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Perspective

The Immortal Strand Hypothesis

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DESCRIPTION

Stem cells are the reason of tissue repairs and maintenance throughout the life of the organism. Mainly in tissues that undergo rapid and continuous turnover, such as the epithelial cells of gut and skin, cellular components of blood, there is a constant demand for stem cells to multiply to generate separated cells and in the process to self-renew. Assumed the high frequency of DNA replication errors resulting in genomic mutations, the chance of any stem cell or its determined progeny obtaining a sufficient number of serious mutations throughout a human life span that would result in cancer is very high were it not for two mechanisms: cell-cycle checkpoints and the ability to notice and repair such mutations.

Stem cells appear to have a compact, rather than an improved, DNA repair capacity compared with other somatic cells in the few populations in which it has been examined. This concludes that stem cells have the capacity to limit the accumulation of impulsive mutations or that, having acquired harmful mutations; they are more disposed to undergo senescence so as to decrease the risk of generating a malignant clone. The level to which stem cell functions are causally associated to the aging process or are determinants of the most life span of a species is a matter of debate, but the probable for stem cells or their progeny to acquire a malignant phenotype and thereby abbreviate an individual's life span is undoubted.

John Cairns put forth the "immortal strand hypothesis" in 1975 in considering mechanisms by which a stem cell population might limit the accumulation of replication-induced mutations. The hypothesis is founded on the fact that each newly formed chromosome contains of the older (grandparent) template strand and a newly synthesized (parent) strand that is likely to contain replication-related errors. Upon a succeeding round of replication, when both the grandparent and parent strands serve as templates for DNA replication, the resulting sister chromatids could, in theory, is different based on the age of the template. The hypothesis is that when the cell then divides, there exists a mechanism to sort all of the chromatids containing the grandparent templates to one daughter cell and all of the chromatids containing the parent templates to the other daughter cell. This would be an unequal cell division based solely on chromatid segregation according to template age. The hypothesis further proposes that this asymmetric cell division is related with stem cell self-renewal and that the new stem cell would receive all of the oldest templates and the other daughter, destined to differentiate and would inherit all of the younger templates with replication-induced mutations. In the extreme, a stem cell pool would retain, throughout the life of the organism, its original DNA strands that were generated when the cell population first arose during growth, and these immortal strands would continue to serve as templates forever. Thus, in theory, the original genetic code would be optimally conserved in the stem cells and replication-related mutations would be reserved to a minimum.

The immortal strand hypothesis posits that the propensity of stem cell compartments to give rise to cancer in later life can be minimized if stem cells, during the process of self-renewal, retain those DNA strands with the fewest mutations acquired during DNA replication. In this Essay, I explore evidence in support of the hypothesis, the biological implications, and the key questions that remain to be answered experimentally to address the fundamental tenets of the hypothesis.

The immortal strand hypothesis can be divided and tested on numerous levels, and studies that headed the clear statement of the hypothesis have provided variable degrees of provision at those many levels. At the base, and apparently most well-founded, is evidence in support of the most important aspect of the hypothesis, namely that template strands can separate not haphazardly to daughters of a dividing cell, organized by template age. Although context dependent, the increasing data suggest mechanisms that exist in cells across a vast phylogenetic spectrum and challenge the dogma that the two strands of the double helix are identical with regard to their roles as templates during DNA replication. At a middle level, the evidence of the hypothesis that such a non-random segregation would happen only during unequal cell division associated with stem cell self-renewal may be true but incomplete. This may need to be generalized to other contexts of tissue expansion and regeneration when the progeny of stem cells undergo asymmetric cell divisions that lead to different fates of the daughters. Finally, at the level with the smallest experiential evidence is the clear evidence of the immortal strand hypothesis that non-random segregation of template strands is a process to limit the tendency of stem cells to obtain mutations leads to tumor formation.

CONCLUSION

One of the most reflective implications of this hypothesis is the proposal that the complementary DNA strands are in fact not

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equal but are unique with regard to their roles as templates for DNA replication. A consequence of the hypothesis is that stem cells capable of retaining immortal strands would not be subject to telomere shortening associated with DNA replication since progressive telomere shortening arises as newly synthesized strands become templates in successive generations. That is not to say that stem cells would not experience other aspects of chronological aging that can lead to telomere shortening. The validity of the immortal strand hypothesis also depends on the assumption that sister chromatid exchange and mitotic recombination are exceedingly low in stem cell populations.