CORRECTION

Correction: Antinematode Activity of Violacein and the Role of the Insulin/IGF-1 Pathway in Controlling Violacein Sensitivity in *Caenorhabditis elegans*

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There is an error in the eleventh paragraph of the Results section. The correct paragraph is: In C. elegans DAF2/DAF16 controls the expression of various effector genes including those relevant for detoxification and antimicrobial activity such as the superoxidase dismutase gene sod-3 and antimicrobial genes *spp-1* and *lys-7* [23,38]. Thus given that the precise molecular target/ s for violacein in C. elegans are unknown we sought to determine which, if any, of these relevant downstream genes are required for the increased resistance to violacein observed in daf-2 null and DAF-16 over-expressing strains. Specifically we chose to test violacein sensitivity in C. elegans mutants defective in sod-3, spp-1 and lys-7 (Table 2) because of the previous reported involvement of these genes in immunity to bacterial accumulation [39,40,41]. We found that daf-2;sod-3 double mutant displayed significantly reduced survival compared to the single mutant *daf-2* (p<0.0001, Fig 6A) when exposed to the 20G8 clone in a nematode killing assay. Interestingly a single mutation in gene spp-1 significantly reduced the nematode's life span when compared to wild type animals (p<0.0001), while the viability of the nematode was not affected by mutations in the *lys-7* and *sod-3* genes (p>0.05, Fig 6B). These data indicate that resistance to violacein in *daf-2* mutants is at least in part driven by SPP-1 and SOD-3, with the antimicrobial LYS-7 having little or no involvement.

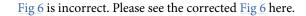


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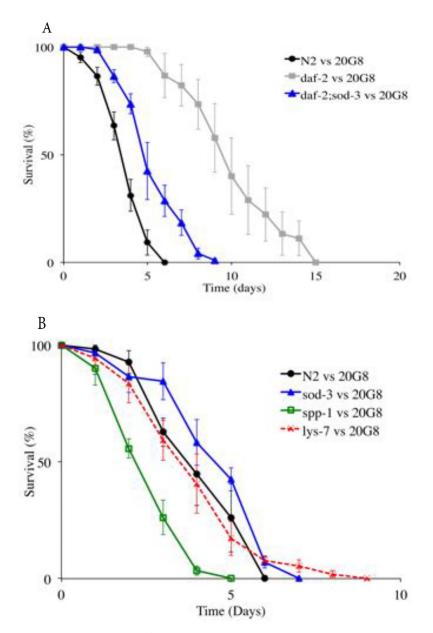


Fig 6. Nematode killing assay (wild type animals and *daf-2*, *daf-2*;*sod-3*, *spp-1*, *lys-7*, *sod-3* **mutant nematodes vs the 20G8 clone).** (A) The survival of the nematode was tested using *C. elegans* double mutant *daf-2*;*sod-3*, and (B) the single mutant animals *sod-3*, *spp-1*, and *lys-7*. Each data point represents means ± the standard error of three replicate plates. p values were calculated on the pooled data of all of the plates done in each experiment by using the log-rank (Mantel–Cox) method and the values are provided in the text.

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Table 2 is incorrect. Please see the corrected Table 2 here.

Strain name	Genotype/allele designation	Relevant characteristics	Source or reference
N2 Bristol	C. elegans wild isolate	Wild type isolate	CGC ^a
CU1546	smIs34	ced-1p::ced-1::GFP + rol-6(su1006)	CGC ^a
CB1370	daf-2(e1370) III	Mutated in the insulin-like receptor DAF-2. Temperature sensitive dauer constitutive	CGC ^a
IU10	daf-16(mgDf47) I; rrf-3(pk1426) II	Mutated in the FOXO-family transcription factor DAF-16	CGC ^a
TJ356	<i>zIs356</i> IV	Integrated DAF-16::GFP roller strain. Daf-c, Rol, fluorescent DAF-16::GFP. Overexpression of DAF-16	CGC ^a
JT9609	<i>pdk-1 (Sa680)</i> x	Mutation in the gene encoding for 3-phosphoinositide-dependent protein kinase	CGC ^a
RB1178	wwp-1(ok1102) I.	Mutation in the gene encoding for the WW domain protein 1	CGC ^{ab}
TM127	daf-2(e1370) III; sod-3(sj134) X	Double mutant in the insulin-like receptor DAF-2 and in the superoxide dismutase SOD-3	CGC ^a
GA186	<i>sod-3(tm760)</i> X	Mutated in the iron/manganese superoxide dismutase SOD-3	CGC ^a
RB1286	<i>lys-7(ok1384)</i> V	Mutated in the putative antimicrobial lysozyme LYS-7	CGC ^a
RB2045	<i>spp-1(ok2703)</i> III	Mutated in the antimicrobial peptide caenopore SPP-1	CGC ^a

Table 2. C. elegans strains used in this study.

^a *Caenorhabditis* Genetics Center, the University of Minnesota.

^b C. elegans Gene Knockout Project http://www.celeganskoconsortium.omrf.org.

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Reference

 Ballestriero F, Daim M, Penesyan A, Nappi J, Schleheck D, Bazzicalupo P, et al. (2014) Antinematode Activity of Violacein and the Role of the Insulin/IGF-1 Pathway in Controlling Violacein Sensitivity in *Caenorhabditis elegans*. PLoS ONE 9(10): e109201. https://doi.org/10.1371/journal.pone.0109201 PMID: 25295516