this cut point might not be relevant for a chemotherapy cohort with different baseline characteristics and different therapy approaches. Similarly, a cut point defined in one chemotherapy trial might be different in a second chemotherapy trial with different treatment and response rate. From our point of view, this shows that a data-derived cut point optimization is not possible. However, this does not imply that Ki67—and other proliferation markers—are not useful; it just shows that the cut points are context dependent. Ki67 could be measured as a continuous marker with a robust and standardized methodological approach, such as image analysis. In addition, the scientific community could define cut points for different practical purposes.

C. Denkert1,2 & G. von Minckwitz2

1 Institute of Pathology, Charité-Universitätsmedizin Berlin, Berlin, Germany
2 German Breast Group, Neu-Iseburg, Germany

(*E-mail: carsten.denkert@charite.de)

disclosure

The authors have declared no conflicts of interest.

references


doi: 10.1093/annonc/mdt564
Published online 15 January 2014

Meta-analysis procedure for the effect of statin on the recurrence of prostate cancer

Overviewing the recent publication, Park et al. [1] conducted a meta-analysis to clarify the effect of statin on the recurrence of prostate cancer with special reference to two major treatments. In this letter, I have a concern on their basic procedure of selecting information stratified by the use of statin.

First, the authors selected appropriate references according to the standard flow diagram, but there was a difficulty of extracting information for conducting pooled estimates in studies of radiotherapy. Namely, the number with positive outcome in statin treatment group and control group can be identified three of seven studies. Almost the same meta-analysis was presented by Mass et al. [2], and Park et al. added one reference from proceeding.

Second, although hazard ratios (HRs) with 95% confidence intervals were listed in their Figure 2, Zaorsky et al. selected multiple logistic regression analysis [3]. As this reference occupied 10.3% of all the data, re-analysis by excluding this reference should be conducted for calculating pooled estimate of HR. In addition, Park et al. quoted one abstract in studies of radiotherapy, which is contrary to the full-text analysis.

Taking together, it seems invalid to accept their final conclusion that beneficial effect of statins on prostate cancer patients. As related research outcomes are continuously presenting in the database [4, 5], revision of meta-analysis is strongly recommended to confirm their conclusion.

T. Kawada*
Department of Hygiene and Public Health, Nippon Medical School, Bunkyo-Ku, Tokyo, Japan
(*E-mail: kawada@nms.ac.jp)

disclosure

The author has declared no conflicts of interest.

references


doi: 10.1093/annonc/mdt563
Published online 12 January 2014

Reply to ‘Meta-analysis procedure for the effect of statin on the recurrence of prostate cancer’ by T. Kawada

We greatly appreciate Dr Kawada’s interest in our meta-analysis. The respondent makes several points worthy of additional discussion.

The first comment explores the challenges of extracting information to determine pooled estimates of the outcome variable. As we noted in our discussion, we excluded 4 of 17 potentially eligible studies that failed to report a unified effect estimate for biochemical recurrence as a function of peritreatment statin use [1]. Of these four studies, three did not address biochemical recurrence [2–4]. The one study that did address this outcome (Lavery et al. [5]) reported a nonsignificant effect of statins on biochemical recurrence among men treated with radical prostatectomy, which is consistent with the findings of the other radical prostatectomy studies.