## H122 Thanatophagy in Brain and Heart Tissues

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After attending this presentation, attendees will learn how thanatophagy occurs in a Postmortem Interval (PMI) -dependent manner in brain and heart tissues of heart attack victims. Specifically, attendees will learn what autophagy marker is actively expressed after a human dies.

This presentation will impact the forensic science community and practitioners interested in thanatophagy and its relationship to PMI determination and cadaver tissues.

Postmortem autophagy, also known as thanatophagy, commences after a human dies and endures as a function of the time of death. The progression of thanatophagy is a possible technique to estimate the time lapse since death. Approximately 30 various genes have been identified in mammalian autophagy. Among those gene products, Beclin-1, the mammalian orthologue of yeast Atg6, is a critical protein involved in the formation of autophagy-regulating macromolecular complex.

This study analyzed whether thanatophagy would increase proportionally compared with various postmortem intervals. Heart and brain tissues from four whole cadavers at different time frames of death were collected from autopsies of cardiac arrest victims in criminal cases. The tissues were analyzed using Western blot techniques and densitometry. First, the levels of LC3-I and LC3-II in cardiac tissues collected from different times of death, namely 6h, 16h, 36.5h, and 58h, were measured. It was found that thanatophagy occurred in a manner that correlated with the time of death from data demonstrating the levels of LC3 II expression along with other autophagy proteins: p62, BNIP3, Beclin-1, and Atg7. Next, the levels of an autophagy adaptor protein, p62, as an alternative marker of autophagy flux, which is co-degraded with LC3-II, were measured. Intriguingly, there was no observed decrease of p62 levels in either cardiac and brain tissues. Remarkably, BNIP3, a potent inducer of autophagy, was reduced as the time of death increased in the heart but was elevated in the brain; however, there was no expression of BNIP3 at 58h after death in either brain and cardiac tissues. Similar to LC3-II, both Beclin-1 and Atg7 increased as a function of time of death in cardiac tissues. Beclin-1 reached its maximum at 36.5h after death and continued expression until 58h after death.

This study suggests that although thanatophagy in the heart may occur in association with Beclin-1, brain thanatophagy appears separate from Beclin-1. Therefore, the current study reveals for the first time that thanatophagy occurs in the heart and the brain of cadavers in a manner dependent on the time lapse since death.

This study provides a potential insight into thanatophagy as a new method for precise determination of the time of death.

**Thanatophagy, Cadaver, Postmortem Interval**