

(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 Tc3 and Tc5 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 chimeric *gfp* reporter plasmids were produced by inserting various fragments into plasmid pAZ132 (ref. 26). Plasmid pAZ1 (TIR fusion) contained nucleotides 1–54 of Tc1 (genomic sequence) in the sense orientation in the *SgrAI* site. Plasmid pAZ4 (*unc-22* fusion) contained nucleotides 11,137–11,190 of *unc-22* (spliced sequence) in the sense orientation in the *SgrAI* site. Plasmid pAZbb (TIR 3' stop) contained nucleotides 1–54 of Tc1 in the sense orientation in the *BsaBI* site. To prevent transgene silencing due to the presence of high transgene copy numbers²⁷, low-copy-number transgenic lines were generated by ballistic transformation²⁸ 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 *SacII*- and *BglII*-digested genomic DNA and *gfp*- and pBlueScript-specific probes. Crosses using *pKIs1660* 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: *pKIs1660*, 1%; *pKIs1661*, 10%; *pKIs1662*, 10%; *pKIs1663*, 2%; *pKIs1664*, 80%; *pKIs1665*, 0%; *pKIs1666*, 1%; *pKIs1667*, 50%; *pKIs1668*, 0%; *pKIs1669*, 0%; *pKIs1671*, 0%; and *pKIs1672*, 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 Tc1 TIR sequence, as it also occurs upon crossing *mut-7* to *pKIs1665*, an *unc-22* fusion line.

dsRNAs

Plasmids for dsRNA production in *E. coli* comprised pTS302 dsRNA¹¹ (for *unc-22*) and pTS303 (containing nucleotides 1–441 of the Tc1 genomic sequence inserted into the *SmaI* site of vector L4440 (ref. 29)). *E. coli* expressing *mut-16* dsRNA were obtained from well number 17C5 of the *C. elegans* feeding library³⁰.

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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.’ □