ent effects at the cellular level. L-NAME is relatively nNOS and eNOS selective. However, it is not clear that L-NAME is more potent in vivo, especially since it is believed that iNOS is the important target in patients with cardiogenic shock.[4] Refractory shock is characterized by lower-than-expected systemic vascular resistance and hypotension, both potential effects of excess NO.[5] Effects of NOS inhibition in patients with cardiogenic shock should differ from normal volunteers. Importantly, the initial positive single-center experience with NOS inhibition in patients with cardiogenic shock used L-NMMA.[1] We believe our conclusion is valid: L-NMMA, at the dose and duration studied in TRIUMPH, had no effect on mortality. It is possible that other dosing strategies, perhaps taking into consideration baseline renal function, or other NOS inhibitors might have a different effect. Determining this would require another large randomized clinical trial with mortality outcomes.

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CORRECTIONS

Incorrect Data and Omission of Trial Site and Personnel: In the Original Contribution entitled "Effects of Tamoxifen vs Raloxifene on the Risk of Developing Invasive Breast Cancer and Other Disease Outcomes: The NSABP Study of Tamoxifen and Raloxifene (STAR) P-2 Trial" published in the June 21, 2006, issue of *JAMA* (2006;295(23):2727-2741), incorrect data were reported. In Table 2 on page 2731, the risk ratio (RR) for estrogen receptor–positive patients should have been reported as 0.94. In the "Invasive Breast Cancer" panel of Figure 2 on page 2732, the number at risk in the raloxifene group at 36 months should have been reported as 6702. In the "invasive cancer" row of Table 3 on page 2732, the rate per 1000 for tamoxifen should have been reported as 1.99, the difference in rate per 1000 as 0.74, and the RR as 0.63. Also in Table 3, in the "hysterectomy during follow-up" row, the number of events for tamoxifen should have been reported as 221 and for raloxifene as 87, the rate per 1000 for tamoxifen as 12.24 and for raloxifene as 4.72, the difference per 1000 as 7.52, and the RR (95% confidence interval [CI]) as 0.39 [0.30-0.50]). On page 2733, top of column 1, the annual incidence rate for tamoxifen should have been reported as 1.99, the RR for raloxifene as 0.63, and the cumulative incidence rate through 7 years for tamoxifen as 14.6. Also on page 2733, end of first paragraph in column 2, the number of hysterectomies performed in those assigned to tamoxifen should have been reported as 221 and in those assigned to raloxifene as 87, and the RR (95% CI) as 0.39 (0.30-0.50). In the "Invasive Uterine Cancer" panel of Figure 3 on page 2733, numbers at risk in the raloxifene group at 18, 36, 54, and 72 months should have been reported as 4311, 3233, 2103, and 409, respectively; in the tamoxifen group, the numbers at risk at these same points should have been reported as 4301, 3120, 1984, and 371, respectively. In Table 5 on page 2735, the rate per 1000 for ischemic heart disease in the tamoxifen group should have been reported as 2.99 and the difference per 1000 as –0.30. In the first paragraph of the Comment section on page 2736, the terms "raloxifene" and "tamoxifen" were reversed in the second sentence; the sentence should have read "The cumulative incidence rates were 25.1 per 1000 women (tamoxifen) vs 24.8 per 1000 (raloxifene) ($P = .83$)." Also, a trial site and its personnel were inadvertently omitted: in the list of active NSABP STAR P-2 clinical centers appearing on page 2739, "*Boston Medical Center, Boston, Mass:* Marianne N. Prout (PI), Liz Pottier (PC);" should have appeared between the entries for Boca Raton Community Hospital and CAMC Health Education and Research Center.

Incorrect Data and Wording: The Original Contribution entitled "Patient-Reported Symptoms and Quality of Life During Treatment With Tamoxifen or Raloxifene for Breast Cancer Prevention: The NSABP Study of Tamoxifen and Raloxifene (STAR) P-2 Trial" published in the June 21, 2006, issue of *JAMA* (2006; 295[23]:2742-2751) reported incorrect data and included incorrect wording. One page 2746, in Figure 1, the *P* value for the SF-36 Mental Component Summary should have been .14 and for the SF-36 Physical Component Summary, the *P* value should have been .23. On the same page the *P* values should have been reported similarly in text: "($P = .14$, MCS and $P = .23$, PCS)." Also, on the same page, the text that read "Forms were not expected after death or consent withdrawal, which occurred at some point during follow-up for 197 women (1.0%) in the tamoxifen and 1352 (6.8%) in the raloxifene groups" should have read "Forms were not expected from the 207 women (1.0%) who died or from the 1352 women (6.8%) who withdrew consent at some point during follow-up."

Incorrect Data in Tables: In the Original Contribution entitled "Nonvalidation of Reported Genetic Risk Factors for Acute Coronary Syndrome in a Large-Scale Replication Study" published in the April 11, 2007, issue of *JAMA* (2007;297[14]: 1551-1561), incorrect data were reported in 3 tables. In Tables 1 and 2, the gene symbol *LPA*, should have been identified as *LPL* and in Table 3, the mean age for men with acute coronary syndrome (ACS) should be 60.7 years and for the controls, 60.0 years.