start of adequate drug treatment is a predictor of the persistence of symptoms in major depression. The aim of this study is to examine the 'no-treatment interval' variable in a group of patients undergoing first admission to two Dublin Psychiatric Hospitals, using time to remission as a measure of outcome.

Consecutive admissions to two Dublin Psychiatric Hospitals were screened. Those patients meeting International Classification of Diseases-9 (I.C.D.-9) criteria for major depression undergoing their first psychiatric admission were entered in the study. Patients with organic brain disease or those unable to give consent were excluded from the study. A cohort of 100 patients was established. Details of the index episode were obtained using the Schedules for Clinical Assessment in Neuropsychiatry (SCAN). The 'no-treatment interval' was defined as the duration between onset of the episode and the commencement of 75 mg per day of tricyclic antidepressant or equivalent. The 17 item Hamilton Rating Scale for Depression (H.D.R.S.-17) was preformed on admission, fortnightly during the admission, and three and nine months post discharge. Remission was defined as H.D.R.S.-17 < 8 for at least 2 weeks.

21 of 100 patients were excluded because of inability to date onset of episode or onset of adequate treatment, or failure to complete the study. The excluded group did not differ significantly on clinical or sociodemographic variables from the final group of 79. This final group consisted of 45 (57%) women, 34 (43%) men. The age range was 18–77, mean 41.4 (s.d 14.4) years. The 'no-treatment interval' range was 2 days to 25 months, mean 14.7 (s.d 16.5) weeks. X^2 (Chi-square) distribution showed a significant relationship between 'no-treatment interval' and time to remission ($X^2 = 5.29$. $X^2a = 3.84$, $a = 0.25$). There was no significant relationship between age, sex and social class and time to remission.

The findings of this study support the proposal that the duration of the 'no-treatment interval' is a predictor of the outcome of major depression. This underlines the importance of early and adequate treatment of depression in primary care.

β-ENDORPHIN AND IMMUNODYSFUNCTION IN DEPRESSION AND ANXIETY DISORDERS

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Objectives: To assess cell-mediated immunity in depression and anxiety disorders and to elucidate whether immunodysfunction might be related to a high opioid activity.

Methods: In a prospective study of patients with major depression (n = 34) or anxiety disorders (n = 21), cellular immunity tests, the *in vitro* effects of naloxone on monocytes, and the plasma levels of β-endorphin were investigated. Peripheral blood mononuclear cells and some monocyte parameters were determined by flow cytometry. Natural killer (NK) cell activity was studied by cytotoxicity using the K-562 cell line, γ-interferon production by a standard bioassay, monocytic phagocytosis by ingestion of *Candida Albicans* and latex, and blastogenesis by stimulation with phytohaemaglutinin.

Results: In most patients from both groups it was observed: 1) a dysfunction of monocytes, characterized by a marked reduction in the number of these cells that ingest particles and express cytoskeletal intermediate filaments and surface structures (CR1 receptors and HLA-DR antigens); 2) a monocytosis that was not able to normalize the count of normally functioning monocytes; 3) *in vitro* correction of the monocyte alterations with naloxone; 4) normal concentrations of T lymphocytes and CD4 and CD8 populations; 5) decrease in NK cell number and activity; 6) normal synthesis of γ-interferon; and 7) energy to candidin and tuberculin and a diminished lectin-induced blastogenesis. Some of these immune changes correlated closely with plasma levels of β-endorphin, which were abnormally high in all the cases.

Conclusion: A naloxone-reversible monocyte dysfunction, associated to alterations both in NK count and function and in cell-mediated hypersensitivity, was related to high circulating concentrations of β-endorphin.

IN VIVO BRAIN PET RESPONSES AND NEUROENDOCRINE ALTERATIONS FOLLOWING SEROTONIN RELEASE IN DEPRESSED PATIENTS VERSUS HEALTHY CONTROLS

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The indoleamine hypothesis of depression proposes that major depression is due to a deficiency of available serotonin or subsensitivity of key serotonin receptors in relevant brain regions. We and others have reported results from the serotonin-releasing fenfluramine challenge test which demonstrate a blunted serotonin-mediated prolactin (PRL) response to d-I fenfluramine (FEN) in depressed patients compared with normal controls. The limitations of such results are that these studies only assess hypothalmic neural pathways and do not inform us about where in the brain such serotonin changes occur.

We have recently described a methodology for visualizing *in vivo* regional brain responses to serotonin release with positron emission tomography (PET) by comparing regional brain glucose metabolism after administration of FEN, relative to placebo. We now report on differences between the neuroendocrine responses to FEN and regional brain metabolic responses (rCMRglu) following FEN in 11 patients with an untreated major depressive episode versus 6 healthy controls.

The PRL response to FEN did not distinguish between groups. However, several statistically highly significant prefrontal cortical areas of increase in rCMRglu were seen in healthy controls, whereas no significant increases or decreases in regional glucose metabolism were seen in patients. No overlap was seen in degree of response in patients compared to controls.

These results provide direct *in vivo* support for the indoleamine hypothesis of depression, and are a further step towards visualization of brain regions associated with neurotransmitter alterations that may underlie major depression.

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DEVELOPMENT OF THE COMPREHENSIVE DESCRIPTIVE AND SEVERITY SCALE OF BEREAVEMENT: THE STARDUST BEREAVEMENT SCALE

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Objective: Despite the extensive literature on bereavement, there is a scarity of Comparatative Clinical Data on grief responses whether normal or abnormal. The aim here is to design and validate an easily administered and comprehensive scale of grief.

Method: In the course of studying the effects of a mam made disaster the opportunity arose of sampling the vast majority of bereaved family members (N = 147). A literature Review narrowed the emotional and behavioural responses to 32 items. These were rated in analogue form over a two year time span and provided qualitative and quantitative measures of grief.

Results: The items and ratings were easily understood and quantified by the bereaved. Factor analysis supported hamageneity of the