

## The Functioning of “Aged” Heterochromatin

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Total heterochromatin constitutes regions of chromatin that mainly consist of two different classes of heterochromatin, constitutive and facultative, which are defined as transcriptionally inactive and compact chromatin, respectively. In the present investigation, we assessed the modification of total, constitutive (pericentromeric, telomeric and nucleolus organizer region (NOR) heterochromatin) and facultative heterochromatin in cultured lymphocytes exposed to the influence of heavy metals and bioregulators from individuals aged 80 years and over.

The results showed that: (1) progressive heterochromatinization of total, constitutive (pericentromeric, telomeric and NOR heterochromatin) and facultative heterochromatin occurred with aging; (2) a decrease in repair processes and an increase in frequency of chromosome aberrations with aging is secondary to the progressive heterochromatinization of chromosomes; (3) peptide bioregulators induce deheterochromatinization of chromosomes in old age and (4)  $\text{Co}^{2+}$  ions alone and in combination with the tetrapeptide bioregulator livagen (Lys-Glu-Asp-Ala), have different chromosomal target regions; that is, deheterochromatinization of pericentromeric ( $\text{Co}^{2+}$  ions) and telomeric ( $\text{Co}^{2+}$  ions in combination with livagen) heterochromatin regions in lymphocytes of older aged individuals.

The proposed genetic mechanism responsible for constitutive (pericentromeric, telomeric and nucleolus organizer region (NOR) heterochromatin) and facultative heterochromatin remodeling (hetero- and deheterochromatinization) of senile pathogenesis highlights the importance of external and internal factors in the development of diseases and may lead to the development of therapeutic treat.