Accepted Manuscript

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PII: S0006-291X(17)32130-7

DOI: 10.1016/j.bbrc.2017.10.145

Reference: YBBRC 38762

To appear in: Biochemical and Biophysical Research Communications

Received Date: 23 October 2017

Accepted Date: 27 October 2017

Please cite this article as: L. Zhan, J. Li, B. Wei, Autophagy in endometriosis: Friend or foe?, *Biochemical and Biophysical Research Communications* (2017), doi: 10.1016/j.bbrc.2017.10.145.

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Autophagy in endometriosis: friend or foe?

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Abbreviations: 3-MA, 3-methyladenine; ERK, extracellular signal-regulated kinase;

MIS, Mullerian inhibiting substance; mTOR, Mammalian target of rapamycin; NF-κB,

nuclear factor-k-gene binding κ; OH-1, heme oxygenase-1; TRPV, transient receptor

potential vanilloid type

Abstract

Endometriosis is a chronic, estrogen-dependent disease and characterized by the

implantation of endometrial glands and stroma deep and haphazardly into the outside

the uterine cavity. It affects an estimated 10% of the female population of

reproductive age and results in obvious reduction in health-related quality of life.

Unfortunately, there is no a consistent theory for the etiology of endometriosis.

Furthermore, the endometriosis is hard to diagnose in early stage and the treatment

methods are limited. Importantly, emerging evidence has investigated that there is a

close relationship between endometriosis and autophagy. However, autophagy is a

friend or foe in endometriosis is puzzling, the precise mechanism underlying

autophagy in endometriosis has not been fully elucidated yet. Here, we provide an

integrated view on the acquired findings of the connections between endometriosis

and autophagy. We also discuss which may contribute to the abnormal level of

autophagy in endometriosis.

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