referred to in the personal communication to Swedberg, but in our trial withdrawals for this reason were according to prespecified criteria and in line with usual clinical practice.

The size of our trial was indeed based on a comparison of active with placebo treatment, but, as we pointed out, an assessment of the two active agents was an important secondary objective from the outset. The advantage of the diuretic over the β blocker was clear, particularly for coronary events, and is also suggested by other recent results.' Swedberg is surely not suggesting that findings of such immediate clinical relevance should remain unreported.

STANLEY PEART

Hunterian Institute, Royal College of Surgeons, London WC2A 3PN

T W MEADE

S J РОСОСК

MRC Epidemiology and Medical Care Unit, Wolfson Institute of Preventive Medicine,

London EC1M 6BQ

Department of Epidemiology and Medical Sciences, Medical Statistics Unit, London School of Hygiene and Tropical Medicine, London WCIE 7HT

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Life events and breast cancer prognosis

EDITOR,—As Jennifer Barraclough and colleagues' point out, the findings of their prospective study of life events and breast cancer prognosis differ from those of a cross-control study undertaken at Guy's Hospital.² Possible reasons for these apparently discrepant results should be sought before any influence of severe life experiences on relapse of breast cancer is dismissed.

At Guy's Hospital cases and controls were carefully matched for the major factors that influence outcome, including histological grade and number of involved nodes. At Southampton this information was not available. Thus important imbalances could have been present between patients with and without severe life experiences.

Clearly, knowledge of relapse status introduces the possibility of bias, but this is possible in any life events study as events can be ascertained only retrospectively. In the Southampton study the patients were not blind to their disease status at the time of an interview, and it must have been difficult for the interviewer to remain so. Bias in the rating of severity of events can be minimised if this is done by a panel of judges who are unaware of disease status, as was the case at Guy's Hospital.

At Guy's Hospital all interviews were conducted with the patients themselves, whereas for 11 of the 47 patients who relapsed at Southampton a final interview was conducted only with next of kin and for two other patients who relapsed no interview was performed. This could have led to underreporting of severe life experiences in a most important group.

The apparently high incidence of severe "own health" experiences not related to breast cancer observed at Southampton, affecting 21 patients during the 42 month follow up, was surprising. No such severe health experiences were recorded at Guy's Hospital. We were therefore interested in the finding that severe own health experiences had a significant adverse effect on outcome (p=0.01). The authors comment that it makes intuitive sense that patients with breast cancer in poor general health are more vulnerable to relapse. This is unproved.

The power of the Southampton study was diminished by the fact that 42 patients declined to participate and that these patients had a higher death rate (and presumably higher relapse rate) than those interviewed. We estimate that with 204 patients entered and a 23% relapse rate the study had about a 50% chance of detecting a difference between the groups if the true relative risk attributable to serious life experiences is 2-0.

Differences in the use of tamoxifen between the two studies may be important. At Southampton most postmenopausal women with positive nodes received tamoxifen. None of the patients at Guy's Hospital received tamoxifen (all patients presented initially before 1987). An intriguing possibility arises that the beneficial effect of tamoxifen could be partly mediated by counteracting any adverse effect of severe events. This benefit might be most pronounced in hormone sensitive tumours. In the Guy's Hospital study the influence of severe events on relapse was most apparent among the patients with tumours positive for oestrogen receptors.³



Recurrence free survival in women with breast cancer who had a severe event in first two years of follow up compared with other women in study (patients followed up to January 1991)

In our view the question of whether serious life experiences influence relapse is still unresolved. We are conducting a prospective study similar to that reported by Barraclough and colleagues, in which we are also assessing the impact of severe events on immunological and endocrinological variables. We would also encourage Barraclough and colleagues to re-examine the outcome for their patients after a longer follow up. This would not entail collection of further data on life events as we found that severe events occurring within the first two years after diagnosis were largely responsible for the positive outcome of our study (figure).

A J RAMIREZ	J P WATSON
M A RICHARDS	I S FENTIMAN
W M GREGORY	R D RUBENS
T I K CRAIG	

Imperial Cancer Research Fund Clinical Oncology Unit and Division of Psychiatry, Guy's Hospital,

London SE1 9RT

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EDITOR,—Jennifer Barraclough and colleagues report the lack of an association between psychosocial stress and relapse in breast cancer.¹ The results of their prospective study contrast with those of two studies showing a positive association between adverse life events and the onset and relapse of breast cancer.²³ The results of all three studies are based on data obtained with the same interview (the life events and difficulties schedule), suggesting that Barraclough and colleagues' opposite conclusions arise from differences in the design and analysis of their study.

Given the slow evolution of many breast cancers, the period of assessment before relapse may need

to be several years. Geyer found an excess of adverse life events in the eight years before diagnosis in patients with breast cancer compared with controls.² The study of Ramirez *et al* covered life events over a median of 30.5 months before relapse.³ A comparable figure is not given in Barraclough and colleagues' paper, but as the maximum period of assessment was 42 months the average period covered by the life events and difficulties schedule was probably shorter.

Barraclough and colleagues' patients were older $(54 \cdot 3 \text{ years})$ than those in the studies of Ramirez *et al* (49 \cdot 5 years) and Geyer (45 \cdot 5 years). These age differences may be clinically important as they are likely to be associated with differences in menstrual status, pathology, and the types of adverse life events experienced. Sixty per cent of Barraclough and colleagues' patients were postmenopausal compared with 31% in the study of Ramirez *et al*. As it may be hypothesised that psychosocial stress influences the onset or progress of cancer through indirect hormonal effects, these differences between study samples may be important in explaining contradictory results.

It would also be relevant to know what proportion of patients in both studies received tamoxifen, and their hormone receptor status. Allied to this, certain combinations of medical and surgical interventions may conceivably conceal the limited effects of psychosocial adversity on the disease process. Differences in treatment may also account for the lack of an association between life events and prognosis in certain studies.

These studies make the questionable assumption that all patients react similarly to adverse events. Yet, clearly, some people become more upset than others after the death of a spouse, loss of employment, or other major events. The link between adverse events and physical health may become clearer if an attempt is made to understand the way in which psychosocial stress interacts with personality and coping style to influence physiological processes.

The possibility of an association between psychological factors and the course of physical disease is a fascinating but clinically unimportant issue in the assessment of patients with cancer. Although future studies may confirm that adverse life events do not shorten the life span of such patients, psychological interventions can result in a significant improvement in quality of life for many of them.⁴

CHWEN CHENG CHEN THOMAS FAHY

Department of Psychological Medicine, King's College Hospital and Institute of Psychiatry, London SE5 9RS

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Psychological influences on cancer and ischaemic heart disease

EDITOR,—Anthony J Pelosi and Louis Appleby summarise recent research by Eysenck and Grossarth-Maticek about psychosocial influences on cancer and ischaemic heart disease.¹ They raise several critical questions; among them they suggest that "one is left to speculate whether the authors have made the mistake, during reanalysis of their data, of reassigning individuals to personality types after causes of death were known."

In an issue of *Psychological Inquiry*² a target