DNA damage may result in various pathological conditions and contributes to aging and development of cancer. Evolutionarily conserved DNA damage response prevents the acumulation of mutations and protects against genomic instability. Tumor suppressor p53-binding protein 1 (53BP1) is an important regulator of the cellular response to DNA double-strand breaks (DSB) and is a canonical component of ionizing radiation-induced foci which are formed at DNA DSB following radiation exposure. Recently, new insights have been gained into its functions in the DNA damage response. Apart from its subtle role in the DNA damage checkpoints signaling, 53BP1 is a well established player in the DNA DSB repair pathway choice. The outcome of DNA repair is influenced by 53BP1 in several contexts. 53BP1 controls 5' end resection at DNA ends, improves DSB repair in heterochromatin, promotes the mobility of uncapped telomeres and mediates synapsis of DNA ends during V(D)J and class switch recombination. 53BP1 contributes to repair defect in BRCA1 (breast cancer type 1 susceptibility protein)-deficient cells, which may have an impact on the treatment of some types of breast cancer. The aim of this bachelor's thesis is to summarize new findings about the role of 53BP1 in the cellular response to DNA DSB.