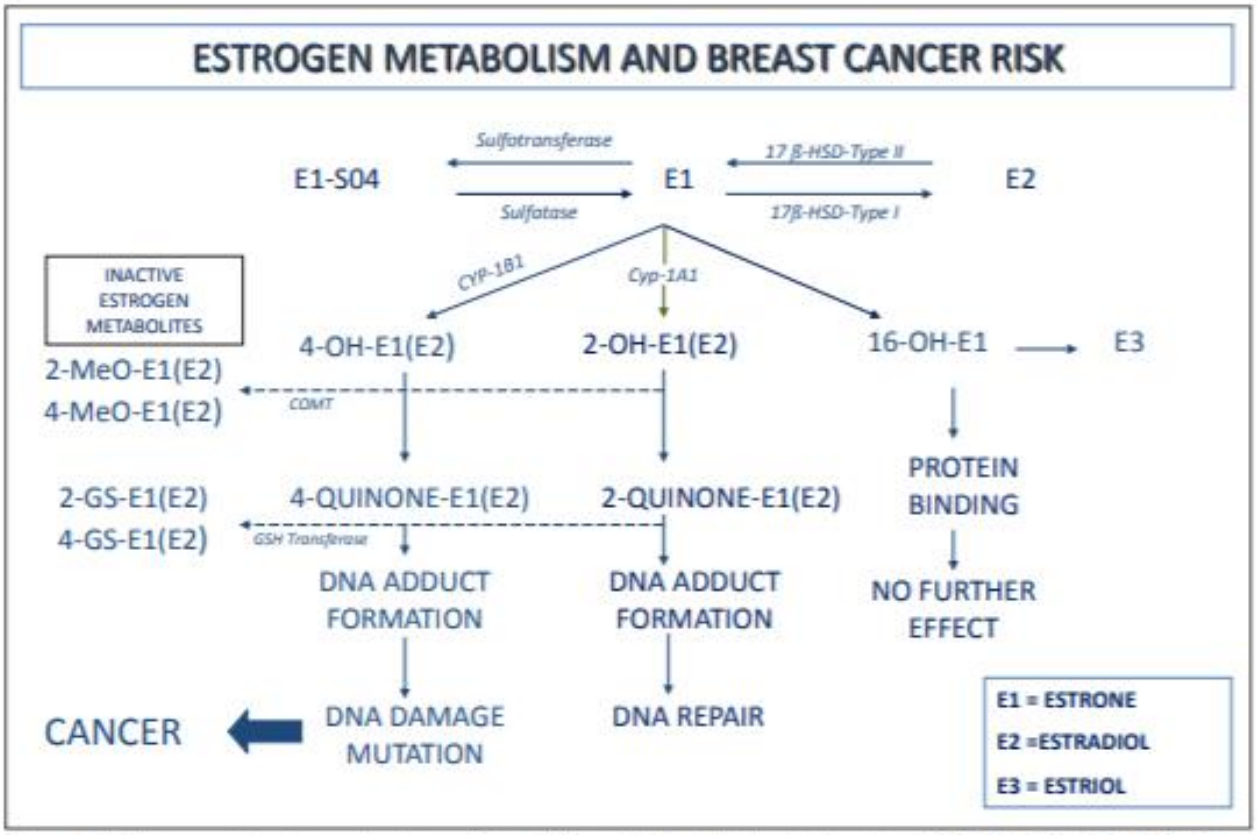

Shown in this figure are the stages of estrogen (E1 and E2) metabolism that lead to harmless (2-OH-E1, 2-OH-E2, 16-OH-E1, and E3) or potentially toxic (4-OH-E1 and 4-OH-E2) estrogen metabolites. Both 2- and 4-OH estrogens (often referred to as catechol estrogens) are inactivated by methylation with COMT (Catechol-Ortho-Methyl-Transferase) to form 2- and 4-methoxy estrogens (2-MeO-E1 and –E2 or 4-MeO-E1 and –E2). If methylation does not occur due to lack of COMT substrates (vitamins B6, B12, folate, betaine), or defective gene polymorphisms in COMT, the catechol estrogens can further oxidize to 2- and 4-estrogen quinones. Formation of these oxidized quinone estrogens occurs more readily in the presence of excessive oxidized lipids, such as trans-hydrogenated fats, and heavy metals. Under ideal situations the quinone estrogens react with glutathione, which inactivates them. In the absence of adequate cellular glutathione and glutathione transferase, these highly reactive and electrophilic estrogen quinones bind covalently to DNA forming adducts that can lead to mutations that increase risk for cancers in estrogen target tissues such as the breasts, uterus, ovaries, and prostate.

Courtesy ZRT labs