



Correction to: Selenite-mediated production of superoxide radical anions in A549 cancer cells is accompanied by a selective increase in SOD1 concentration, enhanced apoptosis and Se–Cu bonding

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In the original article there were errors in the methods section. Thus, within Table 1: (i) the primer sequence pair for SOD-2 was incorrectly cited; (ii) the primer sequence pair used for SOD 1 was incorrect and did not target the gene of interest. Additional experiments were performed with correctly designed SOD1 primer pair and the outcomes documented here.

The following table now contains corrected primer sequence pairs.

Expression of the target gene products for SOD1 has been validated by independent additional experimentation using new cell samples treated under identical conditions as described in the original paper. Conclusions drawn from the

gene analyses were unaltered by these errors and were supported by independent assessment of SOD-1 protein expression and SOD total activity in the original paper—refer to (Fig. 6c; reproduced in the new figure) and original Fig. 7.

The following figures and tables replace outcomes originally reported.

New Fig. 6 and Tables 1 and 2

Gene expression and antioxidant activity

Next, the expression of SOD1, its corresponding activity and its cellular distribution in selenite-treated cells were investigated. In response to treatment with 5 μM selenite, the relative SOD1 mRNA levels increased two-fold after 24 h (Fig. 6b). Concomitant with increases in O_2^- levels, total SOD activity increased slightly after 4 h and 6 h and there

The original article can be found online at <https://doi.org/10.1007/s00775-014-1113-x>.

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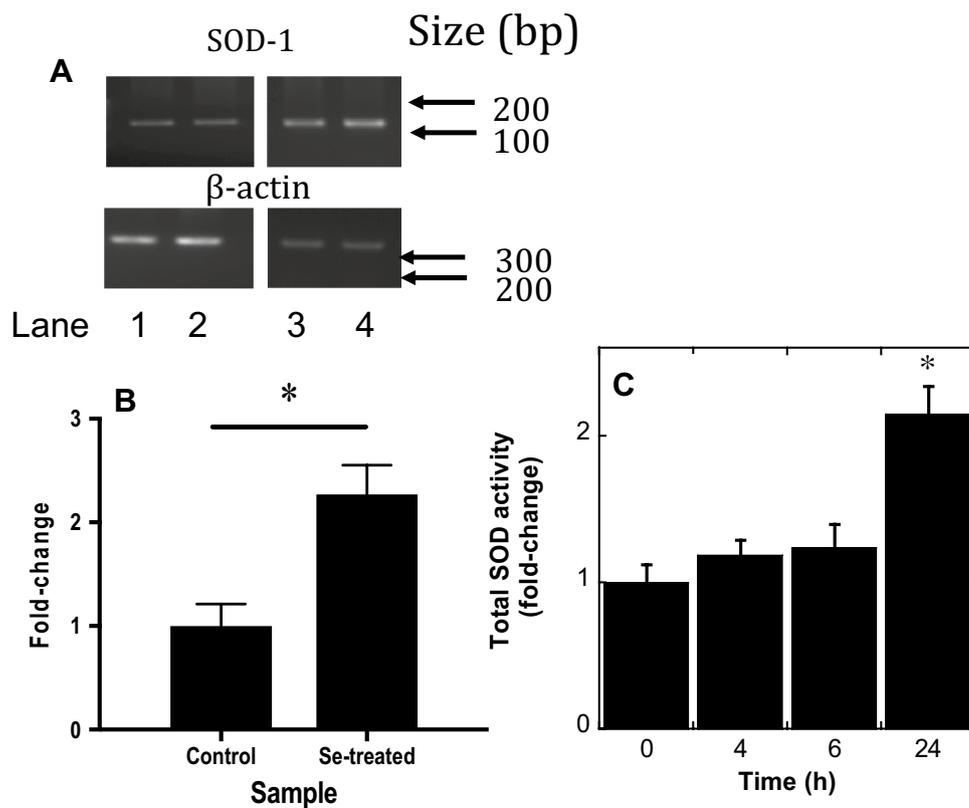
² The Discipline of Pathology, The University of Sydney, Sydney, NSW 2006, Australia

Corrected Table 1 Sequences of primers used for mRNA detection by RT-PCR

Gene	Sense primer	Anti-sense primer	Annealing temperature (°C)
β -Actin	5'-GGA CTTC GAGCAAGAGATGG-3'	5'-AGCACTGTGTTGGCGTACAG-3'	62
SOD1	5'-GGTGTGGCCGATGTGTCTAT-3'	5'-CCTTTGCCCAAGTCATCTGC-3'	60
SOD2	5'-CTGGACAAACCTCAGCCCTA-3'	5'-CTGATTTGGACAAGCAGCAA-3'	60
HO-1	5'-GAGATTGAGCGCAACAAGGA-3'	5'-AGCGGTAGAGCTGCTTGAACT-3'	55
Catalase	5'-ACATGGTCTGGGACTTCTGG-3'	5'-CAAGTTTTTGATGCCCTGGT-3'	60
GPx-1	5'-TGAGAAAGTGCAGGTGAATG-3'	5'-AACACCGTCTGGACCTACCA-3'	60

Primers were designed using a BLAST search of protein databases and converting sequence data to the primary DNA sequence. Primers were designed using this consensus sequence with specific fragments between 100 and 200 bp chosen for each of the genes of interest

Corrected Fig. 6 SOD1 expression is upregulated and SOD activity is increased in selenite-treated cells. A549 cells were incubated with 5 μ M selenite, harvested and probed for SOD1 gene expression and total SOD activity. **a** Representative SOD1 gene expression normalised to the corresponding β -actin in the absence (lanes 1 and 2) and presence (lanes 3 and 4) of selenite for 24 h. Quantitative data representing mean \pm SD; $n=3$ experiments each run in duplicate. Data represent mean \pm SD; $n=4$ independent experiments. *Significantly different to the control; $P=0.012$



Corrected Table 2 Select mRNA levels in A549 cells before and after exposure to sodium selenite

	SOD1	SOD2	CAT	GPx1	HO-1
Incubation time 24 h					
Control	1.0 (0.2)	1.0 (0.3)	1.0 (0.1)	1.0 (0.1)	1.0 (0.3)
2 μ M Se	2.8 (0.2)*	1.3 (0.4)	1.0 (0.3)	0.7 (0.1)	0.9 (0.3)
5 μ M Se	2.3 (0.3)*	1.1 (0.2)	1.0 (0.4)	1.6 (0.4)*	0.9 (0.2)
Incubation time 72 h					
Control	1.0 (0.2)	1.0 (0.3)	1.0 (0.2)	1.0 (0.8)	1 (0.2)
2 μ M Se	2.2 (0.1)*	1.0 (0.2)	1.4 (0.5)	3.8 (1.3)*	1.4 (0.3)
5 μ M Se	2.4 (0.3)*	1.3 (0.4)	1.7 (0.8)	5.7 (0.2)*	0.9 (0.3)

^aGene regulation studies were performed as described in the “Methods” section. Data are expressed as mean \pm SD; $n=6$. Antioxidant stress elements (SOD1/2; CAT; HO-1 and GPx1) were measured and normalised against the corresponding β -actin housekeeping gene. The mRNA levels in each treatment group were then expressed as a fold-change compared to the vehicle-treated control (arbitrarily assigned unitary value)

*Different to the control; $P < 0.05$

was eventually a significant twofold increase in activity 24 h after selenite treatment (Fig. 6c). The levels of SOD1 mRNA remained elevated 72 h after addition of 5 μ M selenite; however, the fold increase was lower than that measured at the 24-h time point (Table 2). Similarly, exposure of the cells to 2 μ M selenite for 24 or 72 h enhanced the levels of SOD1 mRNA, although the absolute fold increase remained similar to that obtained with the higher selenite dose (Table 2).

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