As a first step towards understanding the mechanisms underlying parental histone mobilization from UVC-damaged regions, we measured in parallel histone and DNA density in damaged regions. Thus, we observed that parental histone H3.3 redistribution was accompanied by a loss of DNA density in UVC-damaged regions (Figure 3A), indicative of local chromatin opening. While both histone loss and DNA loss increased with the exposure time to UVC laser, the extent of histone loss exceeded the extent of DNA loss in all conditions examined (Figure 2B), which cannot be explained solely by chromatin opening. To account for this, we built a mathematical model that invokes both chromatin opening and nucleosome sliding (Figures 3C-D and S3). Our modeling approach shows that parental histone redistribution in response to UVC damage can indeed be driven by a combination of chromatin opening and nucleosome sliding, whose relative contributions depend on the amount of UVC damage (Figure 3E). Nucleosome sliding saturates when a maximum number of nucleosomes are damaged in the irradiated region. Interestingly, both the fraction of histones lost by chromatin opening and by nucleosome sliding (before sliding saturates) are directly proportional to the amount of UVC damage according to the model (Figure 3F). This strongly suggests that these chromatin dynamics are controlled by factors binding to UVC damage.