COMPARATIVE GENOMICS

Rapid genome shrinkage in a self-fertile nematode reveals sperm competition proteins


To reveal impacts of sexual mode on genome content, we compared chromosome-scale assemblies of the outcrossing nematode Caenorhabditis nigoni to its self-fertile sibling species, C. briggsae. C. nigoni's genome resembles that of outcrossing relatives but encodes 31% more protein-coding genes than C. briggsae. C. nigoni genes lacking C. briggsae orthologs were disproportionately small and male-biased in expression. These include the male secreted short (mss) gene family, which encodes sperm surface glycoproteins conserved only in outcrossing species. Sperm from mss-null males of outcrossing C. remanei failed to compete with wild-type sperm, despite normal fertility in noncompetitive mating. Restoring mss to C. briggsae males was sufficient to enhance sperm competitiveness. Thus, sex has a pervasive influence on genome content that can be used to identify sperm competition factors.

Sex between individuals is nearly ubiquitous in eukaryotic life (I). However, in multicellular organisms, the costs of sex and scarcity of mates sometimes favor the evolution of uniparental reproduction through asexual parthenogenesis or self-fertilization (2). Such changes in sexual reproduction have consequences for both sexual traits and genome content. Comparative genomics using closely related species with different modes of sexual reproduction can reveal sex-related factors that might otherwise remain cryptic. In the nematode species C. elegans, C. briggsae, and C. tropicalis, animals with two X chromosomes that would normally be female have evolved into self-fertilizing hermaphrodites (Fig. 1A) (3). Nearly all progeny of these selfing XX hermaphrodites are themselves XX. Rare haplo-X (XO) male progeny experience weaker sexual selection than males from outcrossing species, exhibit atrophy of traits required for efficient mating (4–7), and are hypersensitive to pheromone-induced mortality (8). Sexually antagonistic sperm-female interactions have also been relaxed in self-fertile Caenorhabditis (9).

Self-fertile Caenorhabditis have smaller genomes and transcriptomes than outcrossing Caenorhabditis (10, 11), as also observed in the selfing plant Arabidopsis thaliana (12). However, comparisons of self-fertilizing to outcrossing Caenorhabditis have involved species as divergent at the nucleotide level as humans are from mice (10, 13), so it remains unclear how quickly genomic shrinkage occurs. We hypothesized a direct link between the degradation of sexual traits and genome contraction in selfing species. Here, we describe genomic resources and functional experiments that confirm its existence.

Comparison of C. nigoni and C. briggsae genomes

Of the ~50 known Caenorhabditis species, the most closely related pair with different sexual modes are the outcrossing C. nigoni and the selfing C. briggsae (14–16). They remain partially interfertile, yet they have numerous genetic and reproductive incompatibilities (9, 13, 17–19). To compare their genomes, we assembled the C. nigoni genome from 20-kb Pacific Biosciences (PacBio) and Illumina short-read libraries (table S1) (20). The final C. nigoni chromosome-scale genome assembly totaled 129 Mb with a size-weighted median (NS50) contig length of 3.3 Mb; it was estimated as 99.6% complete (21). The genome was 19% larger than that of C. briggsae (108 Mb) but was similar in size to genomes of the more distantly related outcrossing species C. remanei, C. sinica, C. brenneri, and C. japonica, which range from 131 to 135 Mb (Fig. 1A) (10). Therefore, larger genome sizes were probably the ancestral condition, and genomic shrinkage occurred in the C. briggsae lineage after it diverged from C. nigoni. More than 90% (118 Mb) of the assembly can be aligned to the chromosomes of C. briggsae without large translocations or inversions, despite megabase-size contigs (fig. S1). Thus, the two genomes are essentially colinear but differ in many small species-specific segments. C. nigoni's six chromosomes are 6.6 to 16.6% larger than their C. briggsae homologs (table S2).

We used whole-genome alignment to identify species-specific genomic segments (20). In C. nigoni, 47.7 Mb (36.9%) did not align with C. briggsae, and C. briggsae had 27.7 Mb (25.6%) of 108.4 Mb) that did not align with C. nigoni. This 20.0-Mb difference accounted for 95% of the difference in genome sizes. Nonalignable genomic regions were concentrated on the distal arms of all six holocentric chromosomes, where small inversions and repetitive sequences were abundant and gene densities were low (Fig. 1B). Both regions were mostly small (median ~500 base pairs; Fig. 2A), but larger (1 to 65 kb) insertions or deletions accounted for 17 Mb (81%) of the genome size difference (Fig. 2B). In both assemblies, nonalignable sequences were most common in intergenic regions and introns (fig. S2). C. nigoni harbored 5.4 Mb more species-specific protein-coding sequences than C. briggsae, consistent with a net loss of genes in C. briggsae (see below). For orthologous genes in both species, exon lengths were highly correlated (Fig. 2C and table S3). In contrast, ortholog intron content was weakly correlated and was significantly larger in C. briggsae. Because both genomes had similar repetitive DNA fractions (C. nigoni 27% versus C. briggsae 25%), disproportionate loss of repetitive sequences (seen in plants) did not contribute to different genome sizes (table S1) (10, 12, 22).

Impact of genome shrinkage on C. briggsae gene content

We predicted 29,167 protein-coding genes for C. nigoni (table S4), with 88.9% (25,929) being expressed in adults [≥0.1 transcripts per million (TPM)]. By equivalent methods, we predicted 22,313 genes in C. briggsae (20), 23.5% less than C. nigoni. The published gene annotations for C. briggsae (23) were even fewer (21,814 genes). This 6854-gene difference could have several causes, including gene family contraction and loss of sequence classes in C. briggsae, as well as C. nigoni–biased gain of novel sequences. We compared genes of C. briggsae and C. nigoni to genes of the outgroups C. remanei, C. brenneri, and C. elegans (20). In C. nigoni, 24,341 genes (83.5%) were orthologous to 21,124 C. briggsae genes, reflecting larger multigene families in C. nigoni versus C. briggsae (Fig. 3A and table S4) (24). Another 2948 C. nigoni genes without C. briggsae orthologs (10.1%) represent losses in C. briggsae based on homologs in Caenorhabditis outgroups (fig. S3). Finally, 1577 C. nigoni genes (6.4%) lacked homologs entirely and were classified as orphans. These genes could be exceptionally divergent, recently arisen in C. nigoni, or arisen shortly before the C. nigoni–C. briggsae split but then lost in C. briggsae. Overall, gene loss in C. briggsae appears to be the primary driver of the gene number difference.

To characterize genes lost in C. briggsae, we first compared Pfam protein domains encoded by C. nigoni versus C. briggsae. We found 26 Pfam domains that were overrepresented in C. nigoni (fig. S4 and table S5); of these, seven were consistently overrepresented in outcrossing C. nigoni, C. remanei, and C. brenneri relative to the selfing species C. briggsae and C. elegans. Three of these
domains (F-box, FBA_2/F-box associated, and BTB) are predicted to mediate protein-protein interactions. Male-female *Caenorhabditis* had 272 to 1074 genes in these families, whereas hermaphrodite *Caenorhabditis* had only 101 to 258 genes per family. Two other domains (Peptidase_A17 and DNA_pol_B_2) are associated with repetitive DNA. The final two overrepresented domains were Asp_protease_2 (possibly associated with retroelements) and DUF3557 (a nematode-specific domain, currently of unknown function). One overrepresented domain specific to *C. nigoni* was zf.RING2_finger; the RING domain gene *spe-42* is important for sperm-egg interactions in *C. elegans* (25).

Because *C. nigoni*–specific genes might encode fast-evolving proteins that lack known domains, we compared other gene properties. We found that although genes encoding medium to large proteins (>200 residues) are similar in frequency in both species, *C. nigoni* encodes disproportionately more small proteins (<200 residues) than *C. briggsae* (Fig. 3B and table S6). As seen in other *Caenorhabditis* ([11]), genes with male-biased expression outnumber female-biased genes (Fig. 3C and table S7). However, even against this background, *C. nigoni* genes without *C. briggsae* homologs are disproportionately male-biased in expression. Preferential loss of small and fast-evolving proteins thus occurred in *C. briggsae* after the adoption of selfing.

**mss genes encode sperm glycoproteins lost in hermaphrodites**

We hypothesized that genes with highly male-biased expression that are present in outcrossing species, but lost in selfing species, might function in sexual selection. Among such genes we identified the *mss* (*male secreted short*) family.
found one to four mss genes in the outcrossing species C. nigoni, C. sinica, C. remanei, C. brenneri, C. sp. 34, C. japonica, and C. afra, but found none in the selfing C. elegans, C. briggsae, and C. tropicalis. The mss family encodes small proteins (median 111 residues) with N-terminal signal sequences present in the mss-specific sequence types to genome size. (Fig. 3D) Enzyme treatments confirmed that mss proteins were heavily glycosylated (fig. S5).

Although we failed to detect mss genes in selfing species, we did discover a larger family of mss-related protein (msrp) genes, within which mss forms a monophyletic clade (fig. S6) (20). Notably, msrp genes are found both in outcrossing Caenorhabditis and in the hermaphroditic C. elegans, C. briggsae, and C. tropicalis (fig. S6). Like MSs proteins, MSRP proteins are small and are predicted to be secreted, O-glycosylated, and (often) GPI-anchored. Both mss and msrp genes show male-biased expression in C. nigoni and other species (table S8). In cases where their chromosomal loci can be identified, mss and msrp genes are autosomal; this linkage fits a general pattern in heterogametic male species of male-biased genes being autosomal rather than X-chromosomal (26) and references therein.

Because we observed mss genes in two C. elegans outgroups (C. japonica and C. afra) (fig. S6 and table S8), their absence from hermaphrodites most likely reflects independent gene losses rather than phylogenetic restriction to close relatives of C. elegans. Examination of the C. briggsae genome region syntenic to the C. nigoni mss locus revealed fragments of mss-1 and mss-2 coding sequences and a nearly complete mss-3 pseudogene (fig. S3E) (20). Mutations that ablate Chr-mss-3-ps function in the AF16 reference strain also occur in 11 wild isolates that span the known diversity of C. briggsae (fig. S7) (20, 27). Orthologs of all three C. nigoni mss genes were therefore present in the common ancestor of C. nigoni and C. briggsae but were lost in C. briggsae before its global diversification.

In the outcrossing species C. remanei, mss transcripts were expressed only in adult males (Fig. 4A), with strongest expression in spermatocytes during mid-pachytene of meiosis I (fig. 4B). To determine subcellular localization of MSS peptides, we used CRISPR/Cas9 editing to tag the Cre-mss-1 gene of C. remanei with the hemagglutinin (HA) epitope. Crem-MSS-1::HA expression was first detected in large vesicles and on the plasma membrane of spermatocytes, with intensity increasing and localization restricted to secretory vesicles in mature spermatozoids (Fig. 4, C to E). The secretory vesicles of nematode sperm, known as membranous organelles (MOs), fuse with the plasma membrane upon ejaculation and sperm activation (28).

MSS peptides might be processed by a signal peptidase to release a soluble fragment into the MO lumen, which could then be dumped into seminal fluid upon sperm activation. However, their transient plasma membrane localization in spermatocytes and predicted C-terminal GPI attachment signals (Fig. 3D and table S8) suggested that MSS peptides might instead be attached to membranes. Consistent with this latter hypothesis, Crem-MSS-HA remained associated with activated sperm dissected from inseminated females (Fig. 4F). We observed staining of the plasma membrane and of MO-derived puncta...
Fig. 3. Comparison of the *C. nigoni* and *C. briggsae* proteomes. (A) Scatterplot of sizes of OrthoFinder gene families, excluding one-to-one orthologs (table S4). Of 2367 families with unequal numbers of *C. nigoni* and *C. briggsae* genes, the majority (1624) were larger in *C. nigoni* than in *C. briggsae* (P < 2.2 × 10⁻¹⁶, Wilcoxon signed-rank test). Dotted line indicates equal family sizes. (B) Length distributions of *C. nigoni* and *C. briggsae* proteins and of *C. nigoni* proteins that lack *C. briggsae* homologs. (C) Length distributions of *C. nigoni* and *C. briggsae* proteins and of *C. nigoni* proteins that lack *C. briggsae* homologs. (D) For genes with sex-biased expression, male bias was seen for 50.9% of 6804 genes with homologs. (E) Comparison of *mss* gene regions in *C. nigoni* and *C. briggsae*. Pastel shapes connect homologous sequences. Except for Cni-*mss-3*, all genes are transcribed from left to right. Genes surrounding the three *C. nigoni* *mss* paralogs are conserved in *C. briggsae*, but only fragments and a pseudogene (*Cbr-mss-3-ps*) of the *mss* genes remain. The pseudogene has a lost start codon and a +1 frameshift. CBG26068 has a novel 3' exon derived from part of the Cni-*mss-1* second exon. See fig. S7 and (20) for details.

**mss** genes mediate sperm competition and affect sex ratios

Because the four *C. remanei* *mss* paralogs form a 7-kb tandem array (Fig. S8A), we deleted the entire *mss* cluster via CRISPR/Cas9 editing. To avoid inbreeding depression associated with homozygosity of entire chromosomes (30) (Fig. S8B), we generated the *mss* deletion in two different *C. remanei* strains and crossed them to create hybrid *mss*-null mutants. The resulting males showed no intrinsic fertility defects, as judged by overall brood size (fig. S8C). However, when competing against heterozygous *mss*(null/+) males, *mss* mutants sired fewer progeny than nonmutants in both offense (mutant male second) and defense (mutant male first) scenarios (Fig. 5, A and B). The *mss* family is therefore required for male sperm competitiveness in multiple mating situations, but not for fertility itself. Sperm lacking *mss* compete poorly even when the female reproductive tract is conditioned by wild-type sperm. Thus, *mss* proteins probably do not function as a secreted signal, but instead act cell-autonomously.

We then introduced *mss-1* and *mss-2* genes from *C. nigoni* into *C. briggsae* via a low-copy, germline-expressed *mss* transgene; this transgene was strongly expressed in *C. briggsae* males, while also being detectable in hermaphrodites (fig. S9). Remarkably, sperm from transgenic *mss*(+) *C. briggsae* males outcompeted those of wild-type males (Fig. 5, C and D). After *mss*(+) sperm were exhausted, however, wild-type *mss* (null) sperm were still fertilization-competent.
**Fig. 4. C. remanei MSS is a male-specific protein localized to the surface of activated sperm.**

(A) Quantitative reverse transcription polymerase chain reaction (for Cre-mss-2) of mixed-sex populations (top) versus larval and adult sex-specific populations (bottom), showing that mss expression is specific to adult males. Data are means ± SEM. Female data are two to three orders of magnitude below male data. (B) Cre-mss-1 transcripts are detected in pachytene-stage primary spermatocytes. (C) Dissected testes expressing HA-tagged Cre-MSS-1, viewed with differential interference contrast (DIC, left) or anti-HA confocal fluorescence (right) microscopy. Cre-HA-MSS-1 is first detectable in spermatocytes (sc) and becomes enriched in spermatids (st). (D) Some Cre-HA-MSS-1 is localized to the plasma membrane of spermatocytes, as indicated by the arrow. Blue fluorescence: Hoechst-stained DNA. (E) Enlarged view of the boxed region in (C), showing complete restriction to membranous organelles (MOs). (F) Cre-HA-MSS-1 remains attached to sperm after activation and transfer to the female. (G) Cre-HA-MSS-1 sperm cells dissected from a female and stained with anti-HA immunohistochemistry, imaged with DIC (top) and confocal (bottom) microscopy. Cre-HA-MSS-1 is visible in the plasma membrane and fused MO remnants.

(Fig. 5, C and D). In addition, mss(+) males were more consistently able to suppress use of a hermaphrodite mate’s self-sperm (Fig. 5E).

Because 50% of outcross progeny are male whereas selfed progeny are almost exclusively hermaphrodite mate, more consistent ability to suppress use of a hermaphrodite mate from mixed-sex populations (top) versus larval and adult sex-specific populations (bottom), showing that mss expression is specific to adult males. Data are means ± SEM. Female data are two to three orders of magnitude below male data. (B) Cre-mss-1 transcripts are detected in pachytene-stage primary spermatocytes. (C) Dissected testes expressing HA-tagged Cre-MSS-1, viewed with differential interference contrast (DIC, left) or anti-HA confocal fluorescence (right) microscopy. Cre-HA-MSS-1 is first detectable in spermatocytes (sc) and becomes enriched in spermatids (st). (D) Some Cre-HA-MSS-1 is localized to the plasma membrane of spermatocytes, as indicated by the arrow. Blue fluorescence: Hoechst-stained DNA. (E) Enlarged view of the boxed region in (C), showing complete restriction to membranous organelles (MOs). (F) Cre-HA-MSS-1 remains attached to sperm after activation and transfer to the female. (G) Cre-HA-MSS-1 sperm cells dissected from a female and stained with anti-HA immunohistochemistry, imaged with DIC (top) and confocal (bottom) microscopy. Cre-HA-MSS-1 is visible in the plasma membrane and fused MO remnants.

Discussion

Comparison of the C. nigoni and C. briggsae genomes revealed that C. briggsae experienced rapid contraction of chromosomes and loss of protein-coding genes. However, loss of ancestral genomic content in C. briggsae does not fully explain their genomic divergence; the ongoing birth of novel sequences in both species, along with loss of ancestral DNA in C. nigoni, is also important. Net shrinkage of the C. briggsae genome therefore resulted from a substantial increase in the ratio of losses to gains. These losses included many coding sequences, reducing the C. briggsae gene count by nearly one-quarter.

Multiple observations implicate the evolution of selfing as the cause of genome shrinkage in C. briggsae. Reduced genome and transcriptome sizes are observed in all three selfing Caenorhabditis species (10, 11). Continued interfertility of C. briggsae and C. nigoni (15) indicates that self-fertility and genome shrinkage evolved in quick succession. Genes with male-biased expression, such as the mss family, are disproportionately and consistently lost from selfing species (11). This suggests that genes with male reproductive functions that are either dispensable or maladaptive in the new sexual mode are purged from the genome. Finally, the net genome shrinkage we observed has been predicted to arise from a partially selfing mating system coupled with transmission distortion of autosomal deletion alleles (32, 33). Such distortion is driven by imbalanced chromatin during meiosis I of XO males, and causes preferential inheritance of shorter alleles by hermaphrodite progeny and their increased fixation in the population.

Larger autosomal deletions, influenced most by the deletion segregation distortion mechanism, are primarily responsible for the smaller genome of C. briggsae (Fig. 2). However, such deletions and net shrinkage were also found on the X chromosome (table S2), which should be unaffected. Moreover, orthologous genes have larger introns in C. briggsae than in C. nigoni (Fig. 2), and introns constitute a greater fraction of the C. briggsae genome (fig. S2). X-chromosomal C. briggsae introns are also larger than those of the outgroup C. remanei (10) (fig. S2C), which suggests that introns of many genes expanded in C. briggsae. Thus, additional processes must also contribute to shrinkage of the C. briggsae genome. Spontaneous short (1- to 5-nucleotide) mutations in C. elegans are biased toward insertions rather than deletions.
served regardless of mating system, we found crossing sperm is likely due to ongoing sexual cavities of selfing hermaphrodites, sterilizing or mss
duction. Unlike expression are disproportionately lost in
ive (C. nigoni genome size, given its recent divergence from
mss-C. remanei mss
mate first (“defense”), heterozygous C. remanei mss(nmDf1/+). males have a slight advantage over wild-type males; mss-null mutants, in contrast, do not (N = 15 for both). Heterozygote success is assumed to be double the observed nmDf1 frequency in their progeny. For both defense and offense, P < 0.01 (two-sample Kolmogorov-Smirnov test).
(C and D) Wild-type young C. briggsae hermaphrodites were mated sequentially (4 hours each) with conspecific males carrying either a C. nigion mss(+). transgene or a control mCherry:histone reporter (RW0025). Progeny laid 0 to 18 hours and 18 to 42 hours after the second mating were scored for green (MSS+), red (RW0025), or no (self) fluorescent markers. In both offense (C) and defense (D), MSS+ males sire several times as many progeny as control males in the first laying window. *P < 0.001. (E) MSS+ C. briggsae males suppress selfing more effectively than do control AF16 (wild-type) males. Strain JU936 is a second control strain bearing two transcriptional GFP reporters in the AF16 background. *P < 0.001 (Kolmogorov-Smirnov test); ns, not significant. (F) Male frequency in MSS+ and wild-type AF16 C. briggsae populations in which male frequency was artificially elevated to 50% at the start of the experiment. In all panels except (E), error bars denote SD.

Fig. 5. mss genes are necessary for sperm competitiveness in an outcrossing species, and sufficient to enhance it in a selfing species. (A) When mated after a wild-type male (“offense”), C. remanei mss(nmDf1/+). males sire more than twice the progeny of nmDf1/nmDf2 mss-null mutants (N = 16 for both). (B) When allowed to mate first (“defense”), heterozygous C. remanei mss(nmDf1/+). males have a slight advantage over wild-type males; mss-null mutants, in contrast, do not (N = 15 for both). Heterozygote success is assumed to be double the observed nmDf1 frequency in their progeny. For both defense and offense, P < 0.01 (two-sample Kolmogorov-Smirnov test).

(C and D) Wild-type young C. briggsae hermaphrodites were mated sequentially (4 hours each) with conspecific males carrying either a C. nigion mss(+). transgene or a control mCherry:histone reporter (RW0025). Progeny laid 0 to 18 hours and 18 to 42 hours after the second mating were scored for green (MSS+), red (RW0025), or no (self) fluorescent markers. In both offense (C) and defense (D), MSS+ males sire several times as many progeny as control males in the first laying window. *P < 0.001. (E) MSS+ C. briggsae males suppress selfing more effectively than do control AF16 (wild-type) males. Strain JU936 is a second control strain bearing two transcriptional GFP reporters in the AF16 background. *P < 0.001 (Kolmogorov-Smirnov test); ns, not significant. (F) Male frequency in MSS+ and wild-type AF16 C. briggsae populations in which male frequency was artificially elevated to 50% at the start of the experiment. In all panels except (E), error bars denote SD.

(34), although biases in formation of larger indels remain uncharacterized. Regardless, the relative rates of insertion and deletion mutations likely evolved too slowly to explain C. briggsae’s reduced genome size, given its recent divergence from C. nigion (27). Gene loss can sometimes be adaptive (35, 36) and has been proposed as a factor promoting genome shrinkage in selfing Caenorhabditis (37). Our results for C. nigion-C. briggsae pair support this hypothesis.

Genes encoding small proteins with male-biased expression are disproportionately lost in C. briggsae; here, mss provides an instance affecting reproduction. Unlike comp-1, which encodes a kinase required for male versus hermaphrodite sperm competition in C. elegans (37) and which is conserved regardless of mating system, we found mss orthologs only in outcrossing species. In interspecies matings, sperm from males of outcrossing species rapidly invade the ovaries and body cavities of selfing hermaphrodites, sterilizing or killing them (9). This cryptic toxicity of outcrossing sperm is likely due to ongoing sexual selection in outcrossing species. Given their pronounced role in sperm competition, MSS proteins may contribute to sperm invasiveness.

How MSS improves sperm competitiveness remains unclear; but mature MSS proteins are substantially glycosylated (fig. S5). Such post-translational modification may impose little constraint on MSS proteins, explaining how they can have weak sequence conservation yet strong functional conservation. Another poorly conserved O-glycosylated protein, the mucin PLG-1, forms a copulatory plug found in all male-female Caenorhabditis species but lost in many wild isolates of C. elegans (4). Glycoproteins form the glyocalyx coat of mammalian sperm and play important roles in fertility (38). Caenorhabditis provides a useful model for how the glyocalyx and female tissues interact and how these interactions affect sperm competition.

Independent loss of mss in the three known hermaphroditic Caenorhabditis species could reflect relaxed sexual selection coupled with mutation and drift, or it could reflect adaptive con-vergence. Other changes in selfing species—such as loss of plg-1 and of plep-1, which mediates reliable male discrimination between the vulva and excretory pore (4, 6)—are likely due to relaxed selection. However, restoring mss to C. briggsae enhances male fitness (Fig. 5, C and D), and mutations inactivating the Cbr-mss-3-ps pseudo-gene are not deletions that would be subject to loss via transmission ratio distortion (fig. S7). These findings suggest that loss of mss may instead reflect adaptive convergence, permitting proto-hermaphrodites to adapt to a selfing lifestyle and resolve emergent sexual conflicts related to mating (39–41). Selfing Caenorhabditis species lack in-breeding depression (42) and reproduce in spatially isolated habitats colonized by small numbers of founders (3). Reduced male mating success creates hermaphrodite-biased sex ratios (Fig. 5F), which may be adaptive under these conditions (41, 43–45). Thus, evolutionary transitions in re-productive mode may produce conditions for selection to rapidly eliminate formerly constrained reproductive genes.
ACKNOWLEDGMENTS

We thank E. Antoniou, P. W. Sternberg, C. T. Brown, the Michigan State University High-Performance Computing Center (supported by USDA grant 2030-65205-20361 and NIFA-NSF grant IOS-0923812), and the UC Berkeley V. J. Coates Genomics Sequencing Laboratory (supported by NIH5RO1GM029660) for sequencing and computational support. We thank G. Williams and R. H. Waterston for transcriptome data, the Caenorhabditis Genomes Project for prepublication access to the genomes of C. atra and C. sp. 34, Z. Zhao and M. A. Félix for nematode and plasmid reagents, and S. Mount for helpful discussions. Supported by NSF award IOS-1255169 and NIH grant GM07414 (E.S.H.); NIH grant AI111173, Moore Foundation grant 4551, and Cornell University start-up funds (E.M.S.); and NIH grant GM03702 (B.J.M.). B.J.M. is an investigator of the Howard Hughes Medical Institute. Some strains were provided by the CGC, which is funded by the NH Office of Research Infrastructure Programs (P40 OD010440). Data availability and accession codes. C. nigoni genomic and transcriptomic data have been archived as NCBI BioProject accessions PRJNA384657 and PRJNA384658, and the C. nigoni genome assembly as DDBJ/ENA/GenBank accession PRJNA30000000. Genome assembly, gene prediction, and gene expression data for C. nigoni, with supplementary data sets for other species, have been archived at the OSEF (C. nigoni: https://osf.io/dbw4 and doi:10.17605/osf.io/dbw4; other species: https://osf.io/b47l6 and doi:10.17605/osf.io/b47l6).

SUPPLEMENTARY MATERIALS

www.sciencemag.org/content/359/6371/55/suppl/DC1

Materials and Methods

Figs. S1 to S9
Tables S1 to S9
Data S1 to S5
References (46–119)

13 June 2017; accepted 17 November 2017
10.1126/science.aao8287
Rapid genome shrinkage in a self-fertile nematode reveals sperm competition proteins

Da Yin, Erich M. Schwarz, Cristel G. Thomas, Rebecca L. Felde, Ian F. Korf, Asher D. Cutter, Caitlin M. Schartner, Edward J. Ralston, Barbara J. Meyer and Eric S. Haag

Science 359 (6371), 55-61.
DOI: 10.1126/science.aao0827

Examining the consequences of selfing

The Caenorhabditis genus of nematodes includes a mix of closely related outcrossing and self-fertilizing (selfing) species. Genome size differs widely among these different species. Yin et al. generated a genome assembly for the outcrossing nematode C. nigoni and compared it with that of its close relative, the selfing C. briggsae. C. briggsae has experienced a substantial decrease in genome size since the two species' recent divergence. The underlying causes of this size difference appear to involve a decrease in protein-coding genes and changes in other types of sequences that have homology with RNAs expressed primarily in C. nigoni males. One of the implicated gene families, the mss family, compromises sperm competitiveness. Thus, in nematodes, selfing appears to result in a decrease in genome size owing to selection to reduce male reproductive function.

Science, this issue p. 55