switch gene, *Sex-lethal*, and thereby initiate female development[6], whereas *C. elegans* represses expression of its target *xol-1* through transcriptional and post-transcriptional mechanisms. No evidence exists for post-transcriptional regulation operating in the initial X counting process of flies, although they do use a post-transcriptional mechanism to lock in the counting choice[6]. Mammals must also assess their X-chromosome number to achieve dosage compensation through X inactivation. Although the mechanism has not been determined, the evidence available[11] implies that it is likely to differ from the chromosome-counting mechanisms in both flies and worms. □

Methods

Genetic mapping. *y303* and *y304* were isolated using a previously described screen[4]. Of 948 embryos counted from *fox-1(y303)* XX hermaphrodites, all developed into fertile adults. *y303* was mapped between *lin-32* and *unc-2* by analysing Unc non-Dpy and Dpy non-Unc recombinant progeny from *dpy-3* + *y303 unc-2*/+ *lin-32* + +mothers. Of 21 Unc non-Dpy recombinants, 4 recombined between *dpy-3* and *lin-32*, 5 between *lin-32* and *y303*, and 12 between *y303* and *unc-2*. Of 6 Dpy non-Unc recombinants, 2 recombined between *lin-32* and *y303* and 4 between *y303* and *unc-2*. *y304* was similarly mapped. Of 16 Unc non-Dpy recombinants, 4 recombined between *dpy-3* and *lin-32*, 7 between *lin-32* and *y304*, and 5 between *y304* and *unc-2*. Of 17 Dpy non-Unc recombinants, 5 recombined between *lin-32* and *y303*, and 12 between *y304* and *unc-2*. *yDf19* is a γ ray-induced mutation that fails to complement *egl-17* and *unc-1* and does not cause X-chromosome non-disjucntion. Of 976 self progeny from *yDf19/unc-1 dpy-3* mothers, none was male. *meDf6* removes *lin-32* but probably not *fox-1*. Fourteen dead embryos from *meDf6/dpy-3 unc-2* mothers amplified a control band and a *fox-1*-specific band in single-embryo polymerase chain reactions (PCRs). *yDf20* is a suppressor of the XO-specific lethality caused by two copies of *yDp14* (I. Carmi and B.J.M., unpublished results) and deletes *dpy-3*, *lin-32* and *fox-1*. Fifteen dead embryos from *yDf20/unc-1 dpy-3* mothers that amplified a control band failed to amplify a *fox-1*-specific band in single-embryo PCR. *yDf17* was isolated as a suppressor of the XO-specific lethality caused by *mnDp66/yDp14*. *yDf17* removes the left end of X, including *unc-2*, and *yDf17*/ + XX animals have an Sdc phenotype similar to that of *yDf14*/ + XX animals (ref. 4, and I. Carmi and B.J.M., unpublished results). For single-embryo PCRs[12], the primer pairs 5′-CCCATTCAGATTCAGAGACC and 5′-CGAGTGAACACGAGCTGTAG amplified an internal fragment of *fox-1* (ref. 9). The control primer pairs 5′-CTACTGTCGACAATGTTGGAATCCTC and 5′-GGGATTTCTGCAGTTG-CAAGATG amplified a fragment of *sdc-3*.

DNA sequence analysis of *y303* and *y304*. The DNA changes caused by *y303* and *y304* were determined by DNA sequence analysis of cDNA generated by reverse-transcribed PCRs of RNA extracted from mutant (*dpy-3 y303* and *dpy-3 y304*) and parental (*mnDp66*; *unc-1 dpy-3*) strains. *fox-1* was amplified using the primers 5′-CCGCTCGAGATGCAAGCCCTGTACCAACT or 5′-CAGTCGGCGTTTGGAATGGATCC with 5′-CGGGATCCCACTCAATACGG AGTAAATCG. The PCR products for each strain were cloned for three independent reactions and were sequenced from three clones. All three sets of sequences from the *y303* strain revealed only a C-to-T transition at nucleotide 109, and from the *y304* strain, a C-to-T transition at nucleotide 481.

***xol-1* reporter genes and transgenic arrays.** The *Pxol::lacZ* transcriptional fusion (pMN21) was made by insertion of the 2,779-bp *Eco*RI fragment from the *xol-1* promoter region into the *Sma*I site of pPD95.03, an intron-rich version of pPD16.01 (ref. 13) (from A. Fire). The *xol-1/lacZ* junction occurs 71 bp upstream of the first ATG in *xol-1*, thus eliminating *xol-1*'s normal 5′UTR, made by the insertion of an SL1 *trans*-spliced leader 13 nucleotides upstream of that ATG. *yIs33* was made by integrating an extrachromosomal array containing pRF4, a plasmid encoding a dominant *rol-6(su1006)* marker, and pMN21 coinjected at 100 :40 μg ml^{-1}. The *gfp*-tagged *xol-1* genomic fusion (pMN45) (*gfp::xol-1*) was made by inserting a PCR fragment of *gfp* coding sequences (a S65T, I167T variant from Y. Jin) in frame at the first ATG of *xol-1*. The *yEx(gfp::xol-1)* extrachromosomal arrays contain p76-16B, a plasmid that encodes *unc-76*(+), and pMN45 co-injected at 150 : 10 μg ml^{-1}. The *Pxol-1::gfp* translational fusion (pMN15) was made by replacing the 3,441-bp *Sph*I–*Apa*I fragment from genomic *xol-1* sequences with the 1,931-bp *Sph*I–*Apa*I *gfp* fragment from the expression vector pPD95.67, which encodes the S65C variant of GFP (from A. Fire). The *yEx(Pxol-1::gfp)* extrachromosomal arrays contain p76-16B and pMN15 coinjected at 150 : 20 μg ml^{-1}. More than one thousand embryos of each appropriate genotype were scored in experiments using *xol-1* reporter constructs. The XO-specific expression of transgenic arrays carrying these reporter constructs was shown by the lack of embryonic activity in transgenic strains that produced only wild-type XX embryos and the abundant embryonic activity in transgenic lines that produced wild-type XO embryos because they carried a *him-5* mutation[14]. Excess *fox-1* was produced from an integrated array, *yIs44*, which causes XO-specific lethality and contains multiple copies of the *fox-1*-containing cosmid R04B3 and pRF4.

FOX-1 antibodies. Rabbit polyclonal antibodies were raised against a bacterially expressed HIS6::FOX-1 fusion protein that included the entire FOX-1 protein. The *fox-1* open reading frame was cloned into the expression vector pRSET-A (Invitrogen), and the HIS6::FOX-1 fusion protein was expressed and purified according to the procedures of Qiagen. Antibodies were affinity-purified using a fusion protein containing the entire FOX-1 protein and the maltose-binding protein (New England Biolabs). Embryos were stained as described[15] using a 1 : 50 dilution of affinity-purified FOX-1 antibodies.

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correction

Structure of the adenylyl cyclase catalytic core

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In Fig. 3, the sequence in line 2 from 908 to 943 should read PGELVHMLNELFGKFDQIAKENEC…MRIKILGDC, and from 1,004 to 1,022 should be NVLCGVIGLQKWQYDVWSH. An extraneous 19 amino acids appeared at the start of the third line of the last block of sequence. This line should therefore start with AGGRA…. Also, the correct GenBank accession code for bovine type 1 adenylyl cyclase is M25579. □