A micro-irradiation technique was used to study the temporal and spatial distribution of radiation-induced DNA double strand breaks and the recruitment of repair proteins in living human cells. Synchrotron-produced ultrasoft x-rays, used in conjunction with micro-fabricated irradiation masks, were used to irradiate partial volumes of individual cell nuclei in a “striped” pattern. Subsequent DNA double strand breaks (DSB) and associated repair proteins were visualized using immunofluorescence. Microscopic images verify that the DNA damage induced by ultrasoft x-rays is highly localized to the regions of photo-electric interaction. Even more important, it is shown that double strand breaks remain spatially fixed in the nuclear matrix during the initial stages of DNA repair, and that DNA double strand repair proteins (hMre11) migrate to those sites of DNA damage, evidenced by stark striping patterns in the fluorescent images. In normal human fibroblasts (37Lu), the double strand break signal decreased in intensity due to repair, reaching background levels after 60-90 minutes; the striped pattern remained intact during this time, fading but not diffusing. In a double strand break repair deficient cell line (180BR), the stripes of damage still persisted 300 minutes post-irradiation. These data suggest that DNA repair does not involve movement of lesions through the intranuclear volume. The hMre11-rad50 repair protein complex, however, does appear to move through the nucleus, aggregating in the regions of radiation-induced damage.