but enhanced by GTP-γ-S and AIF₂ (Fig. 2, C and D), indicating that the cytoplasmic domains of β₁ and β₁ can directly interact with Gt₁ and that GTP enhances the interaction. The Gt₁β₁ interaction was enhanced in platelets adherent to fibrinogen, and by thrombin, which stimulates GTP binding to Gt₁ via GPCR (Fig. 2E). Hence, the interaction is regulated by both integrin occupancy and GPCR signaling.

To map the β₁ binding site in Gt₁, we incubated cell lysates containing Flag-tagged wild type or truncation mutants of Gt₁ (Fig. S5) with GST-β₁CD beads. GST-β₁CD-associated wild-type Gt₁ and the Gt₁ to 212 fragment containing α-hecoloric region and switch region I (SRI) but not with the Gt₁ 212 fragment containing residues 1 to 196 lacking SRI (Fig. 2F). Thus, SRI appears to be critical for β₁ binding. To further determine the importance of SRI, Gt₁β₁ CD binding was assayed in the presence of a myristoylated synthetic peptide, Myr-LAARPFK1GHEY (mSRII), corresponding to the β₁CD sequence of Gt₁ (179 to 209) (21, 22). The mSRII peptide, but not a myristoylated scrambled peptide, inhibited Gt₁ binding to β₁ (Fig. 2G), indicating that mSRII is an effective inhibitor of β₁-Gt₁ interaction. Therefore, we further examined whether mSRII might inhibit integrin signaling. Treatment of platelets with mSRII inhibited integrin-dependent phosphorylation of ε-Src Tyr152 and accelerated RhoA activation (Fig. 3A). The effect of mSRII is unlikely to result from its inhibitory effect on the binding of RhoGEFs to Gt₁ because Gt₁ binding to RhoGEFs stimulates RhoA activation, which should be inhibited rather than promoted by mSRII (22). Thus, these data suggest that β₁-Gt₁ interaction mediates activation of ε-Src and inhibition of RhoA. Furthermore, mSRII inhibited integrin-mediated platelet spreading (Fig. 3B), and this inhibitory effect was reversed by C3 toxin (which catalyzes the ADP ribosylation of RhoA) or Y27632, confirming the importance of Gt₁-dependent inhibition of RhoA in platelet spreading. Thrombin promotes platelet spreading, which requires cdc42/Rac pathways (23). However, thrombin-promoted platelet spreading was also abolished by mSRII (Fig. 3B), indicating the importance of Gt₁β₁ interaction. Thus, Gt₁β₁-integrin interaction appears to be a mechanism that mediates integrin signaling to ε-Src and RhoA, thus regulating cell spreading.

To further determine whether Gt₁ mediates inhibition of integrin-induced RhoA-dependent contractile signaling, we investigated the effects of mSRII and depletion of Gt₁ in platelet-dependent clot retraction (shinking and consolidation of a blood clot requires integrin-dependent retraction of platelets from within) (7, 8). Clot retraction was accelerated by mSRII and depletion of Gt₁ and Gt₁β₁ (Fig. 4, A and B, and fig. S6), indicating that Gt₁ negatively regulates RhoA-dependent platelet retraction and coordinates cell spreading and retraction. The coordinated cell spreading-retraction process is also important in wound healing, cell migration, and proliferation (24).

The function of Gt₁ in mediating the integrin-dependent inhibition of RhoA contrasts with the traditional role of Gt₁, which is to mediate GPCR-inducible activation of RhoA. However, GPCR-mediated activation of RhoA is transient, peaking at 1 min after exposure of platelets to thrombin, indicating the presence of a negative regulatory signal (Fig. 4, D and F). Furthermore, thrombin-stimulated activation of RhoA occurs during platelet shape change before substantial ligand binding to integrins (Fig. 4, C, D, and F). In contrast, after thrombin stimulation, β₁ binding to Gt₁ was diminished at 1 min when Gt₁-dependent activation of RhoA occurs, but increased after the occurrence of integrin-dependent platelet aggregation (Fig. 4, E and F). Thrombin-stimulated binding of Gt₁ to α₁β₁ and simultaneous RhoA inhibition both require ligand occupancy of α₁β₁ and are inhibited by the integrin inhibitor Arg-Gly-Asp-Ser (RGDS) (Fig. 4, D to F). Thus, our study demonstrates not only a function of integrin α₁β₁ as a noncanonical Gt₁-coupled receptor but also a new concept of Gt₁-dependent dynamic regulation of RhoA, in which Gt₁ mediates initial GPCR-induced RhoA activation and subsequent integrin-dependent RhoA inhibition (Fig. 4C). These findings are important for our understanding of how cells spread, retract, migrate, and proliferate, which is fundamental to development, cancer, immunity, wound healing, hemostasis, and thrombosis.

**References and Notes**


**Functional and Evolutionary Insights from the Genomes of Three Parasitoid Nasonia Species**

The Nasonia Genome Working Group†

We report here genome sequences and comparative analyses of three closely related parasitoid wasps: *Nasonia vitripennis*, *N. giraulti*, and *N. longicornis*. Parasitoids are important regulators of arthropod populations, including major agricultural pests and disease vectors, and *Nasonia* is an emerging genetic model, particularly for evolutionary and developmental genetics. Key findings include the identification of a functional DNA methyltransferase tool kit: hemmopeptin-specific genes including diverse venom; lateral gene transfers among Pox viruses, *Wobachia*, and *Nasonia*; and the rapid evolution of genes involved in nuclear-mitochondrial interactions that are implicated in speciation. Newly developed genome resources advance *Nasonia* for genetic research, accelerate mapping and cloning of quantitative trait loci, and will ultimately provide tools and knowledge for further increasing the utility of parasitoids as pest insect-control agents.

Parasitoid wasps are insects whose larvae parasitize various life stages of other arthropods (for example, insects, ticks, and mites). Female wasps sting, inject venom, and lay eggs on or in the host, where the developing offspring consume and eventually kill it. Parasitoids
are widely used in the biological control of insect pests, and they are very diverse, with estimates of over 600,000 species (1, 2). *Nasonia* is the second genus of Hymenoptera to have whole-genome sequencing, after *Apis mellifera* (Fig. 1), and *Nasonia* comprises four closely related parasitoid species: *N. vitripennis*, *N. giraulti*, *N. longicornis*, and *N. oneida* (3, 4). *Nasonia* are genetically tractable organisms with short generation time (~2 weeks), large family size, ease of laboratory rearing, and cross-fertile species. Like other hymenopterans, haploid males develop from unfertilized eggs, and diploid females develop from fertilized eggs. Cross-fertile species facilitate the mapping and cloning of genes that are involved in species differences. Haplod genetics assist efficient genotyping, mutational screening (5), and evaluation of gene interactions (epistasis) without the added complexity of genetic dominance. As a result, *Nasonia* are now emerging as genetic model organisms, particularly for complex trait analysis, developmental genetics, and evolutionary genetics (4).

We sequenced, assembled, annotated, and analyzed the genome of *N. vitripennis* from sixfold Sanger sequence genome coverage by using a highly inbred line of *N. vitripennis* (6). The draft genome assembly comprises 26,605 contigs totaling 11.5 Gb, amounting to 90.6% of the estimated genome size. These contigs were placed with mate-pair information into 6,181 scaffolds (total size 295 Mb, N50 = 709 kb).

We assessed the *N. vitripennis* assembly for completeness and accuracy by comparing it with 19 finished bacterial artificial chromosome (BAC) sequences and 18,000 expressed sequence tags (ESTs). The genome assembly contained 98% of the BACs and 97% of the ESTs, with an error rate of 5.9 × 10⁻⁴. Thus the assembly is a high-quality representation of both genomic and transcribed *N. vitripennis* sequences.

Highly inbred lines of the two sibling species *N. giraulti* and *N. longicornis* (Fig. 1B) were sequenced with onefold Sanger and 12-fold, 45-base pair (bp) Illumina genome coverage. Assembled by alignment to the *N. vitripennis* reference using stringent criteria (6), these reads cover 62% and 62.6% of the *N. vitripennis* assembly, and 84.7% and 86.3% of protein coding regions, respectively. These were used for genome comparisons and provided resources (for example, single nucleotide polymorphisms [SNPs] and microsatellites) for scaffold, gene, and quantitative trait loci (QTL) mapping. Sequence error rates for the *N. giraulti* alignment are estimated to be 3.8 × 10⁻⁵ for the entire alignment and 1.47 × 10⁻⁶ for coding sequences on the basis of comparison to three finished *N. giraulti* BACs (6). Sequences of 25 coding genes in both species perfectly matched their respective aligned sequences.

Normally, the intracellular bacteria *Wolbachia* prevent the formation of interspecies hybrids; however, antibiotically cured strains are cross-fertile (7). Hybrid crosses (Fig. 1C) (6) were used to map scaffolds and visible mutations onto the five chromosomes of *Nasonia* (Fig. 2). Several interspecies QTL have already been mapped using genetic/genomic resources, including wing size (8, 9), host preference (10), female mate preference (11), and in this study, sex-ratio control and male courtship (6). Linkage analysis has revealed that the genome-wide recombination rate in *Nasonia* is 1.4 to 1.5 centimorgans (cM)/Mb, which is lower than that of honeybees (12, 13), and shows a 100-fold difference in rate between high- and low-recombination regions of the genome (Fig. 2) (6).

An official gene set (OGS v1.1) was generated from comparisons to *A. mellifera*, Tribolium castaneum, Drosophila melanogaster, Pediculus humanus, Daphnia pulex, and Homo sapiens [details are given in (6)]. Overall, *Nasonia* encodes a typical insect gene repertoire (Fig. 3) (6), of which 60% of genes have a human ortholog, 18% are arthropod-specific, and 2.4% appear to be hymenoptera-specific, showing high conservation between *Nasonia* and *Apis* and low conservation or absence in other taxa. An additional 1.2% are either *Nasonia*-specific or without clear orthology. Many (63%) single-copy orthologs shared between *Nasonia* and *Apis* occur in microsynteny blocks, which is similar to the amount of microsynteny blocks in *Aedes aegyptii/Aedes albopictus* and *H. sapiens/Gallus gallus* (14). Four hundred and forty-five orthologs between *Nasonia* and *Apis* and humans lack a candidate homolog in D. melanogaster (Table S1), including the human transcription factors E2F7 and E2F8, which are involved in cell-cycle regulation. Further refinement of the gene set resulted in OGS v1.2 (15), which totals 17,279 genes, of which 74% have tissue microarray or EST support (6).

*Nasonia* is abundant in transposable elements (TEs) and other repetitive DNA (Table S2 and Fig. S1). This contrasts with a paucity of TEs in *A. mellifera* (16). TE diversity in *Nasonia* is 30% higher (2.9 TE types/Mb) than the next most diverse insect (Bombyx mori, 2.1 TE types/Mb), and is 10-fold higher than the average dipteran (6, 17). *Nasonia* also contains an unusual abundance of nuclear-mitochondrial insertions and a higher density of microsatellites (19 kb/Mb) than most other arthropod species (18, 19), suggesting that the accumulation of repetitive DNA is a feature of these insects.

The *Nasonia* genome encodes a full DNA methylation tool kit, including all three DNA cytosine-5'-methyltransferase (Dnmt) types (Fig. 1A).

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**Fig. 1.** Phylogenetic relationships of *Nasonia* and the DNA methylation tool kit. (A) *Nasonia* relationships to other sequenced genomes (6). Right: DNA methylation subfamilies (Dnmt1, Dnmt2, Dnmt3) in these taxa. (B) Relationships among the three sequenced *Nasonia* genomes. (C) Crossing scheme used for mapping scaffolds on the *Nasonia* chromosomes and for studies of nuclear-cytoplasmic incompatibility.
In vertebrates, Dnmt3 establishes DNA methylation patterns, Dnmt1 maintains these patterns, and Dnmt2 is involved in tRNA methylation (20). The Nasonia genome encodes three Dnmt1 genes, one Dnmt2, and one Dnmt3, in contrast with D. melanogaster, which has only Dnmt2. The presence of all three subfamilies in both Nasonia and Apis (Fig. 1) raises the question of whether methylation has similar regulatory functions in Hymenoptera as it does in vertebrates. DNA methylation is important in Apis caste development (21) and is suggested for Nasonia sex determination (22). Coding exons of both Nasonia and Apis show bimodal distributions in observed/expected CpG (Fig. S2) (6, 23), which is consistent with mutational biases due to DNA methylation of hyper- and hypomethylated genes. We confirmed methylated CpG dinucleotides in five examined N. vitripennis genes by bisulfite sequencing (Fig. S3). These results suggest that epigenetic modifications by DNA methylation may be important in Hymenoptera. Nasonia also has the largest number of ankyrin (ANK) repeat-containing proteins (over 200) so far found in any insect (table S3) (6), suggesting a regulatory importance through protein-protein interactions (24).

Systemic RNA interference (RNAi) in Nasonia allows for gene expression knockdowns (4, 25). The Nasonia genome encodes homologs for the majority of genes implicated in small RNA processes (table S4). However, as in Tribolium and Apis, Nasonia lacks an RNA-dependent RNA polymerase (RdRp) ortholog, indicating a different systemic RNAi mechanism than in Caenorhabditis. Using various computational approaches (6), we identified 52 putative micro RNAs (miRNAs) with homologies to known miRNAs (26), nine that were previously unknown, and 11 additional Hymenoptera-specific miRNAs (table S5). Small-RNA library sequencing confirmed 39 predicted and identified 59 additional miRNAs (table S6).

Nasonia shares a long germ- and larval stage embryonic development with Drosophila, but exhibits significant genetic mechanisms involved (5, 27, 28) (see Fig. S4). All major components of the dorso-ventral patterning system are present, with many Nasonia-specific gene duplications in the Toll pathway. Orthologs of vertebrate genes absent from Drosophila include the transforming growth factor-β (TGFβ) ligands ADMF and invesostatin, and the bone morphogenesis protein (BMP) inhibitors Bambi and Dan, but their functions in Nasonia are not yet known. A. mellifera shows an expansion of the yellow/major royal jelly (yellow/RRNP) genes that are linked to caste formation and sociability (29). Nasonia has the largest number of yellow/RRNP genes so far found in any insect, including an independent amplification of RRNP-like proteins (fig. S5) (6, 29). Although their function in Nasonia is unknown, these genes are expressed broadly in different tissues and life stages (table S7). The insect sex peptide/receptor system, which causes female remating refractoriness (30), is highly conserved in insects but is absent in Nasonia and Apis (table S8) (6). Instead, Nasonia males inhibit female remating behaviorally with a special post-copulatory display (31). Additional features analyzed (6) include those related to sex determination (fig. S6), pathogens and immunity (fig. S7), neuropeptides (tables S9 and S10), cuticular proteins (table S11), xenobiotics (fig. S8), and diapause (table S12).

We investigated genome microevolution, including rapidly evolving genes that are potentially involved in species differences and speciation, by using the genomes of the three closely related Nasonia species. Synonymous divergence between N. vitripennis and its sibling species N. giraulti and N. longicornis is 0.031 ± 0.0002 SE and 0.030 ± 0.0002 SE, respectively, and between N. giraulti and N. longicornis is 0.014 ± 0.0001 SE (6), which is comparable to those among Drosophila sibling species (32). We compared the ratio of synonymous-to-nonsynonymous substitutions (dN/dS) between Nasonia species pairs with respect to gene ontology (GO) term categories, using genes with high-quality alignments and 1:1 orthologs between Nasonia and Drosophila. Nuclear genes that interact with mitochondria revealed significantly elevated dN/dS [by comparison of dN/dS distributions for each GO term to resampled distributions, see (6) and table S13], specifically those encoding mitochondrial ribosomes (P < 0.003 for all species pairs) and oxidative phosphorylation complex I (P < 0.03 for N. vitripennis/N. giraulti and N. vitripennis/ N. longicornis) and complex V (P < 0.04 for all species pairs). This finding is consistent with the rapid evolutionary rate of Nasonia mitochondria (33) and studies implicating nuclear-mitochondrial incompatibilities in F2 hybrid breakdown (7, 34). For example, reciprocal crosses between N. giraulti × N. vitripennis have identical F1 nuclear genotypes, but their mitochondrial haplotypes differ. Yet, microarray hybridization (Fig. 2) (6) of DNA from pooled surviving adult F2 haploid males shows distortion in the recovery of particular regions of the genome, which is dependent upon their mitochondrial haplotype (giraulti versus vitripennis). Because hybrid mortality is post-embryonic (7) and embryo ratios are Mendelian (33), these distortions reflect larval to adult mortality. In particular, F2 males with N. vitripennis alleles on the left arm of chro-

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Fig. 2. A high-resolution recombination map of the five Nasonia chromosomes is shown (6), with estimated gene density and locations of visible markers, landmark genes, and QTL. The hybridization percentage to N. vitripennis alleles is shown among surviving adult N. vitripennis × N. giraulti F2 hybrid males with either N. vitripennis (green curve) or N. giraulti (orange curve) mitochondria. Dots specify genome regions with significant differences in the hybridization ratio between the reciprocal crosses (P < 0.01).
mosome 5 and N. girardi mitochondria suffer nearly 100% mortality (Fig. 2). This region contains three genes encoding mitochondrial interacting proteins, aubD, amgK, and naph-thiolquinone oxidoreductase (Fig. 2). Coevolution of nuclear and mitochondrial genomes can accelerate evolution (34, 35), and these findings indicate that such interactions contribute to reproductive incompatibility and speciation in Nasonia.

Sequences of 25 gene regions from multiple strains for the three Nasonia species (6) show low levels of intraspecific variation (table S14) with synonymous site variation ranging from 0.0005 in N. girardi to 0.0026 in N. vitripennis, which are much lower than in Drosophila species and more akin to levels observed in humans (6). This low nuclear variation could be explained by founder events, purging of deleterious mutations in haploid males, or inbreeding.

Recent lateral gene transfers from the bacterial endosymbiont Wolbachia into the genomes of Nasonia and other arthropods have been identified (37). Detecting ancient lateral transfers is more problematic. By examining protein domain arrangements in Nasonia relative to other organisms, we uncovered an ancient lateral gene transfer involving Pox viruses, Wolbachia, and Nasonia. Thirteen ANK repeat-bearing proteins encoded in the N. vitripennis genome also contain C-terminal PRANC (Pox proteins repeats of ankyrin-C terminal) domains. This domain was previously only described in Pox viruses, where it is associated with ANK repeats and inhibits the nuclear factor xB (NF-xB) pathway in mammalian hosts (38). A computational screen revealed ANK-PRANC-bearing genes in some Wolbachia and a related Rickettsiales (Fig. 4). Screening additional Wolbachia confirmed the presence of ANK-PRANC genes in diverse Wolbachia. The Nasonia PRANC genes are clearly integrated in the genome (6) and are expressed in different life stages (table S15). Phylogenetic analysis of the PRANC-domain sequences suggests that the Nasonia lineage acquired one or more of these proteins from Wolbachia, with subsequent amplification and divergence (Fig. 4). Such lateral gene transfers between bacteria and animals could be an important source of evolutionary innovation (37).

Nasonia is a carnivore, feeding on an amino acid–rich diet both as larva and adult (4). Mapping of Nasonia genes onto metabolic pathways (39) revealed loss or rearrangement in some amino acid metabolic pathways, including tryptophan and aminosugar metabolism (fig. S9) (6). The changes may reflect its specialized carnivorous diet and can inform efforts to produce artificial diets for more economical parasitoid rearing.

The venom of parasitoids, injected into a host before oviposition, serves to condition the host for successful development of wasp progeny (1, 2). Unlike the defensive Apis venom that inflicts pain and damage, parasitoid venoms have diverse physiological effects on hosts, including developmental arrest, alteration in growth and physiology, suppression of immune responses, induction of paralysis, oncosis, or apoptosis, and alteration of host behavior (40). The identification of Nasonia
genes with venom features and protocomic analyses of venom reservoir tissues have uncovered a rich assemblage of 79 candidate venom proteins (table S16) (41). Some Nasonia venom reservoir proteins belong to previously known insect venom families such as serine proteases; however, nearly half were not related to any known insect venoms. As expected, many of these venom candidates show highly elevated expression in the female reproductive tract, which includes the venom glands and reservoirs. Venom genes also showed significantly higher D/V/S ratios between N. vitripennis and N. giraldii than nonvenom genes did (Mann-Whitney U test, P < 2 × 10−5), suggesting that changes in host-seen molecules may be accompanied by rapid evolution of venom proteins. The large venom protein set found in Nasonia with diverse physiological effects (40) and abundance of parasitoid species (1, 2) suggests that parasitoids may contain a rich venom pharmacopeia of potential new drugs. N. vitripennis is a generalist parasitoid with a wide host utilization of many fly species, whereas the other Nasonia species are specialists (4, 10).

Genetic tools, a major host preference locus has been mapped to a region of 2 cm (10). Other genes in the Nasonia genome that are potentially involved in host finding include odorant binding proteins (table S1) and chemoreceptors (42), which show expansions, contractions, and pseudogenization, indicative of rapid turnover.

A suite of genetic tools and resources is available or under development for the Nasonia system (4, 6, 11, 28), and the genome resources presented here can be used for fine-scale mapping (6, 9–11) and positional cloning (8) of OTUs. By combining haploid genetics, ease of rearing, short generation time, systemic RNAi, interfertility species, and new genome resources for three species, Nasonia shows promise as a genetic model system for evolutionary and developmental genetics. Genome resources described here and our resulting enhanced understanding of parasitoid biology will also open avenues for improving parasitoid utility in biological control of pests of agricultural and medical importance.

References and Notes
6. Materials and methods and supplementary text are available as supporting material on Science Online.
Zebrafish Behavioral Profiling Links Drugs to Biological Targets and Rest/Wake Regulation

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A major obstacle for the discovery of psychoactive drugs is the inability to predict how small molecules will alter complex behaviors. We report the development and application of a high-throughput, quantitative screen for drugs that alter the behavior of larval zebrafish. We found that the multidimensional nature of observed phenotypes enabled the hierarchical clustering of molecules according to shared behaviors. Behavioral profiling revealed conserved functions of psychoactive molecules and predicted the mechanisms of action of poorly characterized compounds. In addition, behavioral profiling implicated new factors such as either a go-go–related gene (erg) potassium channels and immunomodulators in the control of rest and locomotor activity. These results demonstrate the power of high-throughput behavioral profiling in zebrafish to discover and characterize psychoactive drugs and to dissect the pharmacology of complex behaviors.

Most current drug discovery efforts focus on simple in vitro screening assays. Although such screens can be successful, they cannot recreate the complex network interactions of whole organisms. These limitations are particularly acute for psychoactive drugs because brain activity cannot be modeled in vitro (1–3). Motivated by recent small-molecule screens that probed zebrafish developmental processes (4–7), we developed a whole organism, high-throughput screen for small molecules that alter larval zebrafish locomotor behavior. We used an automated rest/wake behavioral assay (3, 5) to monitor the activity of larvae exposed to small molecules at 10 to 30 μM for 3 days (Fig. 1A) (3). Multiple behavioral parameters were measured, including the number and duration of rest bouts, rest latency, and waking activity (i.e., activity not including time spent at rest) (Fig. 1B) (3). We screened 5648 compounds representing 3968 unique structures and 1680 duplicates and recorded more than 60,000 behavioral profiles. Of these, 547 compounds representing 463 unique structures significantly altered behavior relative to controls, according to a stringent statistical cutoff (3).

Because the alterations in behavior were multidimensional and quantitative, we assigned a behavioral fingerprint to each compound and applied clustering algorithms to organize molecules according to their fingerprints (Fig. 2A and figs. S1 to S3). This analysis organized the data set broadly into arousing and sedating compounds and identified multiple clusters corresponding to specific phenotypes (Fig. 2, B to F; Fig. 3, A to C; Fig. 4, B and C; and figs. S1 to S4). Clustering allowed us to address three questions: (i) Do structural, functional, and behavioral profiles overlap? (ii) Does the data set predict links between known and unknown small molecules and their mechanisms of action? (iii) Does the data set identify unexpected

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Reports: “Functional and evolutionary insights from the genomes of three parasitoid Nasonia species” by J. H. Werren et al. (15 January, p. 343). The author list on page 343 should read as follows: John H. Werren, Stephen Richards, Christopher A. Desjardins, Oliver Niehuis, Jürgen Gadau, John K. Colbourne, and The Nasonia Genome Working Group. The list has been corrected on the online HTML page.