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Termin 3

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Abstract

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1. Introduction

Studies indicate that invertebrate have an extended lifespan when their IGF-1/insulin signaling pathway was reduced. A more recent study on mice found that female mice lived 33% longer with a similar reduction. Bokov A. et al[1] chose to further investigate the claim that a change in IGF-1/insulin signaling can affect longevity.

2. Scientific hypothesis of the paper and justification of the chosen methods

Bokov A. et al. hypothesized that having a haplo-insufficiency of the Insulin-like Growth Factor-1 Receptor (*lgf1r*$^{+/-}$) would increase the lifespan of mice (vertebrate).

Unlike previous studies, Bokov A. et al. used larger sample populations and a more controlled environment combined with more rigorous criteria regarding the evaluation of longevity. They did this in order to make more accurate distinctions between a relevant result and one that might be misleading because of unrelated factors.

3. Results and conclusions

The mice which were haplo-insufficient of the IGF1 receptor showed very little difference in terms of longevity to the control. Male mice showed no improvement and female only by a few percent (<5%). Other results of the study showed glucose intolerance in male elders with the haplo-insufficiency while female subjects became more tolerant to oxidative stress. All *lgf1r*$^{+/-}$ mice became insulin resistant with age.

The conclusions that Bokov A. et al. drew was that *lgf1r*$^{+/-}$ most likely will not be able to assist in longevity if not combined with a specific environmental factor of which *lgf1r*$^{+/-}$ is a counteractor.

4. Possible weakness of the study

Bokov A. et al. used mice with a different genome compared to earlier studies. Perhaps this gene-variation could account for differing results.

5. Potential ethical considerations related to the two papers

Conducting experiments on living things, in this case mice, spurs an ethical discourse to take into consideration. Both groups knowingly asked for particularly bred mice in order to affect their lifespan and very possibly their quality of life. Furthermore they manipulated the mice's environment to suit the means of the study.

6. Medical database search of relevance to the two papers

To find more relevant information on the topic I searched in PubMed and Cochrane using MeSH-terms such as 'IGF-1 receptor' and 'longevity' combined with free text such as 'mice'. Through these keywords I found relevant studies as well as the reviewed studies (Bokov and Holzenberger[2]).

I used the words 'longevity' and 'IGF-receptor' in order to find studies focused on the same area of research and then I combined them with 'mice' in order to narrow down what type of study design I was looking for. Without 'mice' for example I ended up with mostly studies concerning invertebrates changes in lifespan in response to IGF-1 pathway-modulation.

References

[1] Bokov A., Garg N., Ikeno Y., Thakur S., Musi N., DeFronzo R. et al. Does Reduced IGF-1R Signaling in *lgf1r*$^{+/-}$ Mice Alter Aging? PLoS ONE. 2011 May [cited 2011 Nov 23rd];6(11): e26891. doi:10.1371/journal.pone.0026891

[2] Holzenberger M., Dupont J., Ducos B., Leneuve P., Géloën A., Even P. et al. IGF-1 receptor regulates lifespan and resistance to oxidative stress in mice. Nature. 2003 Jan 9;421(6919):125-6. doi: 10.1038/nature01298