The aim of my presentation is to show the current knowledge on dycarbonyl stress in ovarian physiology and the rationale for the possible use of glyoxalase pathway as a therapeutic target in ovarian dysfunctions and aging. Since the evidence of increased AGEs in serum and ovary of women suffering from polycystic ovarian syndrome (PCOS), the interest in dycarbonyl stress and female fertility has been continuously increasing. Afterwards, a possible correlation between dycarbonyl overload and ovarian aging has emerged from studies showing increased intraovarian AGEs in reproductively aged mice. By using a specific antibody against a specific adduct formed by methylglyoxal (MG), increased glycation of ovarian polypeptides were shown in aged ovaries along with decreased glyoxalase 1 (Glo1) expression and activity. Moreover, assays on mouse oocytes have revealed that MG level may contribute to predisposition to aneuploidy and reduced oocyte competence with aging. Notably, the importance of antiglycation defence in folliculogenesis has been shown by the finding that Glo1 and Glo2 transcripts change during meiosis in mouse oocytes, consistently to their role in oocyte development. According to clinical investigation, specific serum levels of AGEs correlate positively with diminished fertility, age and metabolic disorders. Although relevant data were obtained, further investigations on dycarbonyl stress and relative response pathways in the ovarian follicle are needed prior to establish whether AGEs might be predictive markers and/or therapeutic targets in female fertility disorders.