Ozone as a major constituent of photochemical smog has been suspected as a risk factor for the development of lung cancer. The inhalation of ozone is strongly excluded in the strategies for therapeutic use of medical ozone, but ozone inhalation can not always be prevented in outdoor (atmosphere) and indoor (ozone user and patients) situations. Whether ozone has carcinogen potential has been repeatedly examined in isolated cell systems or in experimental animals. Mice seem to be the most sensitive indicators for lung tumor carcinogenesis in comparison to rats. With a new design of inhaled ozone (ultra short x high ppm) we compared our results, from NMRI-mice, with that calculated dosages of other authors and discussed the steps of carcinogenesis (inhibition-promotion-progression). Ozone is not a primary lung carcinogen compared to urethan (neumotropic carcinogen). Ozone enhances the promotion of otherwise initiated lung tumors (pro-carcinogenesis) and also inhibits the promotion (anticarcinogenesis) probably by direct cytotoxic effects. Inhaled ozone has a mitogenic activity on lung cells (pneumocytes Typ II cell hyperplasia) and by thus it enhances the risk for initiation of lung tumors (adenonas) caused by carcinogenic neumotropic chemicals. There was no evidence for ozone carcinogenesis in NMRI-mice without a carcinogenic drug.