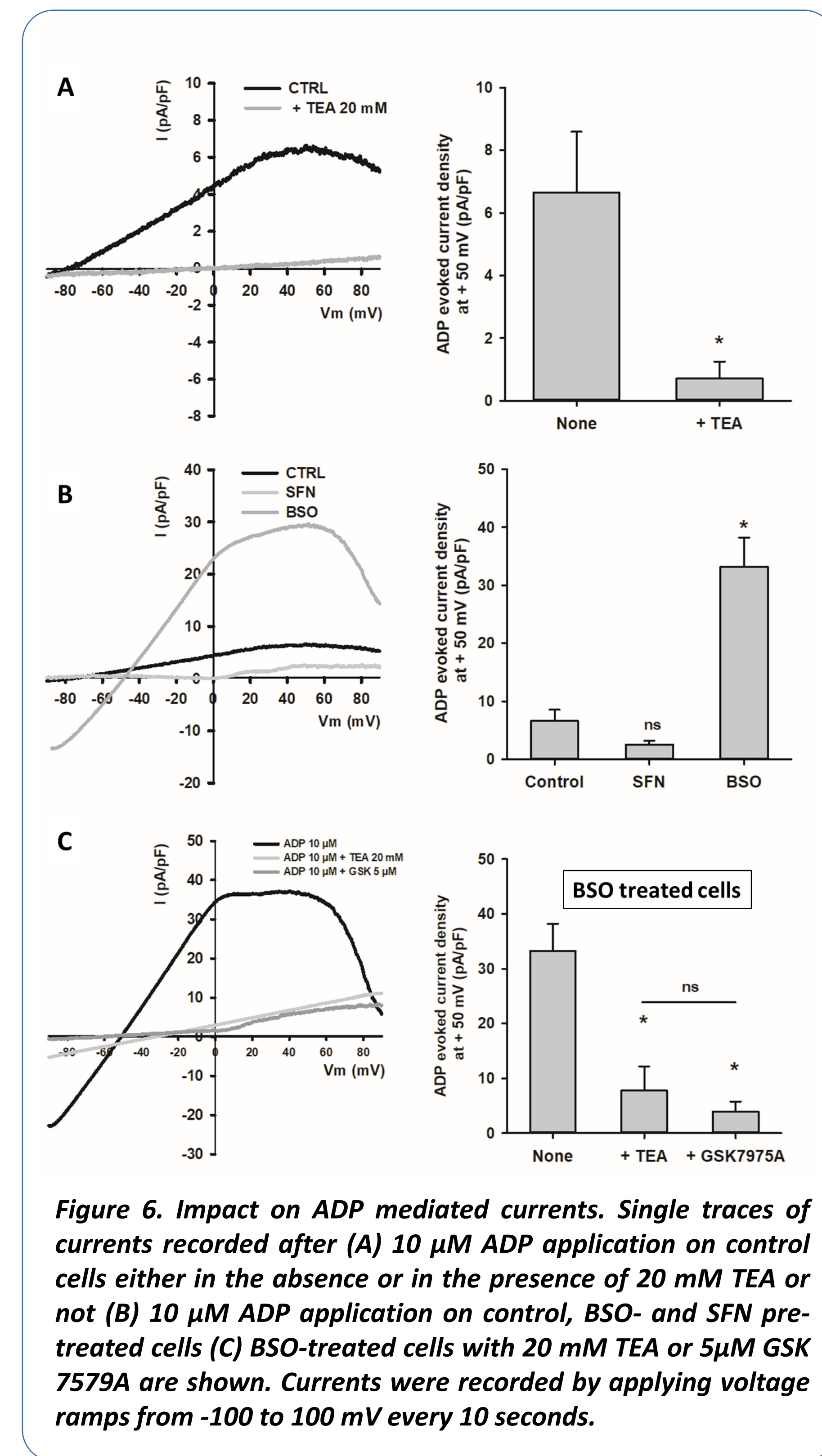
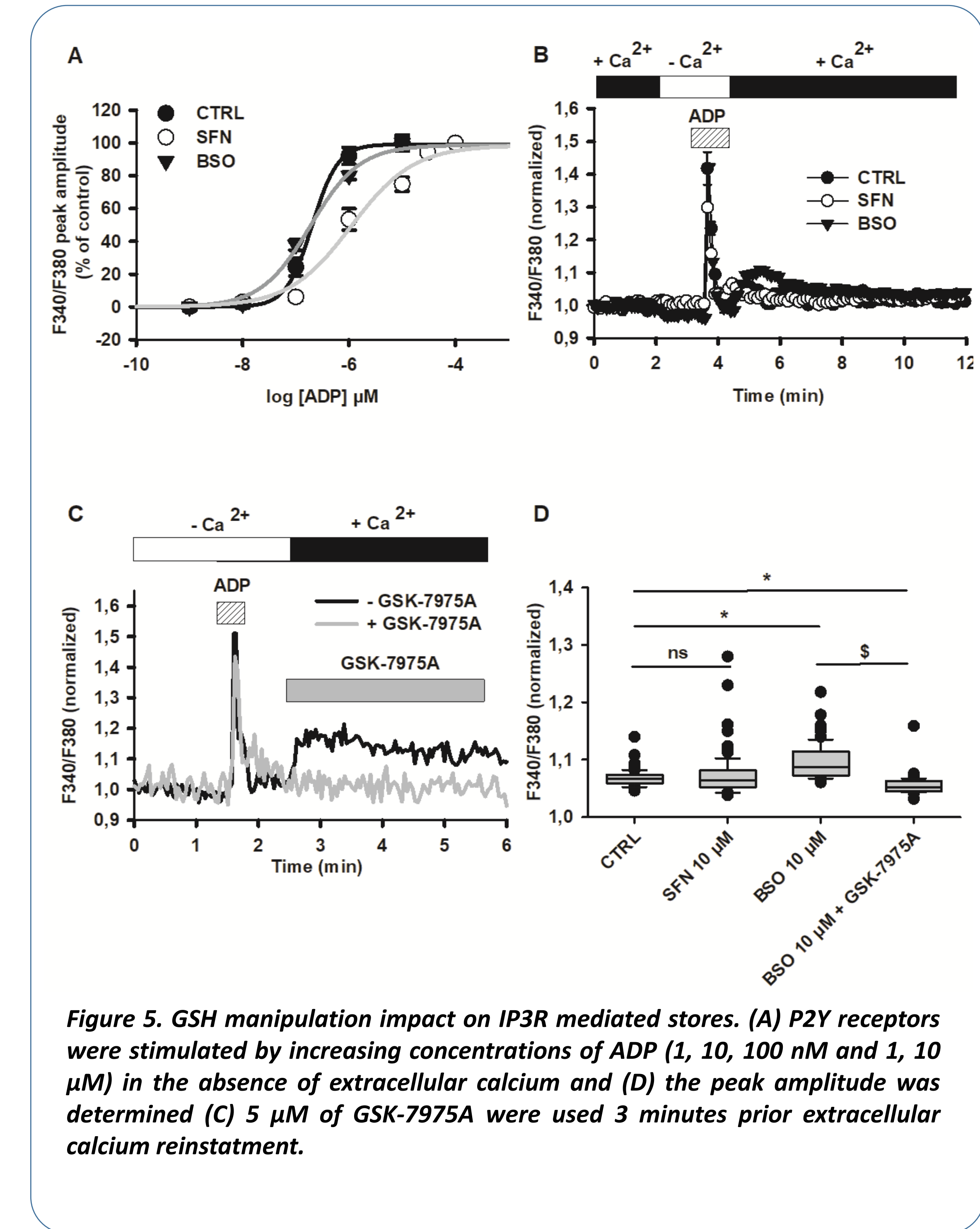
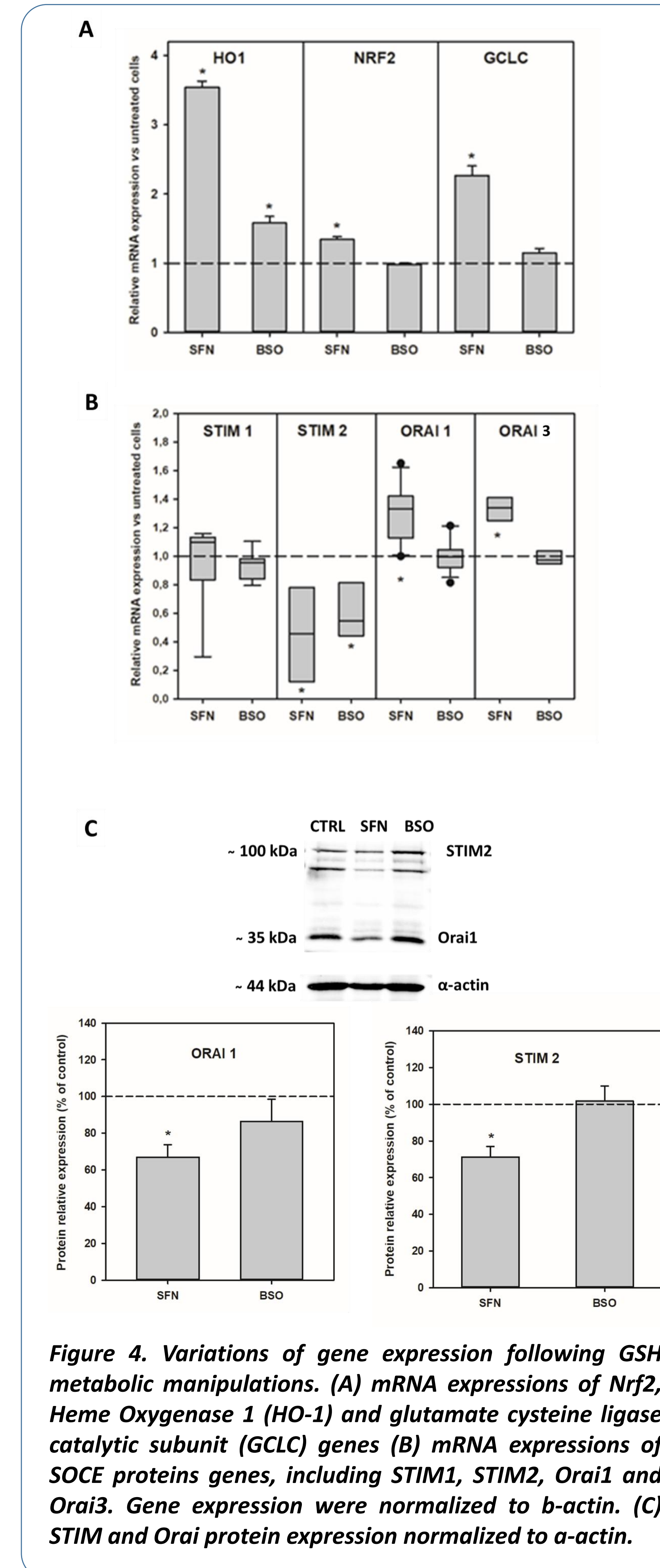
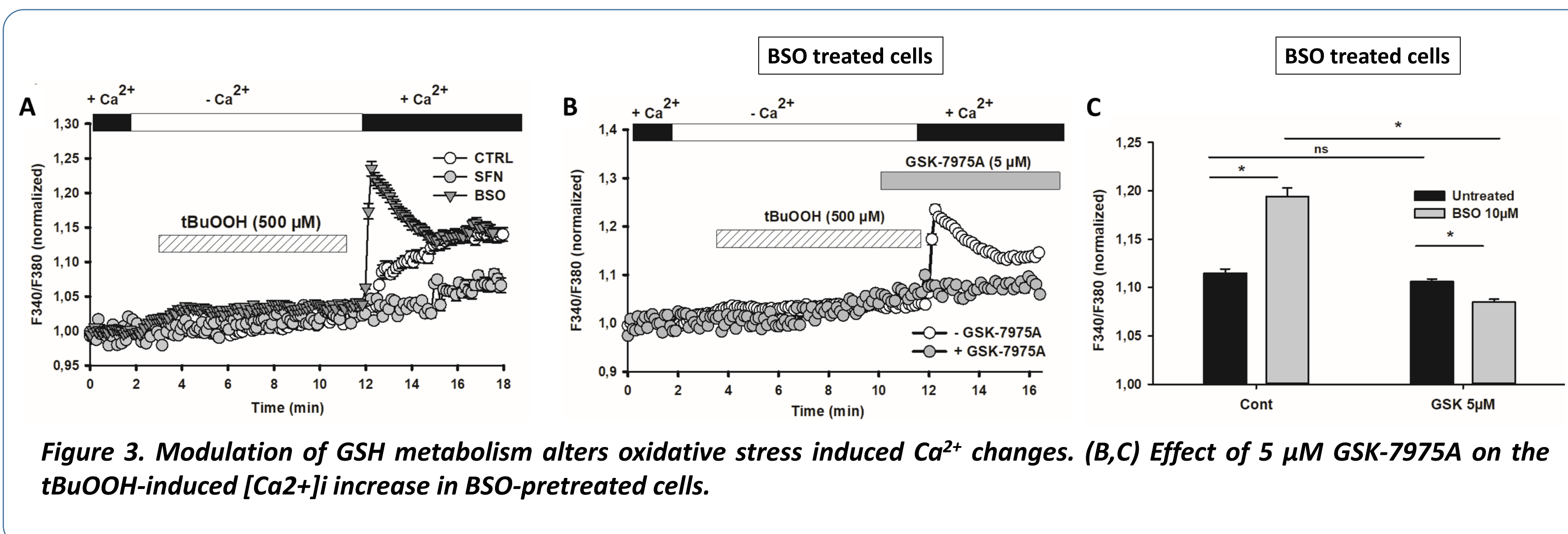
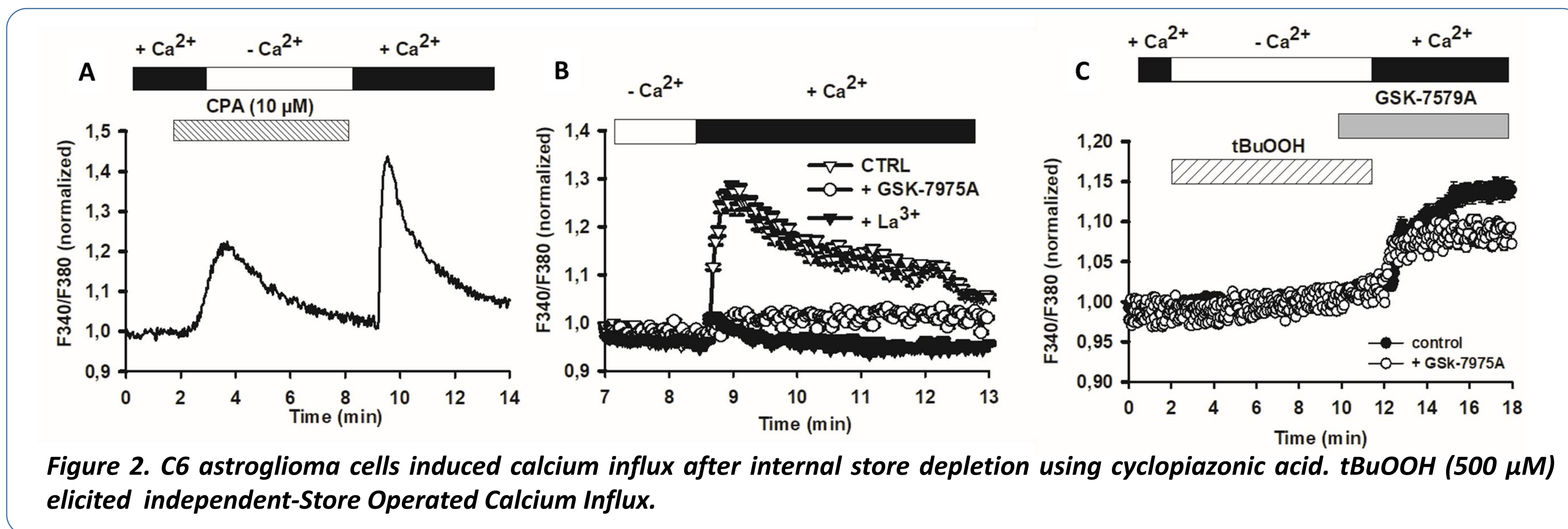
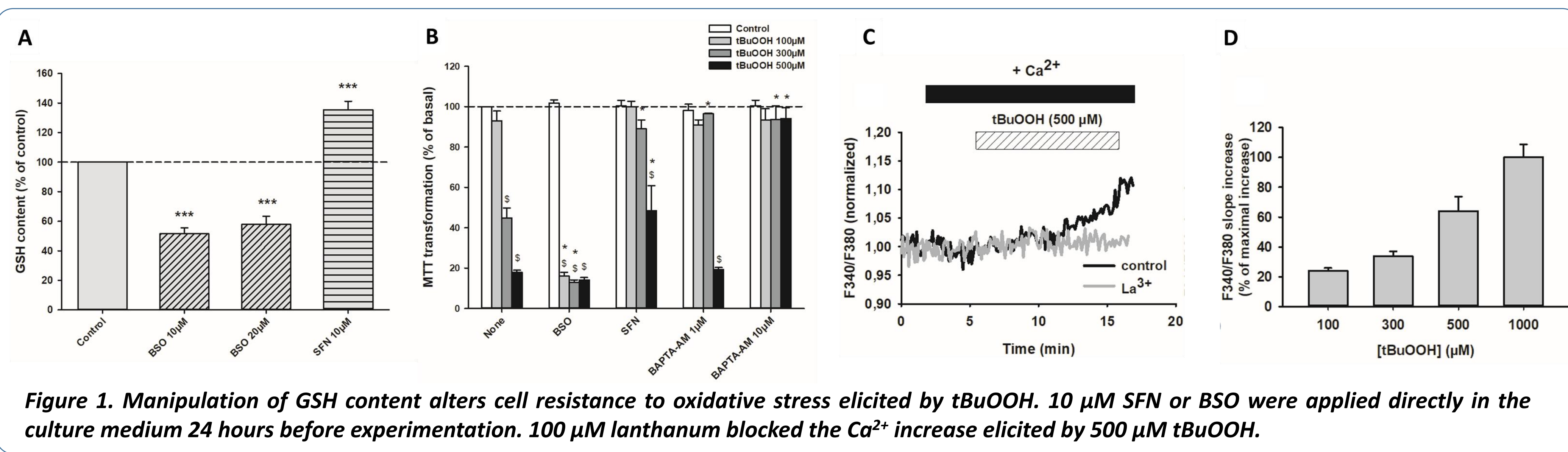
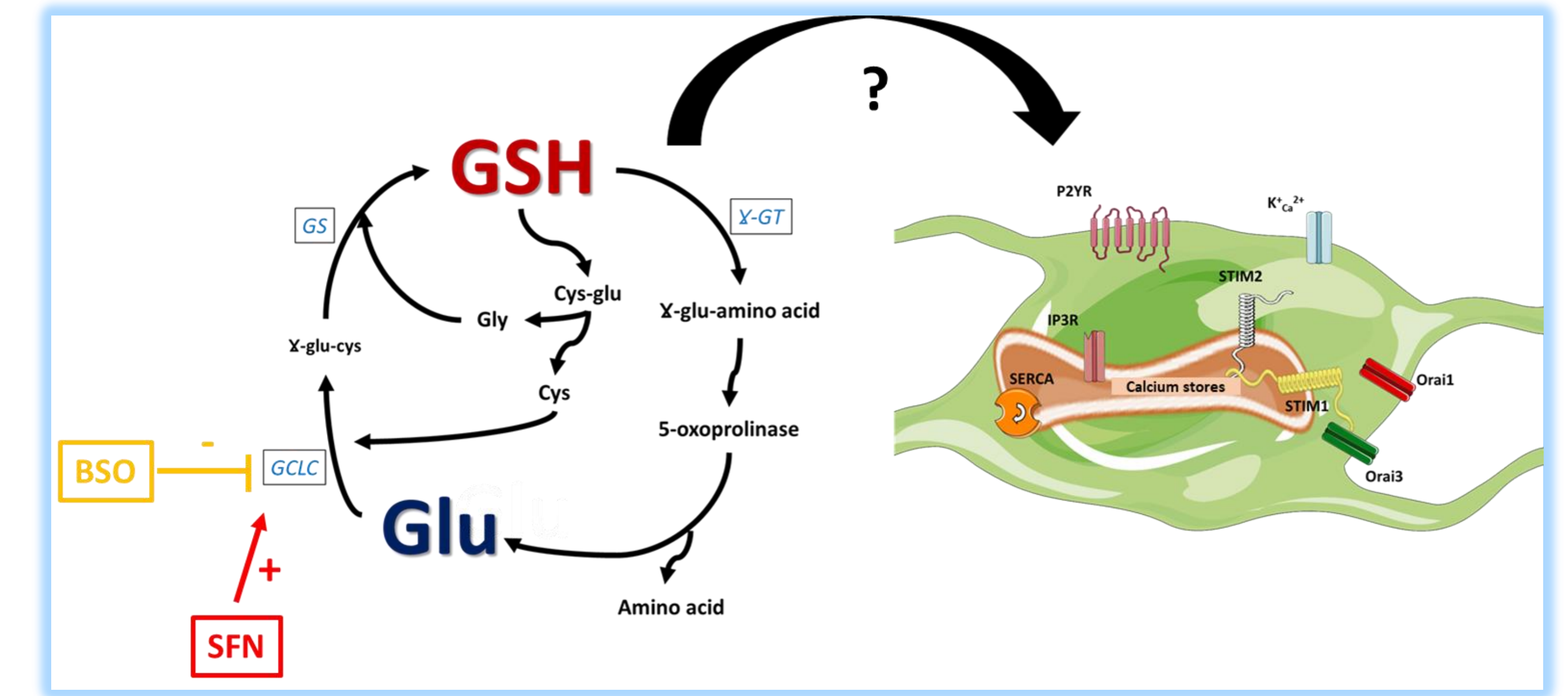


N. Mokrane<sup>1</sup>, T. Cens<sup>1</sup>, P. Charnet<sup>1</sup>, C. Ménard<sup>1</sup>, J. Guiramand<sup>1</sup>, M. Rousset<sup>1</sup>, C. Cohen-Sola<sup>1</sup>, J. Rousset<sup>1</sup>, M. Vignes<sup>1</sup>

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Oxidative stress is an aggravating factor of all neurological disorders, including neurodegenerative diseases. It is due to an exaggerated accumulation of reactive oxygen species (ROS) concomitant to decreased reduced glutathione (GSH) levels; the main antioxidant of brain cells produced by astrocytes in large amounts. Cell death triggered by oxidative damage results from intracellular calcium overload which is deleterious to cell activity. The mechanisms involved in these calcium increases are not fully understood. Here, we have thus investigated whether GSH could protect neurons against oxidative stress through the modulation of calcium homeostasis. For this, GSH cell content was manipulated by modulating its metabolism. To investigate the link between GSH content and calcium homeostasis, we pre-treated rat astrogloma cells (C6) with sulforaphane (SFN) to increase intracellular GSH levels, or buthionine sulfoximine (BSO) to decrease them, during 24 hours for both treatments.



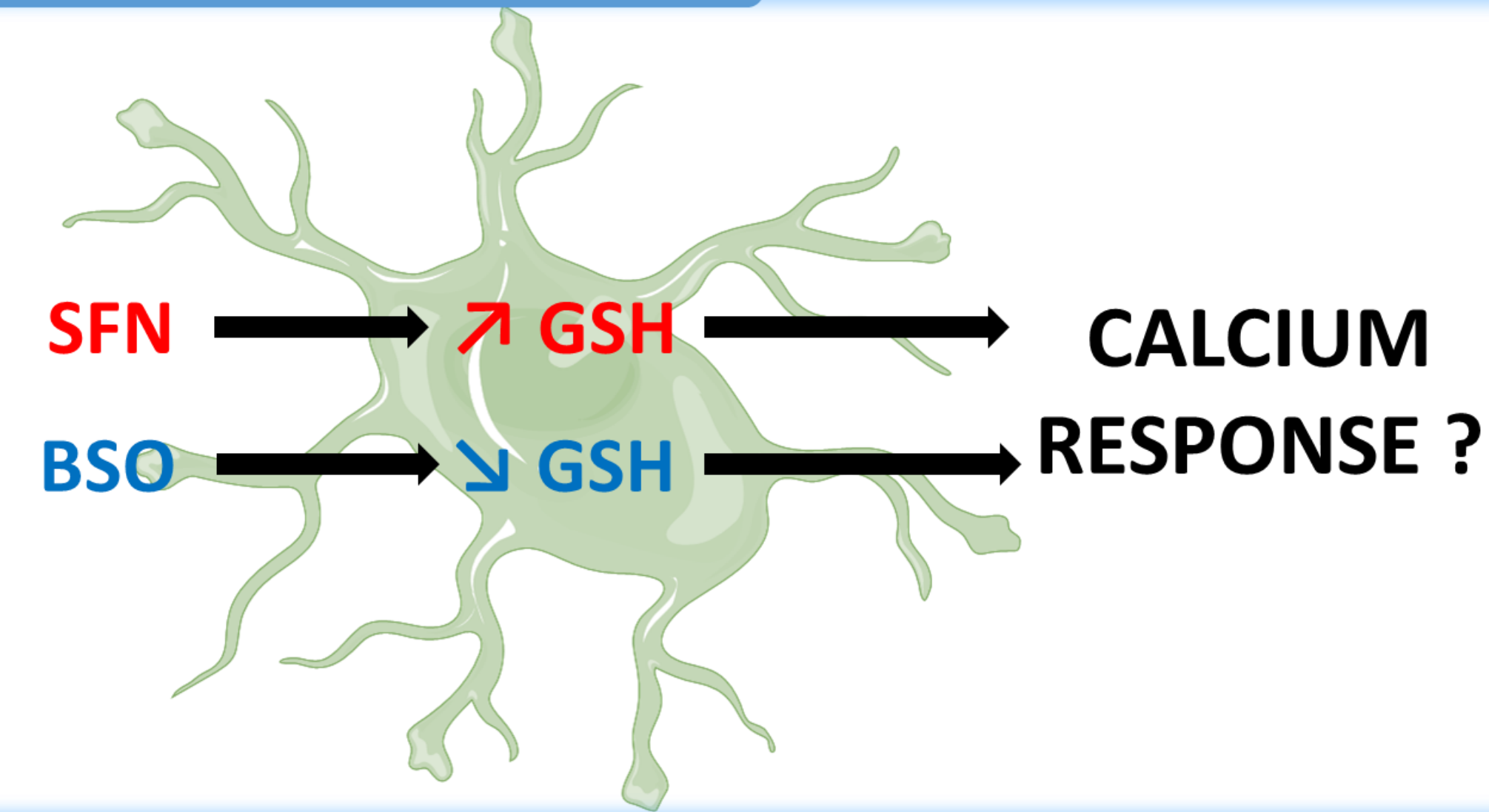
We conclude that GSH cell content changes impact intracellular Ca<sup>2+</sup> homeostasis in C6 glioma cells likely via sensitization/desensitization mechanisms of Calcium Release Activated Channels (CRAC). GSH depletion due to oxidative stress regulation could thus contribute to the alterations of Ca<sup>2+</sup> homeostasis in astrocytes under pathological conditions.

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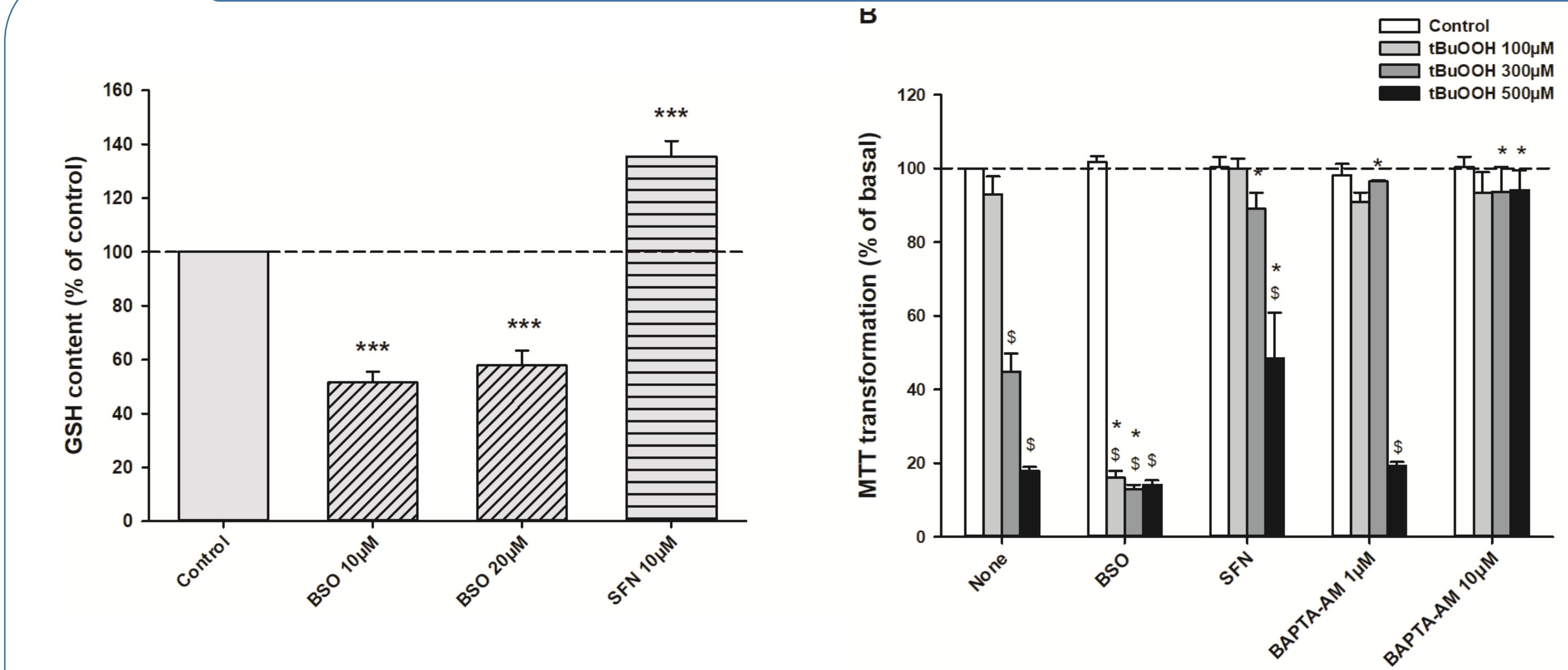
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### Experimental strategies



### 1. Modulation of GSH cycle synthesis alters cells sensitivity to tBuOOH toxicity



### 3/ GSH levels impacts IP3R response

