The plasticity of DNA damage response during cell differentiation: pathways and consequences

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DNA damage constantly arises throughout life either by endogenous sources (e.g. cell metabolism) or by external agents (e.g. radiation). Depending on the time maintenance and function of a specific cell type the risk of accumulating DNA damage may vary. For instance damage to stem cells if not repaired can lead to mutation amplification or propagation through the processes of self-renewal and differentiation, respectively whereas damage to post-mitotic cells can affect mostly tissue homeostasis. To counteract these effects cells are provided of a variety of DNA repair mechanisms and signalling pathways, the so called DNA damage response (DDR), that ensure that DNA damage is repaired. Emerging evidence suggests that stem cells address DNA damage differently from their somatic counterpart. The information available on the common and distinct DDR mechanisms utilized along the self-renewal/differentiation processes will be reviewed. Recent data on the role of DDR in the control of muscle integrity will be presented by using as a model an *in vitro* skeletal muscle cell differentiation system exposed to different types of DNA-damaging agents

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