Erratum to: Overcoming Drug Resistance Through Elevation of ROS in Cancer

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Chapter "Overcoming Drug Resistance Through Elevation of ROS in Cancer" in: B. Bonavida (ed.), *Molecular Mechanisms of Tumor Cell Resistance to Chemotherapy*, DOI 10.1007/978-1-4614-7070-0_7

In Chap. 7, Figs. 1, 2, 3 are replaced with high resolution artwork for readability.

The online version of the original chapter can be found under DOI 10.1007/978-1-4614-7070-0_7

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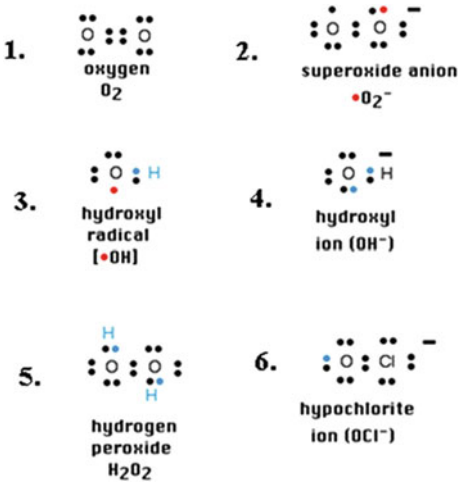

Fig. 1 Most common reactive oxygen species (ROS). *Red* is the unpaired electron which makes an extremely unstable configuration and reacts with other molecules or radicals to achieve stable configuration. The superoxide anion, which is both ion (*2*) and radical (*1*). Hydroxyl radical (*3*) is the most reactive of all radicals. It differs from the hydroxyl ion (*4*) and hydrogen peroxide (*5*). Ions like the hypochlorite ion (*6*) is also very reactive than other ions

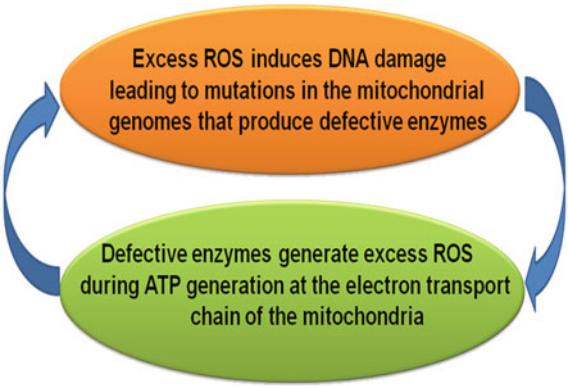

Fig. 2 *ROS generation in cancer cells*. Excess ROS in cancer cells induces mitochondrial DNA damage leading to secondary mutations that virtually produce nonfunctional enzymes and, in turn, generates more ROS through aberrant respiration. The excess ROS production at the mitochondria elevates the ROS level in cancer cells

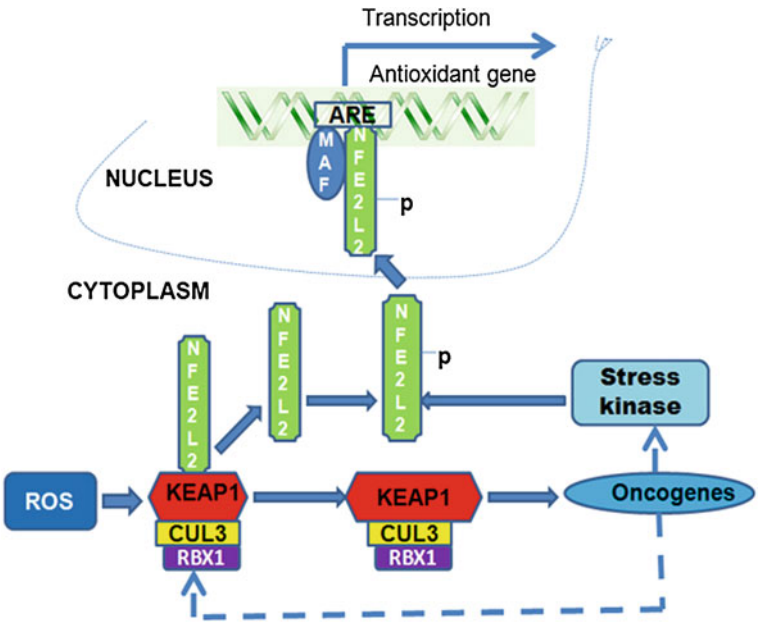

Fig. 3 NFE2L2—KEAP1 *mechanism for maintaining the ROS* ***level in cancer cells***. NFE2L2 remains bound with KEAP1, RBX1 and CUL3 at the cytoplasm. An excess ROS release KEAP1, RBX1 and CUL3 from NFE2L2 and free NFE2L2 is phosphorylated in the cytoplasm. Phosphorylated NFE2L2 travels to the nucleus and binds at the promoter of *ARE* sequence carrying antioxidant genes to facilitate transcription leading to increased antioxidant enzymes production that reduce excess ROS. P, the phosphate group, MAF-oncoprotein v-MAF family members