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Sustained autophagy induction by the stress-resilience mediator neuropeptide Y in hippocampal and cortical cultures

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Autophagy is one of the degradation routes of eukaryotic cells that results in the recycling of macromolecular constituents. In neurons, with their elaborate structure, autophagy has a pivotal role in organising proteins and organelles turnover in between distant cell districts in order to maintain the homeostasis of such complex cells. One of the candidates that has been recently found to induce autophagy in primary hypothalamic and cortical co-cultures is neuropeptide Y (NPY), a prominent mediator of stress resilience in the nervous system.
To understand the role of autophagy in stress-resilience we analysed common autophagic markers p62 and LC3 via immunoblotting and immunocytochemistry and we now demonstrated that NPY stimulates autophagy not only in cortical but also in hippocampal neuronal primary cultures. Mechanistically, in rats cortical neurons, we confirmed via immunoblotting that the NPY increase of autophagy is done through both the activation of NPY Y1 and Y2 receptors.
Interestingly, we found that the elevation of autophagy levels, monitored by LC3, is sustained for 24h and it then goes back to normal levels only 48h after NPY wash-out. This dynamic is observed even if the protein synthesis is blocked in combination with NPY.
Understanding the role of the increased autophagy levels via NPY and its mechanics could give insight into how animals exploit autophagy to find better ways to cope with stress and elucidate novel targets for therapies against post-traumatic stress disorders (PTSDs).