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Opinion

Homozygous Long-Lived Ames Dwarf Mice

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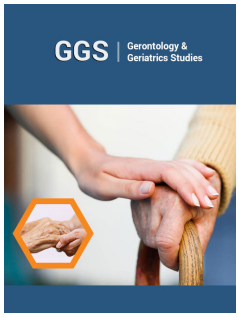

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Introduction

Homozygous long-lived Ames dwarf mice display two different physiological characteristics. On one hand, they are pretty "resistant to cancer" relative to their wild-type or heterozygous siblings. On the other and, they are also "long-lived" relative again to their wild-type or heterozygous siblings. The most simplistic explanation of the existence of these two different characteristics in the homozygous long-lived Ames dwarf mice could be that the "cancer-resistant" characteristics cause the "long-lived" characteristics of these mice. However, this explanation is only statistical with no cause-and-effect biochemical or molecular biological mechanisms.

Biochemical and Molecular Biological Mechanisms

The author of this opinion article has been studying the "cancer-resistant" characteristics of these mice for many years. But the author has never studied the "long-lived" characteristic of these mice. Based on the author's study of "cancer-resistant" characteristics of these mice, the following biochemical and molecular biological mechanisms could be proposed for the "long-lived" characteristics of these mice. First, expression of the protein called p27kip1 is significantly increased in these mice. p27kip1 is a cell cycle repressor protein expressed primarily in the late G1 phase of the cell cycle. When expression of p27kip1 is significantly increased in the late G1 phase, it closes the flood gate between the G1 phase and S phase thereby increasing the total length of each cell cycle. Any other cell cycle regulatory proteins are neither increased nor decreased in these mice.

Second, these mice are the best rodent models of caloric restriction, and the serum levels of glucose and insulin are significantly decreased in these mice. When serum levels of glucose and insulin are significantly decreased, 5'-end caps located at the 5'-upstream nucleotide position -575 of the messenger RNA of p27kip1 is compromised. This in turn increases the expression of p27kip1 mRNA by increasing the attachment of the 40S ribosome to the polypyrimidine tract located at the 5'-upstream internal ribosome entry sites of the p27kip1 mRNA thereby increasing the expression of p27kip1 protein.

Conclusion

With this biochemical and molecular biological mechanisms, the life span significantly increases simply by increasing the length of the time the cells spend in each cell cycle. However, it does not increase the life span by increasing the total number of the cell cycle.