We read with great interest the work of McDougall and colleagues recently appeared on your journal reporting that current users of statins for 10 years or longer had a 1.83-fold increased risk of invasive ductal carcinoma (IDC) and a 1.97-fold increased risk of invasive lobular carcinoma (ILC) compared with never users. Among women diagnosed with hypercholesterolemia, current users of statins for 10 years or longer had more than double the risk of both IDC and ILC compared with never users (1).

More recently, Desai and colleagues reported in your journal contrasting data obtained by Cox proportional hazards analyses conducted on large-scale population from the WHI study (154,587 postmenopausal women with 7,430 breast cancer pathologically confirmed), concluding that statins were not associated with breast cancer risk (2).

A recent review analyses as well as the conflicting results about the chemopreventive statin use in breast cancer population concluded that even if their effects are modest, the overall good long-term tolerability and relative low cost could make them new attractive chemopreventive agents (3).

Considering both the increased statin use over the past few decades and the high breast cancer incidence, morbidity, and mortality, it is mandatory to properly define the exact role of statins in breast cancer risk and recurrence without underestimating the potential implications in ovarian and endometrial tissues. Because of the common hormone-dependent origin and similarities in etiologic factors, gene expression profiles, tumorigenic mechanisms, pathologic changes, and metastatic characteristics, it would be also important to evaluate the effects on ovarian and endometrial tissue. To our knowledge, only one study was properly designed to analyze “Statin use and female reproductive organ cancer risk in a large population-based setting.” Authors suggested that there is a nonsignificant reduced risk of endometrial and ovarian cancers among statin users compared with non-users (4).

As well as was discovered raloxifene antiproliferative effect on endometrium during the monitoring of breast antiblastic effects (making it a possible candidate to substitute tamoxifene), something similar could happen on monitoring statins’ effects on hormone-dependent gynecologic malignancies (5).

To solve the dilemma, an answer to the lingering questions about the association between gynecologic cancer risk and statin use, a meta-analysis will be necessary combining the existing large-scale studies and further prospective ones. In the same way, further prospective studies, even if conducted on breast cancer and statins, should also consider outcomes about ovary and endometrium to increase the amount of data available in this field.

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Long-term Statin Use and Risk of Breast Cancer—Letter
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