Electronic Supplementary Material (ESI) for Food & Function.

Supplementary data

Table S1. PMT-E increased the body bending rate of N2 wild-type *C. elegans*.

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Fig. S1. Identification of bioactive compounds in PMT-E.

Fig. S2. The HPLC standard curves of TSG and EG.

Fig.S3. Effects of TSG and EG on oxidative stress resistance and Aβ-induced paralysis.

Fig.S4. The toxicity of PMT-E.

Table S1. PMT-E increased the body bending rate of N2 wild-type *C. elegans*

Treatment	Body bending rate/30sec (mean ± SD)			
	D4	Change	D8	Change
Control	11.50 ± 0.08		7.64 ± 0.19	
PMT-E	13.61 ± 0.06	18.35% ****	10.67 ± 0.21	39.66% ***

Table S2. PMT-E increased the pharyngeal pumping rate of N2 wild-type *C. elegans*

Treatment	Pharyngeal pumping rate /15sec (mean ± SD)			
	D4	Change	D8	Change
Control	51.60 ± 1.07		41.64 ± 0.43	
PMT-E	57.76 ± 1.13	11.94% *	50.02 ± 0.81	20.12% ***

Table S3. Effect of PMT-E on oxidative stress resistance of different strain *C. elegans*

Strain	Treatment (μg/mL)	Mean lifespan ± SEM (Hours)	Number of worms	Percentage change	*p* value
N2	Control	103.45 ± 1.72	171		
	PMT-E	118.72 ± 3.52	191	14.76%	0.0176
daf-16 (mu86)	Control	46.41 ± 1.41	179		
	PMT-E	43.12 ± 2.75	181	-7.09%	0.3467
sir-2.1 (ok434)	Control	71.62 ± 2.53	149		
	PMT-E	74.89 ± 3.07	145	4.57%	0.4563
skn-1 (zu135)	Control	84.65 ± 3.23	171		
	PMT-E	73.73 ± 3.69	168	-12.90%	0.0898

Table S4. List of primers used in *C. elegans*

Gene	Forward primer	Reverse primer
act-1	CCAGAAGAGCACCCAGTC	TGATGTCACGGACGATTT
daf-2	GCCCGAATGTTGTGAAAACT	CCAGTGCTTCTGAATCGTCA
daf-16	ATCGTGTGCTCAGAATCC	ATGAATATGCTGCCCTCC
sod-3	AGAACCTTCAAAGGAGCTGATG	CCGCAATAGTGATGTCAGAAAG
sir-2.1	TGGCTGACGATTCGATGGAT	ATGAGCAGAAATCGCGACAC
skn-1	CACGCCGTCAGCGAAGTA	ATGCTCGGTGAGTATTGG
gst-4	ACCAGCCCGTGATGATTTCT	ATCCTTTCTTGTTGCCACGT

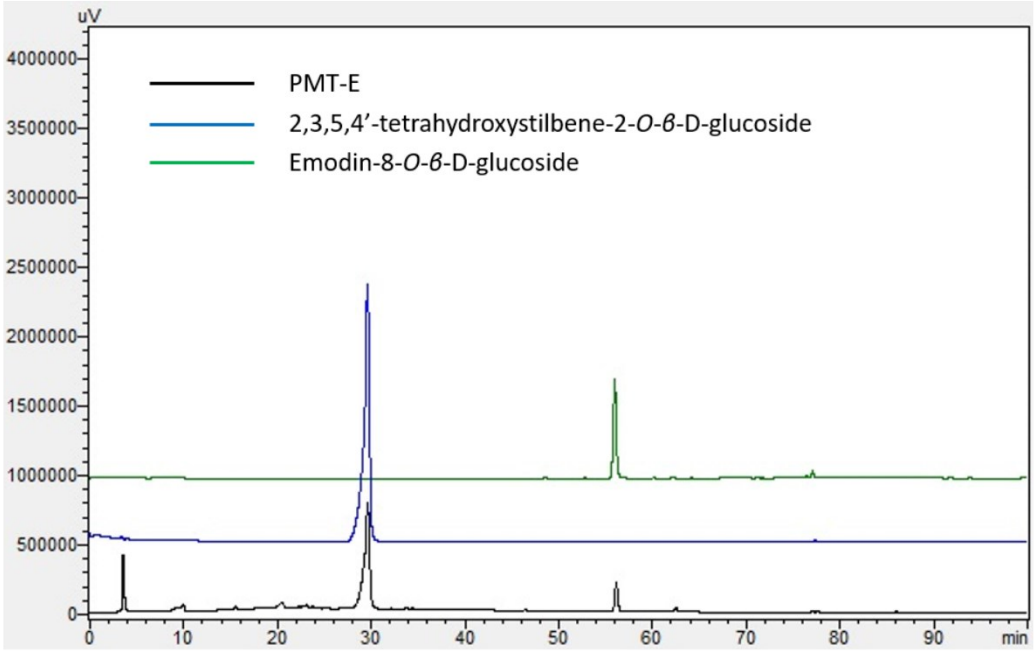
Table S5. PMT-E ameliorated Aβ-induced paralysis of transgenic *C. elegans* strain CL4176

Strain	Treatment (μg/mL)	PT_{50}	Number of worms	Percentage change	*p* value
CL4176	Control	3.77 ± 0.13	87		
	PMT-E	5.20 ± 0.25	112	37.93%	0.0073

Table S6. PMT-E enhanced chemotaxis behavior in neuronal Aβ-expressing strain CL2355

Strain	Treatment (μg/mL)	CI	Number of worms	*p* value
CL2355	Control	-0.10 ± 0.03	222	
	PMT-E	0.15 ± 0.04	182	0.0074

A



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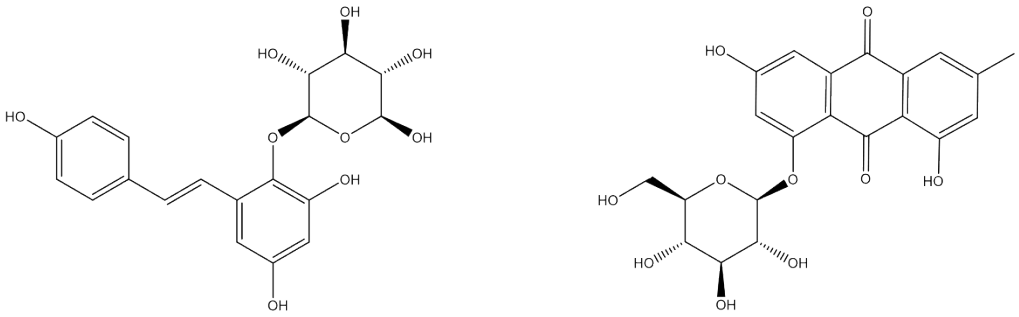

2,3,5,4'-Tetrahydroxystilbene-2-*O*-*β*-D-glucoside (TSG)

Emodin-8-*O*-*β*-D-glucoside (EG)

Fig. S1. Identification of bioactive compounds in PMT-E. (A) HPLC chromatogram of PMT-E and the reference standards of 2,3,5,4'-Tetrahydroxystilbene-2-*O*-*β*-D-glucoside (TSG), Emodin-8-*O*-*β*-D-glucoside (EG). (B) The structure of the TSG and EG.

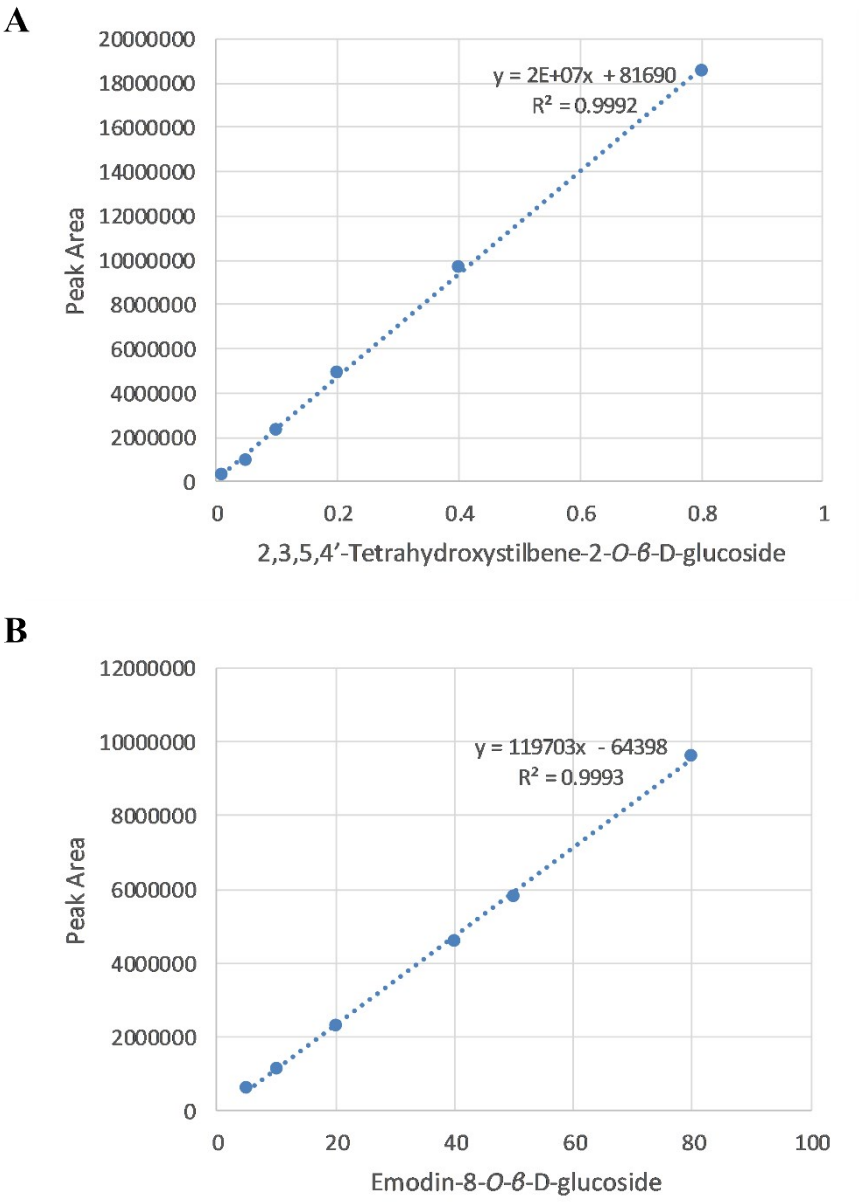

Fig. S2. The HPLC standard curves of two compounds. (A) The HPLC standard curve of 2,3,5,4'-Tetrahydroxystilbene-2-*O*-*β*-D-glucoside. (B) The HPLC standard curve of Emodin-8-*O*-*β*-D-glucoside. Approximately 50 μg/mL of PMT-E contained 11.5 μg/mL TSG and 0.5μg/mL EG. $R^2 > 0.999$ was accepted in HPLC standard curves drawing.

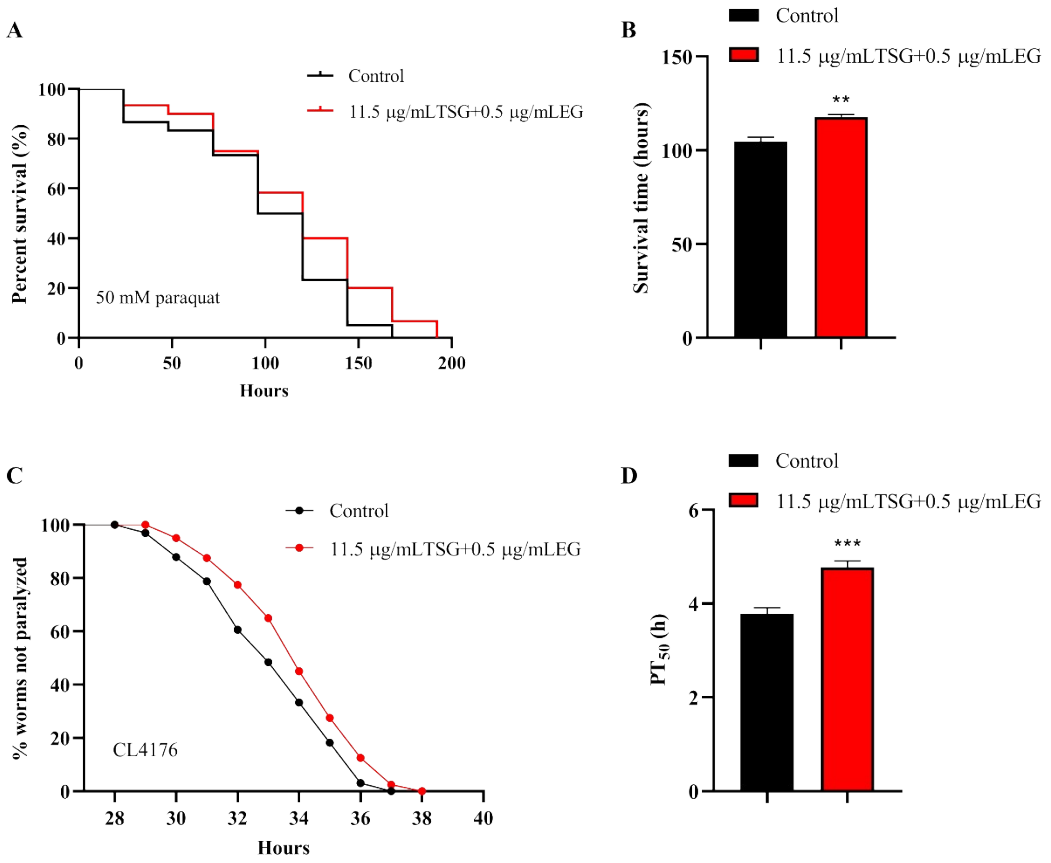

Fig. S3. Major chemical components of the PMT-E increased oxidative stress resistance and delayed Aβ-induced paralysis. (A, B) N2 worms were treated with or without combination of two compounds (11.5 μg/mL TSG and 0.5 μg/mL EG) and then exposed to 50 mM paraquat. The surviving nematodes were scored daily. (C, D) Neuroprotective effects of combination in transgenic strain CL4176. Graphs represent mean ± SD. * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

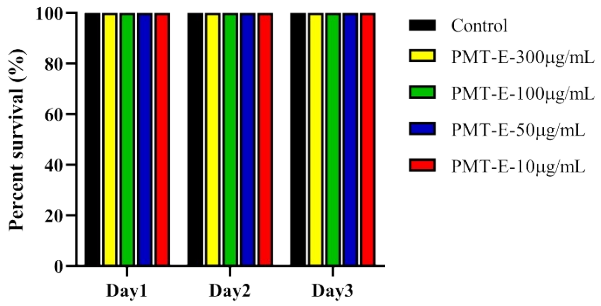

Fig. S4. Percent survival of N2 worms at 20 °C in 96-well plates containing 0, 10, 50, 100 and 300 μg/mL PMT-E.