<table>
<thead>
<tr>
<th><strong>Title</strong></th>
<th>Adjustment disorder: epidemiology, diagnosis and treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Authors(s)</strong></td>
<td>Casey, Patricia R.</td>
</tr>
<tr>
<td><strong>Publication date</strong></td>
<td>2009-11</td>
</tr>
<tr>
<td><strong>Publication information</strong></td>
<td>CNS Drugs, 23 (11): 927-938</td>
</tr>
<tr>
<td><strong>Publisher</strong></td>
<td>Springer</td>
</tr>
<tr>
<td><strong>Item record/more information</strong></td>
<td><a href="http://hdl.handle.net/10197/5830">http://hdl.handle.net/10197/5830</a></td>
</tr>
<tr>
<td><strong>Publisher's statement</strong></td>
<td>The final publication is available at <a href="http://www.springerlink.com">www.springerlink.com</a></td>
</tr>
<tr>
<td><strong>Publisher's version (DOI)</strong></td>
<td>10.2165/11311000-000000000-00000</td>
</tr>
</tbody>
</table>

The UCD community has made this article openly available. Please share how this access benefits you. Your story matters! (@ucd_oa)

Some rights reserved. For more information, please see the item record link above.
Adjustment Disorder
Epidemiology, Diagnosis and Treatment

Professor Patricia Casey, FRCPsych, FRCPI, MD.
Consultant Psychiatrist and Professor of Psychiatry,
University Department of Psychiatry, Mater Misericordiae University Hospital,
Eccles St., Dublin 7, Ireland

Telephone: +35318032176
Fax: +35318309323
Email: apsych@mater.ie
Abstract

Adjustment disorder (AD) was introduced into the psychiatric classification systems almost thirty years ago although the concept was recognised for many years before that. Six subtypes are described based on the predominant symptoms but no further diagnostic criteria are offered to assist the clinician. These are common conditions especially in primary care and in consultation liaison psychiatry where the prevalence ranges from 11-18% and 10-35% respectively. Yet they are under-researched, possibly due to the failure of some of the common diagnostic tools to allow for the diagnosis of AD. Among those to incorporate AD, the concordance between the clinical and interview diagnosis is very poor with the diagnosis being made more commonly in clinical practice than the diagnostic tools allow for. AD is found in all cultures and in all age groups.

The presence of a causal stressor is essential before a diagnosis of AD can be made, while the symptoms vary and include those that are found in other common psychiatric disorders. It is also important to distinguish AD from normal reactions to stressful events.

Suggestion for distinguishing AD from other diagnoses such as major depression, are made and various treatments considered. The role of psychotherapy is highlighted and also that of pharmacotherapy, although the latter has a less vital role apart from the symptomatic management of anxiety and related symptoms. There have been few studies of either treatment modality and some of these are discussed.
Adjustment Disorder: Epidemiology, Diagnosis and Treatment

This paper will examine the diagnostic criteria for adjustment disorder (AD) and outline the diagnostic process both clinically and using structured interviews. It will also discuss the differential and co-morbid diagnoses while the controversy surrounding the diagnosis itself will be considered briefly. Various approaches to management will conclude the paper. Throughout, the lacunae in our knowledge regarding AD will be flagged.

Diagnostic criteria

Adjustment disorder (AD) has been recognised since the Diagnostic and Statistical Manual, 1st edition (DSM-1)\(^1\) was introduced in 1952; although it was then called transient situational personality disorder, finally changing to AD in DSM-111 (1980)\(^2\). AD has been incorporated into the ICD classification since the 9\(^{th}\) revision in 1978\(^3\).

Despite its long history, the criteria for AD in DSM-IV TR\(^4\) continue to be vague and largely unhelpful. The core criterion is that the person must not meet the criteria for any other psychiatric condition, a bar that is set very low indeed, especially for major depression, which requires only 5 symptoms for 2 weeks. Notwithstanding this criticism, DSM-IV does specify that adjustment disorder occurs

- In response to a stressful event,
- When the onset of symptoms is within 3 months of exposure to the stressor,
- When the symptoms are distressing and in excess of what would be expected by exposure to the stressor,
- When there is significant impairment in social or occupational functioning,
- When the symptoms are not due to another axis 1 disorder or bereavement,
- When once the stressor or its consequences is removed the symptoms resolve within 6 months.

Moreover, DSM recognises that AD may be acute, if lasting less than 6 months, or chronic, if longer. Six subtypes are described based on the predominant symptom pattern and these include with depressed mood, with anxiety, with mixed depression and anxiety, with disturbance of conduct, with mixed disturbance of emotions and conduct, and unspecified. The criteria for these are not specified in greater detail.

ICD 10 has similar criteria\(^5\) but specifies that the onset is within 1 month of exposure and it specifically excludes psychosocial stressors of an unusual or catastrophic nature. Seven subtypes broadly similar to those in DSM are identified in ICD 10 but the depressive reactions are divided into brief (less than 1 month) and prolonged (less than 2 years).

Epidemiology in various populations

DSM-IV states that adjustment disorder (AD) is a common diagnosis yet the evidence for this is unclear since it is seldom measured in epidemiological studies.
General population and primary care studies: None of the major international studies such as the ECA, the National Co-morbidity Survey or the National Psychiatric Morbidity Survey included AD among the conditions examined. An exception to this was the ODIN study of depressive disorders in five countries in Europe. Using a two-stage screening method that included the Schedule for Clinical Assessment in Neuropsychiatry (SCAN) only 1% of those with depressive disorders were given this diagnosis. However, a recent study of elderly people selected from the general population identified ADs as occurring with a prevalence of 2.3%, similar to that of major depression.

ADs are said to be very common in primary care where family practitioners deal with the long-term impact of physical illness as well as the consequences of social and interpersonal problems, all of which are associated with AD. Prevalence rates of from 11% to 18% among consulters with mental health problems have been described although these studies are old and more recent studies are conspicuously absent.

Psychiatric out-patient and in-patient clinics: There are few studies of AD among psychiatric in-patients or out-patients. One study of intake assessments at a rural and urban clinics found that AD was the most common clinical diagnosis, made in 36% of those seen, but this dropped to just over 11% using SCID. Concordance between clinical and SCID diagnoses was lower for this than for any other diagnosis. Among adolescents attending an out-patient clinic almost 30% were so diagnosed. As a diagnosis among in-patients, one study identified AD in 9% of consecutive admissions to an acute public sector unit.

Among those presenting to a psychiatric emergency care team AD was diagnosed in 19.2% of women, second only to mood disorders and in 14.5% of men, fourth after “other disorders”, psychoactive substance abuse and mood disorders.

In summary, these studies show that even in the secondary care psychiatric services, AD’s are commonly diagnosed.

Consultation liaison psychiatry: A diagnosis of AD is most likely to be made in liaison psychiatry. Up to 12% of referrals to that service in several university hospitals were so diagnosed and it was considered a rule-out diagnosis in a further 10.6%, figures that resemble those of a large European study that identified AD as the primary diagnosis from 56 centres across 11 European countries. However the frequency with which AD is diagnosed in this setting seems to be declining in tandem with an increase in the diagnosis of major depression. This may not so much reflect a change in their prevalence as a change in the “culture of diagnosis” with the availability of newer antidepressants.

Among specific medical groups, studies have demonstrated that AD was almost three times as common as major depression (13.7 versus 5.1%) in acutely ill medical in-patients and was diagnosed in 35% of cancer patients experiencing a recurrence. In obstetric/gynaecology consultation-liaison, adjustment disorders predominated over mood disorders.

Deliberate self harm: Turning to those who engage in deliberate self harm, a clinical diagnosis of AD is commonly made, and this was confirmed in an emergency
department study where AD was diagnosed in 31.8% of those interviewed while major depression was less common at 19.5%. These proportions changed to 7.8% and 36.4% respectively when a structured interview (SCID) was used. One explanation for this discrepancy is that structured interviews may be overly rigid, having been designed for use by lay interviewers who might apply the criteria in a cook-book fashion. This is especially pertinent for a diagnosis such as AD which relies heavily on clinical judgement, context and on longitudinal course.

What of those with a diagnosis of AD – is there an association with self-harm? The studies to date suggest that there is. A study of adolescents and young adults with a diagnosis of AD who were attending an out-patient clinic found that 25% had engaged in a suicide attempt and compared to the non-suicidal AD patients, had a significantly greater history of prior psychiatric treatment, poorer psychosocial functioning, dysphoric mood, suicide in a significant other and psychomotor restlessness. A history of self-harm is even more common in adults with a diagnosis of AD with over 60% having such a history and over two thirds having a diagnosis of either antisocial or borderline personality disorder, both associated with self harm repetition. In short, AD carries with it the same risk factors for self harm as do other psychiatric diagnoses, so the belief that it is less serious than other axis I diagnoses is belied by these findings.

The profile of suicide attempters among those with AD as compared to major depression includes a greater likelihood of childhood deprivation, orphanhood and parental instability. The act is more likely to be carried out under the influence of alcohol, unplanned and the interval from the onset of disorder until the attempt is significantly shorter in the AD group. This is therefore a group with longstanding vulnerability and a tendency to impulsivity, that is even greater than in those with major depression. These studies all point to the role of personality disorder as a prominent feature of those with AD who engage in self harm.

Issues in the Classification of AD

There are a number of debates taking place with regard to the classification of AD. These are complex and beyond the scope of this review which is focused on the clinical aspects of AD but for completeness they will be briefly outlined here.

AD is a diagnostic category that is ring-fenced in a particular way – on one side is the differentiation from other psychiatric disorders such as major depression, somatisation or minor depression although there have been no studies comparing the latter with AD. The terms minor depression and AD may be used interchangeably since both are characterised by cognitive and mood related symptoms, rather than vegetative symptoms and both are also viewed as sub-syndromes on the trajectory to other disorders.

A debate within the broader debate relates to AD as a sub-syndrome since this excludes the possibility of it being diagnosed when the criteria for another disorder are met – hence major depression will always trump a diagnosis of AD notwithstanding the low threshold for arriving at a diagnosis of major depression. Some argue that the current subsyndromal position should continue while others
content that it should be accorded full syndromal status with its own diagnostic criteria, a position that is supported by this writer. 

With regard to distinguishing AD from major depression, somatisation disorder and others, there are conceptual difficulties since a diagnosis of AD is based on the longitudinal pattern of symptoms triggered by a stressor, that ultimately resolve, while a diagnosis of major depression or somatisation disorder is made cross-sectionally based on symptoms numbers and severity. So different dimensions, one longitudinal and one cross sectional, exert themselves in the diagnostic process. This is likely to render attempts at comparison problematic although to date no differences in symptoms between AD and major depression have been identified.

On the other side of the AD fence lie the adaptive homeostatic reactions to stressful events from which AD must also be distinguished. A system of diagnosis based simply on the presence of symptoms alone is likely to be over inclusive, capturing in its net a variety of appropriate responses to stressors. A warning note was sounded in a recent editorial: “There may well be a latent genius in these labels, for professionals, for laypersons and for society, because they represent psychiatry’s recognition of the existential limits and uncertainties of living. Beware a Trojan horse, however; these categories, if widely used, could medicalise most of life.” Surmounting this requires clinical skills that consider various domains within the symptom complex such as context, cultural norms etc. These will be considered further below (see differential diagnosis).

**Diagnosis using Structured Interviews**

Few of the structured diagnostic interviews incorporate AD. Neither the Clinical Interview Schedule (CIS), nor the Composite International Diagnostic Interview (CIDI) includes AD. The Schedule for Clinical Assessment in Neuropsychiatry (SCAN) does include AD, in Section 13 which deals with Inferences and Attributions. This comes after the criteria for all other disorders have been completed and there are no specific questions to assist the interviewer in making the diagnosis. The Structured Clinical Interview for DSM-IV (SCID) also includes a section dealing with AD but the instructions to interviewers specify that this diagnosis is not made if the criteria for any other psychiatric disorder are met. The Mini International Neuropsychiatric Interview (M.I.N.I.) also incorporates a section on adjustment disorder but, as in SCID it is trumped when any another diagnosis is made.

**Diagnosis in Clinical Practice**

Diagnosing AD in clinical practice can be difficult since there is symptom overlap between the various subcategories of AD and other psychiatric syndromes such as generalised anxiety, major depression etc. Most research in distinguishing AD subtypes from other disorders has focussed on AD with depressed mood and major depression. 

**Stressors:** The essential requirement for diagnosing AD is that the symptoms must be triggered by a stressful event and the maximum time lag in ICD-10 is 1 month and in
DSM-IV, 3 months In this regard it is similar to PTSD. For all other psychiatric disorders a stressor is not a requirement, although there is evidence\(^{38}\) that over 80% of those with major depression experience a recent life event.

Concerning the type of events, there is little to assist the clinician in distinguishing AD from other diagnoses and even events of the magnitude that are typically associated with a diagnosis of PTSD can also trigger AD. A study comparing those with major depression to those with AD identified a higher proportion of events related to marital problems and fewer to occupational or family stressors in the AD group\(^{35}\). Although statistically significant these differences are unlikely to be helpful in making the diagnosis since they are not specific to either diagnosis.

**Symptoms:** In both ICD and DSM the criteria for diagnosing AD are silent with respect to specific symptoms. Nevertheless, there are some symptoms that may be of diagnostic assistance. The loss of mood reactivity, the presence of diurnal mood change, a distinct quality to the mood change and a family history of depression might suggest a depressive episode rather than AD. This was partially supported in a study\(^{39}\) comparing subjects with major depression, with and without physical co-morbidity. Using an instrument designed to distinguish typical melancholic features from other symptoms of depression\(^{40}\) those with physical illness were less likely to experience the former, raising the possibility that the greater the environmental triggers the less likely are typical melancholic symptoms of depression to be present. Since AD represents, *par excellence*, a disorder in which environmental factors are prominent, it is possible that these symptoms will distinguish those with AD from those with more biologically determined depression. Only further studies will demonstrate if these symptoms have sufficient specificity.

With regard to the symptom of low mood itself, the mood state of those with AD often depends more on the cognitive presence of the stressor so that immediate impairment of mood is observed when the stressor is mentioned, followed by a more pronounced mood recovery when the patient is distracted.

Ultimately, due to the limitations in the criteria for diagnosing AD, the diagnosis is based on the presence of a precipitating stressor and on a clinical evaluation of the likelihood of symptom resolution on removal of the stressor.

**Differential Diagnosis**

*Distinction from normal responses:* AD is different from other psychiatric disorders since one element of the diagnosis is whether the response to the stressor is a manifestation of appropriate distress.

The failure to differentiate appropriate, non-pathological reactions to stressful events from those that are pathological could lead to normal sadness being misdiagnosed as AD or depression,\(^{41}\) simply by the presence of symptoms. In the absence of criteria distinguishing normal from abnormal responses, clinical judgement will play a prominent part in deciding whether the responses are proportionate or excessive (table 1).
Table 1 Distinguishing AD from normal responses to stressors

<table>
<thead>
<tr>
<th>Personal circumstances and context of stressor</th>
<th>Proportionality between symptom severity and triggering event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistence beyond expected duration</td>
<td>Cultural norms for emotional response/expression</td>
</tr>
<tr>
<td>Duration of and severity of functional impairment</td>
<td></td>
</tr>
</tbody>
</table>

This will have to take into account the personal circumstances of the individual and the expression of symptoms within the person’s culture. For example the loss of a job for one person might be acceptable while for another it could heap poverty on a family. Cultural differences in the expression of emotion will also need to be considered since some are more expressive than others, a knowledge of “normal” coping with illness and other stressful events is essential and the diagnostic process will be guided by the extent to which an individual’s symptoms are in excess of this, both in terms of severity and duration. For instance failure to appreciate that some cultures grant compassionate leave from work following bereavement might lead to such a person being identified as disordered in another. Finally the presence of functional impairment is also an indicator of a pathological response.

With regard to symptoms and functioning, it is recommended that these should only be regarded as excessive if the are “clinically significant” although this has not been defined and has been criticised as inadequate and tautological.

Distinction from other psychiatric disorders: Because of the symptom overlap between AD and a number of axis 1 disorders such as major depression and generalised anxiety, the possibility that these diagnoses might be present rather than AD must be considered. The failure to diagnose major depression, for instance, could have serious treatment and prognostic implications. Alternatively, diagnosing such disorders as major depression when a diagnosis of AD is more appropriate could reinforce the “culture of prescribing” even when spontaneous recovery is likely. A problem arises if the DSM diagnostic criteria are rigidly applied since once the symptom numbers and duration are reached, the diagnosis of AD cannot be made. In practice it is more likely that major depression will be over-diagnosed at the expense of AD than the converse, due to the low threshold applied to major depression.

Post-traumatic stress disorder (PTSD) and acute stress disorder require the presence of a stressor of a magnitude that would be traumatic for almost everybody and a specific symptom constellation, although these have recently been challenged. However, not everybody exposed to such traumatic events develops PTSD and the possibility that other disorders can, such as AD, occur needs to be considered.

Finally, what may appear to be an adjustment disorder, because of the sub-threshold level of the symptoms or the lack of functional impairment might be an axis 1 disorder in evolution that only emerges as a recognisable syndrome after a period of watchful waiting, especially of symptoms persist despite termination of the stressor.
For those experiencing long-standing stressors, the persistently low mood that is the response to these may be misdiagnosed as dysthymia, as enduring personality change after psychiatric illness (ICD only) or as depressive personality disorder (DSM only).

Co-morbidity

The preamble to the section on AD in ICD-10 points to the greater prominence of personal vulnerability in the aetiology of this disorder as compared to others. While this is suggestive of co-morbidity with personality disorder, the research base for this is limited. Some studies identify cognitive style as a possible contributing feature. In particular, traumatic childhood experiences are hypothesised as stimulating the perception of events as outside of one’s control, thus leading to distress and depressive symptoms. Other studies show that those with pre-existing symptoms at the time of the occurrence of the stressor may be at increased risk of developing AD when compared to those who are symptom free.

Few studies have examined the disorders that are co-morbid with AD, an exercise that is hampered by the fact that the criteria for AD preclude its diagnosis if the criteria for another condition are met. Yet a recent study found that 46.1% of patients exhibited co-morbidity and this was highest for major depression (RR 26.8) and PTSD (RR 5.1). This should not be surprising since co-morbidity is commonly associated with all psychiatric disorder and the finding may represent the co-occurrence with another disorder of different aetiology.

The relationship between substance abuse and AD is also deserving of mention since it may explain the seeming instability of the AD diagnosis. Substances may be misused for relief of symptoms such as anxiety and depression, which are prominent in AD. Alternatively substances such as alcohol are themselves depressants and may present with mood related symptoms leading to misdiagnosis. There is some evidence for the latter from a study which found that 59% of subjects diagnosed with AD were relabelled on discharge as having a primary diagnosis of substance misuse.

Treatment

There are few trials of treatment, whether psychological or pharmacological but in clinical practice the focus has been mainly on psychological interventions.

Psychological interventions

In general, brief therapies are considered the most appropriate as AD’s tend to be short lived although lengthier therapies may be required when stressors are chronic or when there is underlying personality pathology that increases vulnerability to such stressors.

There are three broad components to the psychological interventions for AD.

1. Enabling reduction or removal of the stressor
These measures consist of practical assistance in removing the stressor from the person or the person from the stressor. For example, when an individual is in a violent relationship encouraging the person to obtain protection or to leave is likely to reduce the levels of distress. Moreover, many stressors can be minimised or avoided such as when a person takes on too much work. Problem solving techniques may assist in making these decisions.

2. Measures to facilitate adaptation

When a stressor cannot be removed such as a family member caring for a sick relative, measures such as psycho education, problem solving techniques or cognitive restructuring may help reframe it.

Putting support systems in place can help a distressed person deal with problematic situations especially when it results in practical assistance, such as being available when a carer needs time off. This may involve harnessing family members’ input or encouraging involvement in a support or self-help group.

3. Altering the response to the stressor - symptom reduction/behavioural change

Relaxation techniques can reduce symptoms of anxiety and more general measures that include facilitating the verbalisation of fears and emotions and exploring the meaning that the stressor has for the individual might also ameliorate symptoms. Many who are confronted by life’s problems will engage in DSH, either due to hopelessness, anger or some other emotion. Assisting the person in finding alternative responses that do not involve self-destruction will be of obvious benefit and to date dialectical behaviour therapy (DBT) has the best evidence base.

Interventions may be delivered individually or in groups, and family or interpersonal therapy may be of value in some contexts. So in general, the psychological therapies span the range including supportive, psycho educational, cognitive and psychodynamic approaches. Although not yet tested in relation to AD, resilience enhancing techniques might also have a role.

Unfortunately the evidence base for these approaches is limited. A few studies have focussed on the elderly who are particularly vulnerable to AD’s. One utilised ego enhancing therapy during periods of transition while another used “mirror therapy,” in those with AD secondary to myocardial infarction, both with benefit.

In a younger population, cognitive therapy was helpful when administered to those with AD who experienced work-related stress, while among army conscripts it was beneficial to those with AD. In a study of terminally cancer patients similar improvements were found in those with AD and other psychiatric diagnoses.

A grey literature study of 9 subjects found benefits from eye movement desensitization.
Some of these psychological interventions have been tested in specific medically ill groups such as cancer patients, those with heart disease, HIV, and so on. While improvements in coping has been demonstrated, it is unclear if subjects had AD, some were open pilot studies and survival and quality of life rather than symptoms were the outcome measures in others. Another study confirmed the benefits of brief dynamic and supportive therapy for minor depressive disorders that included AD’s but the sample size (30) was small and diagnostically diverse.

**Pharmacological Interventions**

The pharmacological management of AD consists of symptomatic treatment of insomnia, anxiety and panic attacks and the use of benzodiazepines to relieve these is common. While antidepressants are advocated by some, especially if there has been no benefit from psychotherapy, there is little solid evidence to support their having an effect on depressive symptoms. Nevertheless those with sedative properties targeting sleep and anxiety may have a role when benzodiazepines are contraindicated such as in those with a history of substance dependence.

There are few trials specifically directed to the pharmacological treatment of AD’s and these are mainly on subjects with AD with anxiety. Some are listed in table 2.

**Table 2 Summary of medication trials in the treatment of AD**

<table>
<thead>
<tr>
<th>Author</th>
<th>Treatment</th>
<th>Sample size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nguyen et al 2006</td>
<td>Etifoxine vs Lorazepam</td>
<td>191 out-patients attending GP’s</td>
</tr>
<tr>
<td>Voltz et al 1997</td>
<td>Kava-kava vs Placebo</td>
<td>101 out-patients</td>
</tr>
<tr>
<td>Bourin et al 1997</td>
<td>Valerian and others vs Placebo</td>
<td>91 out-patients</td>
</tr>
<tr>
<td>Ansseau et al 1996</td>
<td>Tianeptine vs alprazolam vs mianserin</td>
<td>152 patients</td>
</tr>
<tr>
<td>Razavi et al 1999</td>
<td>Trazadone vs clorazepate</td>
<td>18 cancer patients</td>
</tr>
<tr>
<td>Hameed et al 2005</td>
<td>Antidepressants in Major depression vs AD</td>
<td>96 primary care patients</td>
</tr>
<tr>
<td>De Leo 1989</td>
<td>Viloxazine vs placebo vs lormetazepam vs S-adenosylmethionine psychotherapy</td>
<td>85 out-patients</td>
</tr>
</tbody>
</table>
A recent study comparing a benzodiazepine and non-benzodiazepine anxiolytic found that the anxiolytic effects of each were similar although more responded to the non-benzodiazepine.

Two randomised placebo controlled studies examined herbal remedies including extracts from kava-kava and valerian plus other extracts among out-patients with AD (with anxiety) and demonstrated a positive effect on symptoms. Two further studies, one in AD patients with anxiety found that anxiolytics and antidepressants were equally effective while a pilot study of cancer patients with anxious and depressed mood found trazodone superior to a benzodiazepine.

One study in primary care examined the response of patients with major depression and with AD to antidepressants using reported changes to functional disability based on case note information. Overall the AD group was twice as likely to respond to antidepressants. However, being a retrospective case note study the relevance of the results is questionable.

One of the few studies to compare pharmacological and psychological interventions randomly assigned 70 subjects diagnosed with adjustment disorders to supportive psychotherapy, an antidepressant, a benzodiazepine and placebo. All improved significantly.

Overall these studies lend little support for the superiority of antidepressants, and arguably for any specific treatment, in the management of AD’s but further studies are clearly required.

Finally the question of the setting in which these interventions should be delivered is important and while it might be tempting to redirect those with AD’s from the specialist services back to their primary care physicians, the demands in terms of time and skills might make this impractical. Management in a community setting in which large numbers are offered an intervention delivered by clinical psychologists is another possibility. This has been tested in those self-diagnosed as “stressed”, by providing a one-day free workshop comprising psycho-education using a cognitive approach. At three months follow-up the intervention group were significantly less symptomatic than the waiting-list control groups. This needs to be tested in those diagnosed with AD since it may have been reaching only those in the throes of normal adaptation to stressors. For the moment those diagnosed with AD by psychiatrists are best treated by members of the psychiatric multidisciplinary team with the appropriate skills.

Conclusions

AD’s are common, yet this diagnosis is made in the absence of specific diagnostic criteria, an issue that has been the subject of criticism. This lacuna has made research into the epidemiology and treatment of these conditions difficult. The diagnosis is currently one that is based on clinical judgement concerning the appropriate response to a stressful event or its consequences. It also demands a judgement that resolution
will occur when the stressor is removed. Treatments are mainly psychological but some brief pharmacological interventions have also been examined, although overall data is sparse. The fact that, despite the conceptual difficulties and diagnostic difficulties, the diagnosis continues to be made is indicative of its utility. Much work is still needed to develop evidence based interventions. Meanwhile the best evidence is for psychological treatments.

10 Wing, J. K., Babor, T., Brugha, T., et al. SCAN: Schedules for Clinical Assessment in Neuropsychiatry. Arch Gen Psychiatry, 1990. 47, 589-93.
41 Trivedi MH, Rush AJ, Ibrahim HM et al. The Inventory of Depressive Symptomatology, Clinician Rating (IDS-C) and Self-Report (IDS-SR), and the Quick Inventory of Depressive Symptomatology, Clinician Rating (QIDS-C) and Self-Report (QIDS-SR) in public sector patients with mood disorders: a psychometric evaluation. Psychol. Med. 2004. 34. 73–82.
43 Maj M. Are we able to differentiate between true mental disorders and homeostatic reactions to adverse life events. Psychother Psychosom. 2007. 76.257-9.
15

58 Fawzy FL, Canada AL, Fawzy NW. Malignant melonoma: effects of a brief structured psychiatric intervention on survival and recurrence at 10 year follow-up. Arch Gen Psychiatry. 2003. 60: 100-03.
63 Nguyen N, Fakra E, Pradel V et al Efficacy of etifoxine compared to lorazepam monotherapy in the treatment of patients with adjustment disorder with anxiety: a double blind controlled study in general practice. Hum Psychopharmacol 2006. 21, 139-49