Host organisms evolved antiviral responses, which can recognize the viral infection and deal with it. One of the frequent signs of viral infection in a cell is appearance of double-stranded RNA (dsRNA). One of the pathways responding to dsRNA is RNA interference (RNAi), which functions as the key antiviral defence system in invertebrates and plants. Mammals, however, utilize for antiviral defence a different dsRNA-sensing pathway called the interferon response. RNAi functions only in mammalian oocytes and early embryonal stages although its enzymatic machinery is present in all somatic cells, where it is employed in the microRNA pathway. A previous study indicated that the functionality of RNAi in mouse oocytes functions due to an oocyte-specific isoform of protein Dicer ($Dicer^{O}$), which is truncated at the N-terminus. In my thesis, I aimed to assess whether $Dicer^{O}$ processes RNAi substrates more efficiently *in vitro* than the full-length Dicer ($Dicer^{S}$), which is found in somatic cells. Therefore, I developed Dicer purification protocol for obtaining both recombinant mouse Dicer isoforms of high purity. I examined their activity in a non-radioactive cleavage assay using RNA substrates with structural features characteristic of RNAi substrates. My results suggest that recombinant $Dicer^{O}$ and $Dicer^{S}$ do not differ in processing of long perfect duplexes or in their ability of performing an internal cleavage. Nevertheless, further experiments are necessary to explain these results as the non-radioactive cleavage assays suffered from substantial technical limitations and the reduced activity of recombinant Dicers, which might be a consequence of the affinity tags at the N-terminus.