Rdl*) gene

(Ffrench-Constant *et al.* 1993), we found terpenoid resistance substitutions (some ~300 million years old) were associated with increased species diversification rates in Lepidoptera—linking single amino acid substitutions to cladogenesis. However, there is no such thing as a free lunch as in the case of the ATPA study. Major heat shock sensitivities were a pleiotropic cost of resistance mutations. But as in the sodium-potassium pump, these costs were ameliorated to some degree through other mutations, in this case, gene duplication events enabling the insects to retain both an ancestral copy and 1 or more resistant copies of *Rdl* that may result in ion channels with different combinations of receptor subunits, both heat resistant and toxin resistant. Again, we found that over deep evolutionary time, plant toxins evolved in response to herbivory and in turn seem to have exerted a major impact on the evolution of their enemies *and* the enemies of their enemies as they co-adapted. The focal toxins we studied, picrotoxinin and thymol, feature beautiful structures with a hexagon at their core, echoing that recurring theme.

After moving to Berkeley, a surprising discovery emerged from the *S. flava* genome. We found a horizontally transferred gene from bacteria (or their phages) called *cdtB* in its genome (Verster *et al.* 2019). The *cdtB* gene was also independently transferred from bacteria (or their phages) into the *Drosophila ananassae* fruit fly species group and was in that case both found as a single copy and in 2 other copies where it was also fused to a portion of a gene called *aip56* (encoding trafficking subunit of another bacterial toxin). When former PhD student Kirsten Verster knocked out all copies of those genes, the flies were highly susceptible to parasitoids—at least those with which they hadn't tightly co-evolved (Verster *et al.* 2023). This suggested the genes provided a basal level of resistance to novel parasitoids, but the Red Queen forces hosts to run faster and faster to stay in the same place (Van Valen 1977).

What was more, we were able to phenocopy or rescue the phenotype in a new "host" fly (Tarnopol *et al.* 2024). When PhD candidate Rebecca Tarnopol integrated this fusion gene into *D. melanogaster*, the flies gained significant resistance to their own parasitoids that had probably not encountered the toxin before. Just as in the native host *D. ananassae*, the fusion toxin was produced by fat body cells of *D. melanogaster* where, thanks to antibodies raised by our Hungarian collaborators in István Andó's group, it then moved into the blood and attacked a single layer of epithelial cells surrounding the wasp embryo called the serosa (cells that can be hexagonal in shape). The flies borrowed a bacterial weapon and co-opted it against their own enemies—an example of how even animals have borrowed solutions from other branches of the tree of life to solve adaptive challenges. Here too, the same trait evolved multiple times independently in the context of co-evolution and by using the tools of *D. melanogaster*, we were able to illuminate how these traits were integrated across different levels of biological organization even to the upper trophic levels—as we did in the case of the cardiac glycosides and terpenoid neurotoxins.

In Book IV of Euclid's Elements, he showed how 1 could draw a perfect hexagon by starting with a circle. The story of my development as a geneticist has also come full circle. In November 2014, I interviewed for a faculty position at UC-Berkeley and was put up in the Women's Faculty Club, nestled among the coast redwoods along Strawberry Creek. The morning of my interview, I was just inside the building waiting for the search committee chair to arrive. A man with a wide smile and eyeglasses appeared—it was Monty Slatkin. As he extended his hand to shake mine, I was electrified. Improbably, it was the same Monty Slatkin who had edited the book on "Coevolution" that ignited my intellectual passion for understanding how species interactions could be a crucible for the evolution of new traits and new species in Darwin's "entangled bank." Only after arriving at Berkeley in 2016 would I find none other than *S. flava* flies attacking the watercress along the banks of that creek (and parasitoid wasps attacking those flies), now a stone's throw from my laboratory. In the same frame right now, water beetles are swimming among the aquatic plants, and above, male black-headed grosbeaks are setting up their breeding territories after arriving from Mexico, monarch butterflies float on by in search of milkweeds and nectar plants, ladybirds scramble in the bramble in search of aphids, and it is impossible not to hear the call of Red-Shouldered Hawks at the top of that trophic pyramid, congeners of the Galapagos hawk—well, not quite at the top—for we cannot forget the feather lice they carry.

Funding

The author declares no funding associated with this article.

Conflicts of interest

The author(s) declare no conflict of interest.

Literature cited

Chittenden FH, United States Division of Entomology, United States Department of Agriculture. 1902. Some Insects Injurious to Vegetable Crops: A Series of Articles Dealing with Insects of this Class. Washington, D.C.: U.S. Department of Agriculture, Division of Entomology.

Cui J, Bahrami AK, Pringle EG, Hernandez-Guzman G, Bender CL, Pierce NE, Ausubel FM. 2005. *Pseudomonas syringae* manipulates systemic plant defenses against pathogens and herbivores. Proc Natl Acad Sci U S A. 102(5):1791–1796. doi:10.1073/pnas.0409450102.

Dobler S, Dalla S, Wagschal V, Agrawal AA. 2012. Community-wide convergent evolution in insect adaptation to toxic cardenolides by substitutions in the Na,K-ATPase. Proc Natl Acad Sci U S A. 109(32):13040–13045. doi:10.1073/pnas.1202111109.

Ffrench-Constant RH, Rocheleau TA, Steichen JC, Chalmers AE. 1993. A point mutation in a Drosophila GABA receptor confers insecticide resistance. Nature. 363(6428):449–451. doi:10.1038/363449a0.

Fink LS, Brower LP. 1981. Birds can overcome the cardenolide defence of monarch butterflies in Mexico. Nature. 291(5810):67–70. doi:10.1038/291067a0.

Futuyma DJ, Slatkin M. 1983, editors. Coevolution. Sunderland (MA): Sinauer Associates. p. 1–555.

Gloss AD, Vassão DG, Hailey AL, Nelson Dittrich AC, Schramm K, Reichelt M, Rast TJ, Weichsel A, Cravens MG, Gershenzon J, *et al.* 2014. Evolution in an ancient detoxification pathway is coupled with a transition to herbivory in the Drosophilidae. Mol Biol Evol. 31(9):2441–2456. doi:10.1093/molbev/msu201.

Goldman-Huertas B, Mitchell RF, Lapoint RT, Faucher CP, Hildebrand JG, Whiteman NK. 2015. Evolution of herbivory in Drosophilidae linked to loss of behaviors, antennal responses, odorant receptors, and ancestral diet. Proc Natl Acad Sci U S A. 112(10):3026–3031. doi:10.1073/pnas.1424656112.

Groen SC, Whiteman NK. 2021. Convergent evolution of cardiac-glycoside resistance in predators and parasites of milkweed herbivores. Curr Biol. 31(22):R1465–R1466. doi:10.1016/j.cub.2021.10.025.

Guo L, Qiao X, Haji D, Zhou T, Liu Z, Whiteman NK, Huang J. 2023. Convergent resistance to GABA receptor neurotoxins through plant-insect coevolution. Nat Ecol Evol. 7(9):1444–1456. doi:10.1038/s41559-023-02127-4.

Karageorgi M, Groen SC, Sumbul F, Pelaez JN, Verster KI, Aguilar JM, Hastings AP, Bernstein SL, Matsunaga T, Astourian M, *et al.* 2019. Genome editing retraces the evolution of toxin resistance in the monarch butterfly. Nature. 574(7778):409–412. doi:10.1038/s41586-019-1610-8.

Koop JAH, DeMatteo KE, Parker PG, Whiteman NK. 2014. Birds are islands for parasites. Biol Lett. 10(8):20140255. doi:10.1098/rsbl.2014.0255.

Mitchell-Olds T. 2001. Arabidopsis thaliana and its wild relatives: a model system for ecology and evolution. Trends Ecol Evol. 16(12):693–700. doi:10.1016/S0169-5347(01)02291-1.

Tarnopol RL, Tamsil JA, Cinege G, Ha JH, Verster KI, Ábrahám E, Magyar LB, Kim BY, Bernstein SL, Lipinszki Z, *et al.* 2024. Experimental horizontal transfer of phage-derived genes to Drosophila confers innate immunity to parasitoids. Curr Biol. 35(3):514–529.e7. doi:10.1016/j.cub.2024.11.071.

Taverner AM, Yang L, Barile ZJ, Lin B, Peng J, Pinharanda AP, Rao AS, Roland BP, Talsma AD, Wei D, *et al.* 2019. Adaptive substitutions underlying cardiac glycoside insensitivity in insects exhibit epistasis in vivo. Elife. 8:e48224. doi:10.7554/eLife.48224.

Van Valen L. 1977. The red queen. Am Nat 111(980):809–810. doi:10.1086/283213.

Verster KI, Cinege G, Lipinszki Z, Magyar LB, Kurucz É, Tarnopol RL, Ábrahám E, Darula Z, Karageorgi M, Tamsil JA, *et al.* 2023. Evolution of insect innate immunity through domestication of bacterial toxins. Proc Natl Acad Sci U S A. 120(16):e2218334120. doi:10.1073/pnas.2218334120.

Verster KI, Wisecaver JH, Karageorgi M, Duncan RP, Gloss AD, Armstrong EE, Price DK, Menon AR, Ali ZM, Whiteman NK. 2019. Horizontal transfer of bacterial *cytolethal distending toxin B* genes to insects. Mol Biol Evol. 36(10):2105–2110. doi:10.1093/molbev/msz146.

Whiteman NK, Gloss AD, Sackton TB, Groen SC, Humphrey PT, Lapoint RT, Sønderby IE, Halkier BA, Kocks C, Ausubel FM, *et al.* 2012. Genes involved in the evolution of herbivory by a leaf-mining, drosophilid fly. Genome Biol Evol. 4(9):900–916. doi:10.1093/gbe/evs063.

Whiteman NK, Groen SC, Chevasco D, Bear A, Beckwith N, Gregory TR, Denoux C, Mammarella N, Ausubel FM, Pierce NE. 2011. Mining the plant-herbivore interface with a leafmining *Drosophila* of *Arabidopsis*. Mol Ecol. 20(5):995–1014. doi:10.1111/j.1365-294X.2010.04901.x.

Whiteman NK, Kimball RT, Parker PG. 2007. Co-phylogeography and comparative population genetics of the threatened Galápagos hawk and three ectoparasite species: ecology shapes population histories within parasite communities. Molecular Ecology. 16(22):4759–4773. doi:10.1111/j.1365-294X.2007.03512.x.

Whiteman NK, Matson KD, Bollmer JL, Parker PG. 2006. Disease ecology in the Galápagos Hawk (*Buteo galapagoensis*): host genetic diversity, parasite load and natural antibodies. Proc Biol Sci. 273(1588):797–804. doi:10.1098/rspb.2005.3396.

Whiteman NK, Mooney KA. 2012. Evolutionary biology: insects converge on resistance. Nature. 489(7416):376–377. doi:10.1038/489376a.

Zhen Y, Aardema ML, Medina EM, Schumer M, Andolfatto P. 2012. Parallel molecular evolution in an herbivore community. Science. 337(6102):1634–1637. doi:10.1126/science.1226630.