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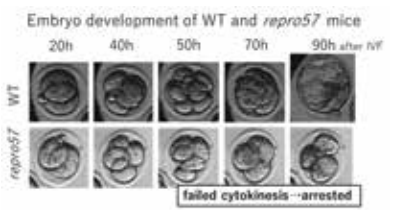
The meiotic abnormalities in mouse and human oocytes and their mechanisms

During the maturation process of human oocytes, numerous genes are responsible for meiosis. Recent studies have begun to reveal that defects in some interrelated genes are the cause of female infertility. We aim to perform functional analyses of the process of oogenesis, during the first and second meiosis using oocytes from mutant mice, which present with a mutation, leading to genetically developed abnormalities on chromosomal crossing-over during meiosis. In this way, we hope to contribute to the existing body of therapeutic strategies and preventive measures which can be taken against human infertility. As ten to twenty percent of cases of infertility are considered to be idiopathic, we also aim to perform a comprehensive analysis of meiotic DNA mutations and thereby elucidate the causes of infertility derived from those mutations.



The elucidation of the mechanism of dysmorphic phenotypes in human oocytes and preventive measures against those dysmorphisms

The dysmorphic phenotypes to be found in human oocytes are unlike any other species, with the exception of chimpanzee oocytes. Among these dysmorphisms, we have been focusing on two phenotypes: smooth endoplasmic reticulum clusters (sERCs) and refractile/lipofuscin bodies. We have found that the incidence of mitotic and meiotic cleavage failure during the second polar body extrusion is significantly higher in oocytes with sERCs than that in oocytes without sERCs. Regarding the refractile body (RB), we have reported that it presents with autofluorescence. Viewed by means of transmitted electron microscopy, the refractile bodies display the conventional morphology of lipofuscin inclusions and consist of a mixture of lipids and dense granule materials. We aim to elucidate the mechanisms of these dysmorphic phenotypes in human oocytes and work towards preventive measures against them.