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[@pone.0038765-CeylanAkdemir1]. Autophagy has been suggested as one of the mechanisms responsible for the degradation of misfolded proteins  
[@pone.0038765-Mizushima1]. Reducing expression of LC3, another marker for autophagy, has been shown to reduce cell death in *Drosophila* and mammalian cells exposed to lethal toxins [@pone.0038765-Sutton1], [@pone.0038765-Chen1]. In this study, overexpression of Atg8 leads to protective effects against the toxicity caused by TG or H<sub>2</sub>O<sub>2</sub>, suggesting that autophagy may function in cell protection against both kinds of insults. However, no significant differences between the TG and H<sub>2</sub>O<sub>2</sub> groups were observed in autophagic flux, which is consistent with the observation of no significant differences between the TG and H<sub>2</sub>O<sub>2</sub> groups in both LC3B immunoreactivity and in the number of autophagic vacuoles. This further supports our view that Atg8 does not influence autophagic flux [@pone.0038765-Nishida1]. The mechanism by which Atg8 protects cells from oxidative stress, especially by increasing the antioxidant levels of cells, is not clear. One possibility is that Atg8 increases synthesis of the antioxidant enzymes thioredoxin [@pone.0038765-Lin1], [@pone.0038765-Wu1]. It is also possible that expression of Atg8 modulates the activity of the antioxidant system. In this study, there was a higher transcription level of the antioxidant genes *catalase* and *Sod1*, in the TG group than in the control group ([Fig. 6](#pone-0038765-g006){ref-type="fig"}). The observation of down-regulation of the antioxidant genes in the *atg8* mutant in our previous study [@pone.0038765-Jiang1] suggests that the antioxidant effect of Atg8 is mediated by its transcriptional regulation of the antioxidant genes. Other possibilities are that Atg8 influences the processes of detoxification. The *Gst* gene is the main enzyme for detoxification