This report has been closed.

Follow-Up Statements
The organization sent these questions or comments before report was closed. You cannot respond.

7/17/2014 3:34 PM - Thank you for sharing your concerns through the Ohio State University Anonymous Reporting Line. The university takes all reports seriously and will investigate the information that you have provided.

If you have additional information regarding your concerns, please post it as a follow-up.

We will provide you with updates of the university's review as it moves forward. We will also provide a final response once the review is completed.

Sincerely yours,

Jennifer Yucel, Ph. D.
Director/Research Integrity Officer

9/30/2014 3:21 PM - Thank you again for forwarding your concerns. The Institution has reviewed the information you provided including your request regarding the release of the data and has determined that the data relating to the research cannot be publicly released as it constitutes intellectual property under the Ohio Public Records Act (R.C. 149.43). The data, if disclosed, would reveal pending research ideas and techniques. Consequently, the release of such information would put those using such data for research purposes in a substantial competitive disadvantage as competitors and researchers would have access to the unpublished intellectual property of the University and its faculty and students.

The Institution has requested that the authors respond to the questions/issues raised in the Stefanek et al., commentary published December 15, 2009. Their response is included below:
Stefanek/Coyne claim: Stefanek/Coyne write, "a priori identification of primary endpoints, fixed observation period or a prespecified (sic) number of events, and prespecification (sic) of plans for data analyses" are "standards of evidence." Continuing, they write, "The trial presented by Andersen et al. fails to meet these modest standards (pg. 5613, col. 1, para. 1)."
Authors Response: The standards Stefanek/Coyne note as failing to be met were provided in the Andersen et al. (2008) manuscript: "The trial was powered to detect a doubling of time to an endpoint, a standard metric in cancer treatment trials, which was estimated to require 27 events (in this case cancer recurrences) in each treatment arm (level of significance =. 05, power = .80). Disease recurrence was defined as the detection of metastatic disease either at the same site (local) or distant from the original site (pg. 3451)."
After identifying 54 events, we rechecked disease status and obtained death certificates for those deceased. During the time in which death certificates were sought there were additional recurrence events, bringing the total number of events to 62.
Stefanek/Coyne claim: Stefanek/Coyne write, "Briefly, it appears that survival was not a primary endpoint," "the observation period was not specified before hand," and continuing, they write,

"and time-to-event prediction that was the basis for their power analysis was too optimistic to be credible." (pg. 5613, col. 1, para. 1).

Authors Response: As was made clear in the Andersen et al. (2008) text (see above in reference to pg. 3451), the stated endpoint was recurrence. The specified number of events determined the observation interval. As detailed in Andersen et al. (2008), the power analysis specified 54 events, i.e., cancer recurrences.

Andersen et al. (2008) and the DoD, NIH, and ACS evaluations from peer review panels did not evaluate the power analysis as "too optimistic." Provided verbatim below is the biostatistical power analysis section from the eventually funded NIMH grant application (1996-2001). Co-investigator Donn Young, Ph.D., then Director of the Biostatistics Unit of the Ohio State University Comprehensive Cancer Center, authored this section. The text is as follows:

"Our computations of required sample sizes for time to recurrence are based on the following data sources: 1) Five year survival rates from NCI's PDQ system recurrence rates (relevant pages are provided in Appendix C); 2) recurrence rates; and, 3) local experience on the relative proportion of patients with Stage II and III breast cancer. First, the PDQ data state that 5-year survival for stage II and III breast cancer are 66% and 41%, respectively. During the five-year interval, the proportion of women developing recurrent disease is approximately equal to the proportion dying of disease plus 10%. At the Ohio State University, of women with stage II-III breast cancer, the ratio of stage II to stage III is approximately 3:1. Data from 1992 for Franklin County (which surrounds Columbus) for stage specific breast cancer diagnoses in the county also indicate a 3:1 ratio. Using these figures, the overall 5 year survival for women demonstrating recurrence during the 5 years is approximately $(1 - 0.60 + 0.10) = 50\%$ (pg. 98)."

The overall 5-year relative survival rate estimated in PDQ for female breast cancer patients improved substantially from the time of first patient accruals into the trial in 1994 to the present day. The current overall survival rate is 90% [American Cancer Society (2014) using data from 2003 through 2009]. The 5-year survival rate for those with regional (Stage II/III) is currently estimated to be 84% (American Cancer Society, 2014). Regardless, we conducted the power analysis based on the best available data sources of the early 1990's, and multiple funding agencies (DoD, NIH, ACS) evaluated and approved the validity of our study proposal and, most importantly, the power analysis for the trial. We find these agencies' peer review evaluations to be rigorous, complete, and highly credible.

Stefanek/Coyne claim: Stefanek/Coyne make a comparison of the Andersen et al. (2008) trial with a Romond et al. (2005) chemotherapy trial of trastuzumab plus adjuvant therapy. They ask the leading question, "Do Andersen et al. (2008) believe that a psychosocial intervention consisting of a mixture of relaxation training, problem solving, and health behavior promotion should be so much more potent that it could be tested adequately with a sample size and event accrual less than one-seventh of those in the trastuzumab trial (pg. 5614, col. 2, para. 2; pg. 5615, col. 1, para. 1)?"

Authors Response: Is the Romond paper the appropriate comparison? Romond et al. (2005) use a completely different research design, combine data from two different trials (B-31 with N=2043 and N9831 with N=1633) into one for analysis. Cooperative groups (NSABP, NCCTG) with multiple study authors (n=25) from multiple institutions conducted the trials. Romond et al. (2005) analyze an accrual N of 3,676 to achieve the 394 events which Stefanek/Coyne note. In the trial the treatments were surgery and radiotherapy, with specific study of chemotherapy, funding for the research was corporate (Genentech), and there were multiple interim endpoint analyses ["The first interim analysis was to take place after 355 events had been reported.

Subsequent interim analyses were scheduled to take place semiannually (pg. 1674)."].
Thus, Stefanek/Coyne hold the Romond paper to be an appropriate comparator, despite the differing study design, intervention (pharmacologic treatment), corporate funding, interim analyses, and many other differences. The only obvious similarity between the trials is that women with breast cancer were enrolled. In sum, Stefanek/Coyne's question was leading and their comparison was inappropriate.

Stefanek/Coyne claim: Stefanek/Coyne write, "In the current study, were recurrence and survival the primary endpoints (pg. 5614, col. 2, para. 2)?"
Authors Response: Andersen et al., (2008) clearly stated that the primary endpoint was recurrence (as was stated above). Stefanek/Coyne were also clear about the endpoint as on the previous page it is written, "Briefly, it appears that survival was not a primary endpoint (pg. 5613)."

Stefanek/Coyne claim: Stefanek/Coyne write, "it is noteworthy that no rationale is given for limiting the study follow up observation period to 7-13 years rather than shorter or longer periods (pg. 5613)."
Authors Response: The authors are making the same inaccurate "observation period" comment as noted above. Stefanek/Coyne should have realized that 7-13 years was, simply, the observed range in follow up time for the 227 patients when the analysis was done with the 62 events. This is not a "limiting" factor, but merely the observed interval. It appears that Stefanek/Coyne are not aware that time-to-event event analysis is not based on any specific time interval.
Andersen et al. (2008) make comment in the paper's discussion of the overall relevance of the observed median follow up interval for the sample being 11 years: "The majority of risk of recurrence (.37) occurs in the first 10 years of follow up (Shairer et al., 2004), a time point that was passed by greater than one half of the sample (pg. 3454)."

Stefanek/Coyne claim: "the inappropriate use of multivariate statistical analyses can "find" effects that do not really exist. This may be particularly true when assumptions are unmet… (Pg. 5613, col. 2, para 2)."
Authors Response: The Andersen et al. (2008) multivariate analyses were appropriate and the specific procedures used are common in the statistical literature and ones that are recommended by statistical and biostatistical experts.
Andersen et al. (2008) specified the basis for the variables included in the analyses. First, the minimization method of White and Freedman (1978) for randomization was noted and recommendations from the literature regarding controlling for minimization factors for survival analyses in clinical trials were cited (Scott et al., 2002). More recently, the same recommendation has been made by others, e.g., Kahan and Morris (2012): "It is widely acknowledged in the statistical literature that the subsequent analysis should reflect the design of the study, and any stratification or minimization variables should be adjusted for in the analysis. If this correlation is ignored and an unadjusted analysis is performed, standard errors for the treatment effect will be biased upwards, resulting in 95% confidence intervals that are too wide, type I error rates that are too low and a reduction in power. Conversely, an adjusted analysis will give valid inference (pg. 328)." Extending the point, Kahan, Jairath, Dore, and Morris (2014) write, "It is essential that stratification factors be accounted for in the trial analysis (pg. 141)." Second, we considered the factors with initial group differences (KPS and POMS). These differences had been reported in a previous paper (Andersen et al., 2004), of which Stefanek/Coyne were aware. The KPS and POMS differences occurred by chance, after

randomization. As has been noted (e.g., Hernandez, Steyerberg, & Habbema, 2004; Kahan, Jairath, Dore, & Morris, 2014), adjusted effect estimates can take into account chance differences in baseline characteristics between treatment arms and thereby improve power. Third, in designing the trial, the existing literature then defined the high-risk breast cancer patient (Clark & McGuire, 1992), age and receptor status were identified as well as the important role of treatments received in predicting eventual survival. This is the same strategy used by other investigators in cancer clinical trials (e.g., Flahavan, Bennett, Sharp, & Barron, 2014). Regarding the use of such predictive factors in analyses, Hernandez, Eijkemans and Steyerber (2006) write: "Adjustment for predictive baseline characteristic yields greater power to detect a true treatment effect rather than unadjusted analysis, without inflation of type I error and with potentially moderate reductions in sample size. Analysis of RCTs with time-to-event outcomes should adjust for predictive covariates (pg. 41)."

In analyzing the data and writing the manuscript, Andersen et al. (2008) were fully aware of opinions and data regarding the use of covariates. See, for example, a recent discussion (2011) among investigators about this issue and the response of Andrew Gelman, an expert on applied Bayesian data analysis and hierarchical models. Gelman's (2011) provided positive recommendations for covariate inclusion and are corroborated by studies examining covariate selection and entry, which appeared prior to and now following Gelman's statement in 2011. For example, Hernandez, Steyerberg, and Habbema (2004) noted that randomized controlled trials (RCTs) with dichotomous outcomes (such as disease endpoints) might be analyzed with or without adjustment for baseline characteristics (covariates). They studied type I error, power, and potential reduction in sample size with several covariate adjustment strategies with simulated data sets (20,000) using a two arm RCT with N=360. They concluded that in RCTs with dichotomous outcomes, covariate adjustment increases statistical power and reduces sample size requirements. Their conclusion with simulated data was confirmed in a follow up study using actual patient data. Hernandez et al. (2006) next conducted an analysis using data from seven RCTs (n=6166) and three surveys (n=2238) of traumatic brain injury patients in which a dichotomous therapeutic outcome was predicted. They found that covariate adjustment led to increased power when using the strongest predictors of outcome, and they recommended that covariate adjustment for strong predictors are included in future trials. This year, Kahan, Jairath, Doré and Morris (2014) made the same recommendations studying data from multiple trials of multiple disease sites (i.e., "Adjustment for known prognostic covariates can lead to substantial increases in power, and should be routinely incorporated into the analysis of randomized trials (pg. 139)." Multiple examples of covariate adjustment increasing power in randomized controlled trials can be found in the literature (e.g., Turner et al., 2012)
Stefanek/Coyne claim: "the inappropriate use of multivariate statistical analyses can "find" effects that do not really exist. This may be particularly true when. …data are over fit (pg. 5613, col. 2, para 2)."
Authors Response: Rather than "multivariate statistical analyses," the appropriate term for the procedures of Andersen et al. (2008) was use of predictive covariates in addition to the treatment variable in a Cox Model. Stefanek/Coyne demand that Kaplan-Meier estimate should have been used ("Andersen et al., do not report standard, unadjusted outcomes, such as a Kaplan-Meier estimate of the survival function…. (pg. 5613)." The first point to note is that covariate adjustment analysis is recommended by experts in clinical trial and decision sciences (e.g., Hernandez, Jkemans, & Steyerberg, 2006) because of the following: 1) it achieves more

appropriate treatment effects (e.g., when correcting for chance imbalance); 2) it determines more individualized treatment effects; and. 3) it has greater statistical power, i.e., the ability to detect a treatment effect when it really exists. Stefanek/Coyne were apparently unaware of these facts and studies. In sum, use of covariate adjustment analysis was appropriate and is a standard practice in clinical trial analyses (see also discussion of this issue above).

Stefanek/Coyne claim: Stefanek/Coyne claimed that a problem existed in the ratio of events per variable (EVP) in the Andersen et al. (2008) analyses. [In Andersen et al. (2008) the EPV was 5.6.] Stefanek/Coyne substantiate their opinion by citing a 1995 article (Peduzzi et al., 1995). Stefanek/Coyne state the following: "The general rule is that predictors should not be added to the equation if the ratio of outcome events to predictor variables is not at least 10:1 (pg. 5613, col. 2, para 2)."

Authors Response: Stefanek/Coyne fail to mention that the Peduzzi viewpoint was drawn from two simulation studies, both of which he, Peduzzi, conducted (i.e., Peduzzi, Connato, et al., 1996; Concato, Peduzzi et al., 1995). In them, only the numbers of EPV were varied; the sample size and the distribution and effects of seven binary predictors were held constant. Using that method, he reported increasing bias and variability, unreliable confidence interval coverage, and problems with model convergence as EPV declined below 10 and especially below five. Peduzzi et al. (1995) concluded that results should be cautiously interpreted with less than 10 EPV. Andersen et al (2008) were fully aware of these studies.

However Andersen et al., (2008) were aware of additional factors, with one being that a rule of thumb is just that, "a rule of thumb," and not a prerequisite nor a fact as Stefanek/Coyne assert. Secondly, our consideration of these issues extended beyond a selective 1990s literature search. For example, a paper by Vittinghoff and McCulloch (2006) is relevant. Vittinghoff and McCulloch also conducted a simulation study, but theirs went beyond the EPV focus of Peduzzi, Concato, Feinstein, and Holford (1995). Namely, Vittinghoff and McCulloch additionally studied several model performance measures such as confidence interval coverage, type I error, and relative bias. Doing so, they found problems in only 8.6% of the simulation scenarios with 5-9 EPV. In summarizing they write, "Our results indicate that problems are fairly frequent with 2-4 EPV, uncommon with 5-9 EPV, and still observed with 10-16 EPV." Continuing, "The worst instances of each problem were not severe with 5-9 EPV and usually comparable to those with 10-16 EPV (pg. 717)." They conclude, "Our simulation study shows that the rule of thumb of 10 or more EPV in logistic and Cox models is not a well defined bright line (pgs. 717)."

Stefanek/Coyne claim:(Pg. 5614, col. 1, para. 2). Stefanek/Coyne insinuate that a backward elimination method for variable selection was not appropriate.

Authors Response: In the analyses of Andersen et al. (2008) a limited number of important possible covariates that were specified, a priori, based on existing theoretical and empirical data (see above), such as those related to breast cancer outcomes in clinical trials. Only then was backward elimination used to reach a parsimonious model. When the backward elimination method is conducted sensibly in a manner such as this, it is used and accepted in psychology (e.g., Cole, Kemeny, Taylor, & Visscher, 1996) but even more commonly in clinical trials such as those with breast cancer patients (e.g., Knoop et al., 2005) as well as studies with other cancer groups [e.g., prostate patients in Flahaven, Bennett, Sharp & Barron (2014)]. In short, our

strategy of covariate identification was scientifically valid despite Stefanek/Coyne assertions that it was not. It was and continues to be a standard practice in clinical trial data analysis.

Stefanek/Coyne claim: Stefanek/Coyne state that findings of the study, such as the difference between the groups in median time to recurrence, were "small (6 months)" and "the differences in overall survival are small (pg. 5613, col. 1, para. 4)."

Authors Response: The median survival time difference between study arms was 1 year and 3.6 months longer for the intervention arm than the assessment only arm. The labeling of these differences as "small" are inappropriate value judgments.

Stefanek/Coyne claim: Stefanek/Coyne claim that the Andersen et al. (2004) paper on biobehavioral intervention outcomes analyzed 7 scales of emotional distress, 15 measures of immunity, 4 measures of social functioning, 8 measures of health behavior, and 4 measures of chemo adherence without any adjustment of Type I error (pg. 5614, cols. 1-2, para. 3-4).

Authors Response: Regarding the 2004 paper published in the Journal of Clinical Oncology (which conducts its own statistical review of manuscripts), Stefanek/Coyne's assertions about the number of analyses were incorrect. Text from the paper is as follows,

"The likelihood of type I errors was reduced by using a contingent, two step analytic procedure. For measures containing subscales (e.g. POMS), the total score was analyzed first, and only if significant did the analysis for the subscales follow. Four measures were used to assess the construct of social adjustment. For this, a MANOVA model was used to examine the effect of the intervention on the four measures simultaneously; if significant, ANOVAs followed for each measure (pg. 3573)."

Table 1 in Andersen et al. (2004) specified the 6 measured intervention outcomes. Regarding immunity, we have repeatedly noted in our papers, that immunity was included as a mechanism, not as an outcome measure of the intervention. T and NK cell counts (4) were reported for descriptive purposes for the reader and were not discussed further. Andersen and colleagues were not interested in cell trafficking (i.e., changes in cell counts) but change in function (i.e., T cell blastogenesis, NK cell lysis) of immunity. The number for the analyses were as follows: 1 scale of emotional distress, 3 measures of immunity, 4 measures of social functioning (combined for 1 analysis), 3 measures of health behaviors, and 1 measure of chemotherapy adherence.

Stefanek/Coyne claim: "In an earlier report of this trial, Andersen et al., demonstrated the exploratory nature with which this trial was designed…. (pg. 5614, cols. 1-2, para. 3-4)."

Authors Response: Stefanek/Coyne used their editorial opportunity to inaccurately represent other work by Andersen and colleagues. We assume that the point of these comments was to imply the use of "exploratory analytic strategies," the practice of over reporting of results, and the application of inappropriate statistics. Stefanek/Coyne's statements are factual errors, and the following are truths about the trial.

The guiding theoretical model for the trial, from which predictions were derived, was published in a 15-page scholarly paper in the American Psychologist (Andersen et al., 2004), "A Biobehavioral Model of Cancer Stress and Disease Course." This manuscript was, in fact, an extended version of the Background and Significance section of the funded NIMH grant. To date, this is the only psychological intervention trial in cancer that has ever provided a theoretical structure for a trial's constructs, intervention components, and measured outcomes (see also Table 1, in Andersen et al., 2004).

Prior to the intervention and disease endpoint papers, other papers were published on relationships/predictions in the biobehavioral model, e.g., a paper on the covariation of cancer stress and immunity (Andersen et al., 1998 in the Journal of the National Cancer Institute); a

paper on the covariation of cancer stress and subsequent declines in quality of life (Golden-Kreutz et al., 2005 in Health Psychology; papers on the psychological and behavioral outcomes of the trial (Andersen et al., 2004 in Journal of Clinical Oncology; Andersen et al., 2007 in Brain, Behavior and Immunity) and testing the mechanism of immunity in predicting health (Andersen et al. 2007 in Brain, Behavior and Immunity), among others. The Biobehavioral Model drove this work and the predictions derived from it. All papers of the trial reference the Biobehavioral Model as the basis of hypotheses tested.

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Thank you again for contacting the Ohio State University and bringing your concerns to the University's attention.
If you have additional information about this issue, now or in the future, please provide them through this Anonymous Reporting Line. The University will otherwise consider this matter closed.

Report Submission Date
7/17/2014

Reported Company/Branch Information

Name	OSU - Research

Violation Information
Issue Type
    Scientific Misconduct
Relationship to Institution
    Other / Remain Anonymous
Please identify the person(s) engaged in this behavior:
    Barbara Andersen - Professor
Do you suspect or know that a supervisor or management is involved?
    Do Not Know / Do Not Wish To Disclose
Is management or the department aware of this problem?
    Do Not Know / Do Not Wish To Disclose
What is the general nature of this matter?
    Inaccurately reported results of research in published article and subsequent grant application.

    Refused to respond to published accusations
Where did this incident or violation occur?
    The article is

    Andersen, Barbara L., Hae-Chung Yang, William B. Farrar, Deanna M. Golden-Kreutz, Charles F. Emery, Lisa M. Thornton, Donn C. Young, and William E. Carson. "Psychologic

intervention improves survival for breast cancer patients." Cancer 113, no. 12 (2008): 3450-3458.

How long do you think this problem has been going on?

Don't know

How did you become aware of this violation?

Other

If other, how?

Read published article and saw response from editor of Cancer on web

Details

Data should be made publicly available for scientists to reanalyze.

Follow-Up Notes
There are no additional notes for this report.

Follow-Up Questions/Comments
There are no questions asked or comments from the organization.

Chat Transcripts
There are no chat transcripts for this incident.

Him