(100–441) and Tc1-intron (321–566) (numbers from genomic sequence). The probe used for *gfp* was gfp1 (19–319) (numbered from ATG). Details of the probes used for analyses of Tc*3* and Tc*5* dsRNA are available on request. 5′-RACE analyses used the SmartII kit (Clontech), SuperScriptII reverse transcriptase (GibcoBRL) and Pwo DNA polymerase. S100H fractions were prepared as described in ref. 25. Standard procedures were used for primer-extension analyses. Sequences of all primers used are available on request. Quantification of protected fragments (RNase protection assays) was performed using ImageQuant software.

Transgenic lines

The chimaeric *gfp* reporter plasmids were produced by inserting various fragments into plasmid pAZ132 (ref. 26). Plasmid pAZ1 (TIR fusion) contained nucleotides 1–54 of Tc*1* (genomic sequence) in the sense orientation in the *Sgr*AI site. Plasmid pAZ4 (*unc-22* fusion) contained nucleotides 11,137–11,190 of *unc-22* (spliced sequence) in the sense orientation in the *Sgr*AI site. Plasmid pAZbb (TIR 3′ stop) contained nucleotides 1–54 of Tc*1* in the sense orientation in the *Bsa*BI site. To prevent transgene silencing due to the presence of high transgene copy numbers[27], low-copy-number transgenic lines were generated by ballistic transformation[28] using a heptamer adaptor (Bio-Rad). Transformants were generated in *unc-119(dp38)* worms. All lines were selected and they were analysed for GFP expression on both wild-type (OP50) and *mut-16* dsRNA food. Worms were grown under these conditions for two generations. Lines not expressing GFP under any of these conditions were discarded; these included six TIR fusion lines, two *unc-22* fusion lines and ten TIR 3′ stop lines. By DNA blot analyses (carried out according to standard procedures), transgene copy number was determined using *Sac*II- and *Bgl*II-digested genomic DNA and *gfp*- and pBlueScript-specific probes. Crosses using *pkIs*1660 showed that all three transgene copies in this line reside at one locus and segregate in a mendelian manner. However, the transgenes can be lost (presumably due to recombination), as is apparent from the presence of worms with an *unc-119* phenotype (PCR analyses confirmed transgene loss in these worms). Transgene loss is not uncommon for ballistic-generated transformants and varies for the lines as follows: *pkIs*1660, 1%; *pkIs*1661, 10%; *pkIs*1662, 10%; *pkIs*1663, 2%; *pkIs*1664, 80%; *pkIs*1665, 0%; *pkIs*1666, 1%; *pkIs*1667, 50%; *pkIs*1668, 0%; *pkIs*1669, 0%; *pkIs*1671, 0%; and *pkIs*1672, 0%. Interestingly, transgene loss strongly increases upon crossing to strains defective in transposon silencing (*mut-7* and *pk732* but not *rde-1*); this transgene loss is not dependent on the presence of the Tc*1* TIR sequence, as it also occurs upon crossing *mut-7* to *pkIs*1665, an *unc-22* fusion line.

dsRNAs

Plasmids for dsRNA production in *E. coli* comprised pTS302 dsRNA[11] (for *unc-22*) and pTS303 (containing nucleotides 1–441 of the Tc*1* genomic sequence inserted into the *Sma*I site of vector L4440 (ref. 29)). *E. coli* expressing *mut-16* dsRNA were obtained from well number 17C5 of the *C. elegans* feeding library[30].

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corrigendum

An NS3 protease inhibitor with antiviral effects in humans infected with hepatitis C virus

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In this Letter, the 'Competing interests statement' should be corrected to: 'The authors declare competing financial interests: R.E.S. was the clinical investigator and received an honorarium from Boehringer Ingelheim. All the other authors are or were employees of Boehringer Ingelheim.'. □