

Renew + Protect: Impact on Telomeres in Cultured Human Dermal Fibroblast Cells

Telomere length is an indicator of cellular age, and telomerase helps maintain the length of telomeres.

TELOMERES AND AGING

Telomeres are the protective caps at each end of cells' chromosomes that protect their DNA. Without telomeres, cells' genetic material may be lost during cell division.

Every time cells divide, a natural process with aging, their telomeres shorten until eventually, they become so short that cells lose their ability to divide properly. This status, called senescence, hampers necessary cellular regeneration and tissue repair, and is associated with cell death¹. Thus, telomere length is a marker of cellular aging².

Telomeres also shorten in response to external and internal stresses³, like oxidative stress attributed to normal bodily functions, such as breathing and inflammation, and disease associated chronic inflammation. Oxidative stress is caused by reactive oxygen containing molecules, that damage DNA, protein, and lipids, and is one of the main contributors to aging⁴. Therefore, oxidative stress and telomere shortening are key factors in the aging process.

Telomerase is an enzyme that helps maintain telomere length during cell division. It is mainly found in stem cells or quickly renewing/immature cells, but it's levels are very low or absent in most normal/adult cells¹. Therefore, without stimulating telomerase activity, telomere shortening is inevitable during normal cellular, and overall aging.

PRECLINICAL TRIAL

STUDY PURPOSE:

To evaluate the effect of Renew + Protect on telomerase activity and telomere shortening in human dermal fibroblast cells under normal and oxidative stress conditions.

PROCEDURE:

Cells were treated with Renew + Protect for a maximum of 8 weeks

Renew + Protect's potential to increase telomere length was determined by measuring the cells' telomerase activity throughout 72 hours of treatment under normal conditions (Figure 1), and it's potential to protect telomeres from shortening was determined by measuring telomere length as the cells aged and divided throughout 8-weeks under oxidative stress conditions (Figure 2).

The statistically significant difference in telomerase activity and telomere length between treated and untreated control cells was determined.



RESULTS

Telomerase Activity: Telomerase activity significantly increased in cells treated with the lowest dose of Renew + Protect compared to untreated cells at 6 hours. There was no difference detected between treated and untreated cells over the remaining 72 hours. Therefore, Renew + Protect temporarily stimulated telomerase activity, which counteracts shortening. It also suggests it activates telomerase early in a cell's life cycle, which may allow it to maintain telomere length longer overtime.

Telomere length: Cells treated with Renew + Protect, under oxidative stress conditions, had:

- Significantly longer telomeres and fewer critically short telomeres compared to the untreated control.
- Decreased telomere shortening rate, which became significant compared to the untreated control by week 8.

CONCLUSIONS

RENEW + PROTECT:

- Significantly slowed the rate of telomere shortening after 8 weeks during exposure to oxidative stress.
- May help cells maintain telomere length over-time by temporarily increasing telomerase activity.

In a cell culture study, Renew + Protect:

Temporarily increased telomerase activity, which may help maintain telomere length over-time

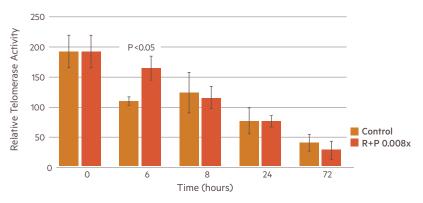


Figure 1: Telomerase activity of Renew + Protect (R+P) treated human fibroblasts compared to an untreated control under normal conditions.

Slowed telomere shortening during oxidative stress

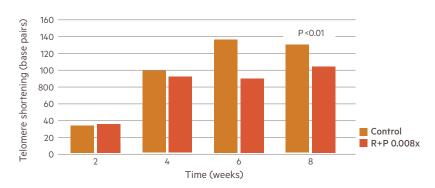


Figure 2: Telomere shortening rate in human dermal fibroblasts treated with Renew + Protect (R+P) vs. untreated controls under oxidative stress conditions.

References:

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