THE COEXISTENCE OF AGING AND OBESITY ADDITIVELY INCREASE CEREBRAL APOPTOTIC AND INFLAMMATORY SIGNALINGS

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BACKGROUND: Recent studies suggest that obesity also affects brain function and is a risk factor for some degenerative brain diseases. Very limited information regarding the brain apoptosis and inflammation in aging obesity was available. The purpose of this study was to evaluate whether the aging and obesity will additively increase apoptotic and inflammatory signaling.

METHODS: Eleven young lean (YL), 10 young obese (YO), 11 old lean (OL), and 11 old obese (OO) Zucker rats were investigated at 4 and 14 months old. The brain tissues were performed by positive TUNEL assays and Western blotting assays.

RESULTS: Cerebral TUNEL-positive cells in the YO group was slightly more than YO, whereas those in the OO group were more than OL group. Bad and Bcl2 were similar between YL and YO group but IGF-1 and p-Akt were enhanced in YO whereas caspase-9 and caspase-3 in YO was still slightly significantly more than YL. In contrast, mitochondria-dependent apoptotic pathways (Bad, 1/Bcl2, Bad/Bcl2, active caspase-9, and active caspase-3) were remarkably increased in OO group, compared with OL. The inflammatory relative proteins (TLR4, IKK, p-IκB, p-NFκB) level were significantly higher in OO group compared with OL group whereas IKK, p-IκB, p-NFκB level were significantly higher in YO group compared with YL group.

CONCLUSION: The cerebral mitochondria-dependent apoptotic pathways and inflammatory relative proteins were further activated in the aging obesity. Less cerebral apoptosis in young obesity may caused by much active compensated survival pathway, such as IGF-1 and p-Akt. The findings may provide a possible mechanism for developing cerebral apoptosis in aging obesity.

Key words: Cerebral, apoptosis, inflammation, elderly, obese