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Localized Movement and Levels of 53BP1 Protein Are Changed by γ-irradiation in PML Deficient Cells

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ABSTRACT

We studied epigenetics, distribution pattern, kinetics, and diffusion of proteins recruited to spontaneous and γ-radiation-induced DNA lesions. We showed that PML deficiency leads to an increased number of DNA lesions, which was accompanied by changes in histone signature. In PML wt cells, we observed two mobile fractions of 53BP1 protein with distinct diffusion in spontaneous lesions. These protein fractions were not detected in PML-deficient cells, characterized by slow-diffusion of 53BP1. Single particle tracking analysis revealed limited local motion of 53BP1 foci in PML double null cells and local motion 53BP1 foci was even more reduced after γ-irradiation. However, radiation did not change co-localization between 53BP1 nuclear bodies and interchromatin granule-associated zones (IGAZs), nuclear speckles, or chromocenters. This newly observed interaction pattern imply that 53BP1 protein could be a part of not only DNA repair, but also process mediated via components accumulated in IGAZs, nuclear speckles, or paraspeckles. Together, PML deficiency affected local motion of 53BP1 nuclear bodies and changed composition and a number of irradiation-induced foci. J. Cell. Biochem. 9999: 1–14, 2016.