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Effects of hypoxia for chromatin accessibility during oncogene induced senescence

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Senescence is characterized by a myriad of epigenetic changes, among which chromatin accessibility plays a pivotal role in regulating gene transcription through the binding of proteins to DNA. Additionally, reduced oxygen availability (hypoxia) is recognized as a regulator of histone methylation and as having an effect on chromatin structure. In this study, we employed ATAC-seq (Assay for Transposase-Accessible Chromatin with high-throughput sequencing) to investigate the intricate interplay between senescence, chromatin accessibility, and the influence of hypoxia. We focused on oncogene-induced senescence (OIS) and its impact on chromatin accessibility under both normoxic and hypoxic conditions. Our findings reveal that OIS increases chromatin accessibility, resulting in a ~15% increase in nucleosome-free regions (NFRs) under normoxia. This occurred more prominently in originally open chromatin regions (OCRs). However, hypoxia partially mitigates the heightened chromatin accessibility driven by OIS. These insights have the potential to advance our understanding of senescence-associated chromatin remodeling in cellular aging or disease processes.