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Prevention and early intervention for depression in adolescence and early adult life

Abstract Over the past decade there has been increasing interest in the possibility that early intervention might prevent mental disorders later in life. Indeed, in the United Kingdom the Department of Health recommends that health promotion should be one of the main functions of child mental health services, a suggestion that has been endorsed by professional bodies. It is easy to see why both purchasers and providers of mental health services would be interested in prevention, but will preventive interventions work in practice? This paper discusses the possibility of preventing depressive disorder in late adolescence and early adult life by intervening in childhood and early adolescence. The paper begins with a description of the phenomenology of depression and its risk factors. It then goes on to describe a framework of prevention and within this framework explores whether there is an adequate knowledge base. The general perspective that is presented is one of cautious scepticism. It is argued that difficulties in defining depression and identifying risk factors that can easily be remedied make it unlikely that within the foreseeable future primary prevention programmes will prove to be more effective than treatment and rehabilitation of affected individuals. The possibility that preventive programmes could do harm will also be discussed. The paper concludes with some proposals about appropriate targets for prevention. It is suggested that apart from a few policy areas where there are some relatively harmless measures that could protect from later depression, a balanced preventive programme will give higher priority to treatment services than to those concerned with early intervention.

Key words Depression · Prevention · Childhood · Treatment

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Introduction

The development of preventive programmes has been slower and more tentative in child psychiatry than in the rest of medicine. The hopes of the Child Guidance movement during the first half of this century that mental problems later in life could be prevented by intensive early intervention were not realized. Subsequently, children's mental health services in the United Kingdom became increasingly allied with hospital-based disciplines and more concerned with the treatment of patients who had established disorders than with prevention. Indeed, it was widely supposed that concepts borrowed from public health programmes for preventing physical diseases would not carry over well into the child mental health field (Kolvin 1994).

Over the past decade there has, however, been renewed interest in the possibility that early intervention might prevent mental disorders later in life. In Britain, children's mental health services have undergone major changes with an increasing focus on community-based treatment programmes and on interventions delivered through the primary care, educational or social care systems (Health Advisory Service 1995). There has also been a huge increase in the number of other services for children at risk of problems, such as counselling services for the bereaved and the abused. All these developments provide opportunities for early preventive interventions (Kurtz 1996). The government White Paper, the Health of the Nation (Secretary of State for Health 1992) has set targets for prevention, including a reduction in morbidity from mental health problems in both childhood and adulthood, and a reduction in mortality from suicide. The Department of Health in the UK therefore now recommends that health promotion should be one of the main functions of child mental health services (Department of Health 1995), which is also the view of professional bodies (Child and Adolescent Section of the Royal College of Psychiatrists 1990) and a Parliamentary Select Committee (Parliamentary Select Committee 1997).

Preventing adult mental illness by intervening in childhood is great in theory and it is easy to see why both professionals and politicians are attracted to the idea. But will it work in practice? This paper discusses the possibility of preventing depressive disorder in late adolescence and early adult life by intervening in childhood and early adolescence. The paper begins with a description of the phenomenology of depression and its risk factors. It then goes on to describe a framework of prevention and within this framework explores whether there is an adequate knowledge base. The general perspective that is presented is one of cautious scepticism. It is argued that difficulties in defining depression and identifying risk factors that can easily be remedied make it unlikely that within the foreseeable future primary prevention programmes will prove to be more effective than treatment and rehabilitation of affected indivuals. The possibility that preventive programmes could do harm will also be discussed. The paper concludes with some proposals about appropriate targets for prevention. It is suggested that apart from a few policy areas where there are some relatively harmless measures that could protect from later depression, a balanced preventive programme will give higher priority to measures directed at recognizing and treating established cases of depression.

What is being prevented?

Affective disorders include a range of neurotic and psychotic illnesses. The most useful distinction is between bipolar and unipolar affective disorders, which differ in their familial associations and epidemiology (Perris 1992). Within unipolar affective disorder, the most valuable differentiation appears to be between depressions with and without psychotic features. Depressive disorders accompanied by delusions and hallucinations have a worse outcome than non-psychotic depressions (Lee and Murray 1988). This paper is concerned with non-psychotic, unipolar depression, which is the most common form of affective disturbance (Smith and Weissman 1992).

Effective preventive programmes require clear definitions of the problem to be prevented. Indeed, the demand for such definitions has dominated psychiatric epidemiology, where great progress has been made in standardizing both methods of assessment and the way "caseness" is defined. At present, unipolar depression is usually conceptualized in epidemiological surveys as a diagnosis, the criteria for which are set out in classification schemes such as DSM-IV. The widespread use of standardized diagnostic schemes has led to much greater uniformity across psychiatric epidemiology. However, it should be borne in mind that these categorical classification systems were mainly devised for use in clinical settings. To clinicians, depression means a diagnosis, something that a patient either does or does not have. It is important that clinicians make categorical distinctions, because most clinical decisions are dichotomous. If a patient has a depressive disorder, then a course of treatment is initiated; if not, then he is reassured that all is well and sent on his way. Doctors do not prescribe a little bit of antidepressant for a little bit of depression.

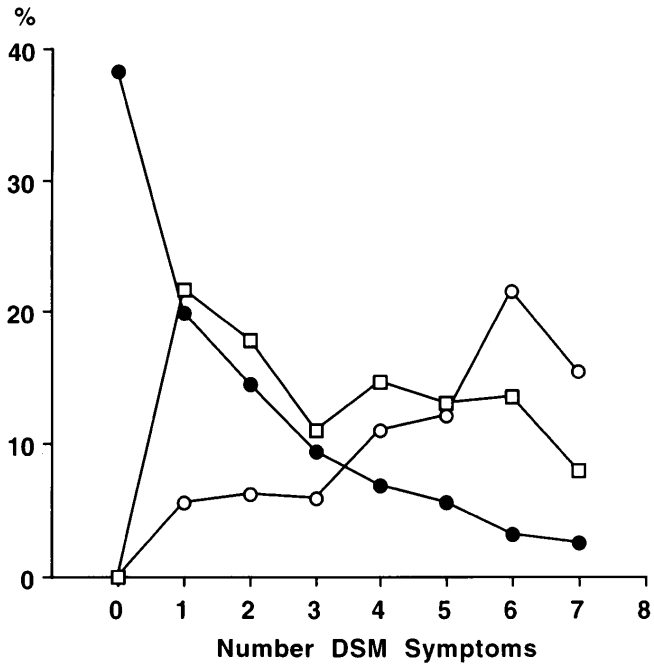

Fig. 1 Findings from a population study of adolescents showing the relationship between the number of DSM depressive symptoms and the percentage of adolescents reporting various numbers of these symptoms *(filled circles)*, the percentage of adolescents with poor school work in excess of that for non-depressed adolescents *(unfilled circles)* and the proportion of this excess at different levels of depression *(squares)*

It may not, however, be appropriate to take a categorical approach to defining depression in epidemiologically based preventive programmes. In such programmes it may be better to consider depression as a continuum. Figure 1 illustrates some of the reasons for this. It shows the data from a survey of depression in 840 11- to 15-year-olds from three London schools, which was conducted by the first author and his colleagues at the Institute of Psychiatry in London. Information from a self-report depression questionnaire was used to calculate a score of DSM symptoms of depression, such as appetite disturbance, sleep problems and so on. Information was also gathered independently from parents about the child's performance at school.

The first point is that it is not obvious where the line should be drawn between "normal" depression and depression as a disorder. In the DSM a score of five or more symptoms is taken as "caseness", but as the figure shows there is no cluster of cases at around the DSM cut-point (line with filled circles). Indeed, depression is very common in adolescents, with only a minority of them reporting no depressive symptoms. Sixty percent report at least one symptom of depression.

The second point is that in the same study it seemed that even mild depression was associated with problems of functioning. Thus, the proportion of young people judged by their parents as doing badly with school work

increased steadily at almost every point on the depression scale (line with unfilled circles). Even children with just one or two symptoms were more likely to be impaired than those without any symptoms. It is better for adolescents to have no depression at all than to be averagely depressed.

The third point is that only one third of the excess morbidity associated with depression occurred in the small number of depressed cases with a five or more depressive symptoms (line with squares). Most of the excess disability came from the larger number of young people with one to four depressive symptoms. This sets a limit on the efficacy of preventive programmes that target only high risk "cases".

It seems therefore that we may need to use both categorical and quantitative approaches to defining depression in preventive programmes. There are good reasons for taking a categorical approach to depression when the intervention involves a yes/no decision, such as whether or not someone should be prescribed antidepressants. However, it may be a mistake to assume that this operational convenience for clinicians reflects the epidemiological reality. Depression comes in all grades of severity, each with significant morbidity, and there is evidence that mild forms lead to severe forms (Harrington and Vostanis 1995). Preventive programmes should therefore be concerned with the whole spectrum of depression, and not just with depression as a disorder.

Why prevent it?

There is a strong case for preventing depression in adolescence and early adult life. Approximately 3% of adults are seen annually by general practitioners with recognized depression and the lifetime risk may be as high as 20% (Smith et al. 1992). The prevalence of depressive disorder in late adolescence and early adult life is probably less than in adulthood, but still occurs in approximately 1–2% of girls in the UK (Cooper and Goodyer 1993). Early-onset depressive disorders are associated with much social handicap (Puig-Antich et al. 1993) and once established they are very likely to recur (Harrington et al. 1995). There is also a clear association between early-onset depression and suicide: the risk of suicide for adolescents who have been depressed is around 5% (Harrington et al. 1994; Rao et al. 1993) and a high proportion of adolescents and young adults who kill themselves have been depressed (Marttunen et al. 1993). Depression is thus associated with significant mortality and morbidity.

It is often argued that preventing mental disorders will not only improve health but also save money. For example, the National Institute of Mental Health (National Institutes of Health, National Institute of Mental Health 1996) estimated that in just 1 year mental disorders cost the United States $148 billion, more than cancer and heart disease combined. Direct treatment and support costs accounted for nearly half of the costs, with most of the remainder coming from lost earning capacity. Early-onset depressive disorders are associated with long-term use of health services and with much employment incapacity (Harrington et al. 1990). Prevention of these disorders could therefore produce useful savings.

There are, however, several reasons for thinking that the savings that might come from preventing mental health problems such as depression would be much less than anticipated. Firstly, reducing the incidence of a disorder does not necessarily mean that the costs of treating it become less. The rising costs of health care can mean that even if a smaller number of patients develop a problem, they cost more to treat. Rates of heart disease have gone down over the past 20 years in the UK, but the number of bypass operations has greatly increased and heart surgeons are busier than ever. Mental health professionals are just as ingenious as heart specialists and have been developing better and longer-term forms of treatment for depressive conditions, sometimes extending over several years (Kupfer 1992). Even if preventive programmes reduced the number of depressed patients, those that remain could be very expensive to treat.

Secondly, the idea that reducing the incidence of a disorder leads to a reduction in treatment costs assumes that we live in an ideal world in which demands for care accurately reflect health needs, which themselves are closely linked to service provision. However, in the world in which most of us actually work there is only a weak relationship between the costs of mental health care and the mental health needs of young people. The money that is spent on the care of young people with mental problems is influenced by many other factors, such as public attitudes towards mental illness, newspaper reports, the location of existing services, government policies and, perhaps most importantly, the organization and attitudes of agencies who fund clinical services. In the U.S., for instance, changes in the remuneration scheme for child health professionals seem to be having more influence on the costs of mental health care than epidemiologically defined need. A similarly weak relationship between the costs of children's mental health services and the need for them is found in the UK. Figure 2 shows the relationship between the average amount of money spent on mental health services per child in each of the 15 health districts of North West England (NHS Executive North West 1997) and an index of how deprived each district is, the underprivileged area score (UPA). The UPA score, which was obtained from local statistics (Ashton 1995), is based on eight indices of deprivation from the 1991 British census. It is often used in the UK as a guide to the need for mental health services (Thornicroft et al. 1992). Higher scores indicate greater social deprivation. As the figure indicates, there is only a weak relationship between per capita spending on the mental health care of children and this index of need for services (Pearson correlation = 0.28). It is unlikely therefore that changes in the incidence of mental health problems would lead automatically to changes in spending.

Thirdly, although depression is strongly associated with lost earning capacity, it remains to be conclusively demonstrated that prevention of depression will improve earning

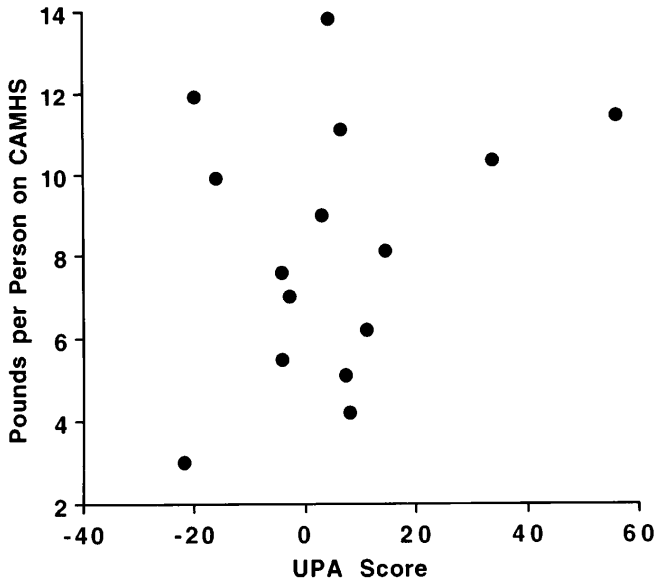

Fig. 2 Information from the National Health Service Executive of Northwest England showing the relationship between pounds per person spent annually on mental health services for children (aged 16 years or less) in each of the 15 health districts, and an index of the degree of social deprivation in that district, the Under Privileged Area *(UPA)* score

capacity. Depression is strongly associated with other problems than can influence earnings such as personality difficulties. Moreover, it may sometimes be the case that loss of job leads to depression.

The main argument for preventing depression, therefore, is altruistic rather than economic. Depression causes great suffering to many individuals and their families. It is better that young people are happy than depressed.

Risk factors and risk mechanisms

There are many risk factors for depression in young people (Harrington 1993), which can be summarized as: personal disposition, social disposition and current adversities. Personal disposition includes genetic factors (such as temperament), difficulties forming social relationships and negative styles of thinking. Social disposition includes poverty and adverse family environment. Current adversities include a wide range of acute negative life events, such as abuse, and chronic difficulties such as family discord and rejection. Since depression among the young has a high risk of recurrence, preventive programmes also need information about the factors that lead to persistence (maintaining factors), because at any given age some affected individuals will be old cases who have failed to recover. Factors that maintain early-onset depressive disorder are not always the same as those that predict its onset (Harrington and Vostanis 1995), but include greater severity of the initial disorder, older age at the time of onset, ongoing family difficulties and comorbidity with other psychiatric problems (Goodyer et al. 1997 a, b).

Identification of risk factors is an important step in the prevention of mental disorder, but several issues need to be borne in mind when interpreting the extant data for early onset depression. Firstly, in many instances it is unclear whether the association represents a risk mechanism or simply a risk factor. For example, there is a strong association between adolescent depression and poor school work (Fig. 1), but it is probably the case that school problems are a cause as well as a result of the depression, and both of course could be due to a third factor, such as lack of parental encouragement.

Secondly, although there is broad agreement that depressive disorders at all ages are multifactorial, we are still a long way from understanding how risk factors combine to produce the disorder. Most current models propose that risk factors, such as genetic disposition and stressful life events, combine interactively. Thus, for instance, genetic factors could influence the risk of major depression by influencing the vulnerability of individuals to the depressive effects of adverse life events. This kind of model, also known as a vulnerability model, has obvious attractions for preventive programmes because an intervention targeted at the way individuals deal with adversity could in theory prevent not only the risk that stems from the adversity itself but also that stemming from the genetic disposition. In such models genetic effects are not deterministic, but are manifest only in the presence of some external stressor.

It has, however, been difficult to reliably demonstrate that risk factors for depression work interactively. Thus, for example, in a recent North American twin study of major depression in adult women it was found that most of the effects of stressful life events and genetic liability were independent of each other (Kendler et al. 1995). Only a relatively small part of the effect of genes and environment could be explained by an interaction between the two. The statistics used to explore the ways that risk factors combine may underestimate the magnitude of interactions (Kendler 1995; Kendler et al. 1995). Nevertheless, the evidence thus far suggests that in considering the contributions of genetic and environmental risk factors to the cause of depressive disorder, additive effects are important. In other words, the overall risk for depressive disorder is in part simply the sum of genetic and environmental risk factors. This means that a preventive programme must tackle several of these risk factors to have a significant impact on the rate of depression. There may be no single point at which to aim an intervention.

Thirdly, many risk factors for depressive disorder in young people occur at a low frequency in the general population and the risk that can be attributed to any single factor is often quite small. For example, only approximately 3% of children will experience the death of a parent before the age of 16 years (Garmezy and Masten 1994) and once adjustment is made for other risk factors the relative risk of depression in bereaved children is probably no more than 50% greater than in the non-bereaved (Brent et al. 1994). This means that even if every case of depression after bereavement was prevented by therapy, the

overall number of depressed cases would fall by less than 2%. (From information on the base rate of a risk factor and the size of risk that is poses for disorder it is possible to calculate the attributable fraction, which is the degree to which the incidence of disorder could be reduced if the risk factor were eliminated).

Types of preventive activities

Traditional public health definitions of prevention (Caplan 1964; National Academy of Sciences 1994) recognize three kinds of preventive activity. Prevention (primary prevention) involves activities that reduce the incidence of the disorder in those who have not already developed it. Treatment (secondary prevention) includes case identification and standard treatment for established disorder. Maintenance treatments (tertiary prevention) have a similar objective but aim to reduce the risk of recurrence of disorder and to reduce complications arising from it.

There has been much debate as to whether treatment and maintenance activities qualify as prevention proper or whether they are better regarded simply as parts of routine clinical work (Newton 1988). In our view such strategies do qualify as prevention simply because they do prevent something. Depressive conditions in young people are recurrent and frequently accompanied by secondary complications such as suicidal behaviour. Secondary and tertiary preventive strategies could therefore prevent much of the morbidity arising from depression. It will be appreciated, however, that for many mental health problems the distinction between "prevention" and "treatment" is blurred. This is a particularly important issue when depression is the target of the intervention because the boundary between being a "case" and being a "non-case" of depressive disorder is difficult to define. As Fig. 1 shows, there is no obvious point of discontinuity between "symptoms" and "disorder", and most of the morbidity associated with depression occurs in people with symptoms rather than in those with major depressive disorder. In this paper all interventions that target depressive symptoms are therefore referred to as "treatment" whether or not these symptoms are severe enough to warrant the diagnosis of depression. The term prevention is reserved for interventions that target risk factors, rather than the symptoms of depression itself.

Two main types of primary preventive strategy are recognized, universal and selective.

Universal prevention

Universal preventive interventions are aimed at the whole population, regardless of whether or not they have risk factors for a disorder. Childhood immunization is a good example of a universal intervention for infectious diseases such as diphtheria. Because universal interventions are aimed at everyone in the population they have the potential to produce large changes in disease incidence. Moreover, for problems such as depression in which morbidity is distributed across much of the population, nearly everyone benefits a little. Such interventions are also less stigmatizing than selective strategies.

Problems using universal strategies to prevent depression

What kinds of universal strategies could prevent depression? The first point is that in many instances we do not know what to do. Some risk factors for depression are unavoidable. For example, it is of little use advising people to avoid threatening life events such as bereavement. For other risk factors we do not understand the mechanisms that underlie their association with depressive disorder. Until we do it would be easy to waste resources changing risk factors that are not directly involved in causing the disorder. For instance, since deliberate self-harm (DSH) in adolescence is strongly associated with poverty and educational disadvantage (Kerfoot et al. 1996), it seems obvious that improvements in standards of living should reduce the incidence of DSH. It could be argued therefore that a good way to improve the emotional well-being of children would be to put more money into education and housing. In fact, the massive improvement in the standard of living of most Europeans during this century has been accompanied by an increase in rates of depressive disorder and suicide (Diekstra 1993; Fombonne 1995). This suggests that, although poor living standards may increase other risk factors (such as family dysfunction and breakup), the effects on depressive disorder are indirect. For instance, it may be that inequalities in living standards are more important than the absolute standard of living.

The second problem is that even if we knew what would be desirable, would we be able to put it into practice? Let us suppose for instance that research shows that parental skills training given to the parents of primary-school-aged children prevents depression and other adverse outcomes for the children when they reach adolescence. Let us further suppose that in light of these findings, it is decided to implement parenting programmes widely by, for example, offering courses to all parents through the child health surveillance system.

There are, however, at least two reasons for thinking that such universal interventions would have limited impact on the overall risk for subsequent psychopathology. Firstly, compliance in the general population may be low because if we are honest with the participants, we would have to point out that a decision to take part is likely to make only a slight difference to a particular parent's chances of having a troubled child. For most universal preventive interventions many must participate to prevent the adverse outcome in a few. This phenomenon has been stated as the "prevention paradox": a preventive measure that brings large benefits to the community offers little to each participating individual (Rose 1981). People are unlikely to be motivated by programmes in which the main purpose is to prevent an outcome several years later for only a few of the people who take part. Such programmes

would have to be designed to have more immediate and tangible benefits to the parents or the child, or both.

Secondly, it is a common irony of universal preventive programmes that those at highest risk of having later problems are the least likely to participate. For instance, in a study of home visitation for pregnant mothers, Osofsky and colleagues (Osofsky et al. 1988) found that the benefits were only obtained by those who accepted visiting, and these mothers were the least disadvantaged group in the first place. In an investigation of two school-based primary preventive programmes for depressive symptoms in adolescents, Clarke and colleagues (Clarke et al. 1993) reported that in both studies subjects who withdrew from the programme were significantly more depressed than those who were retained.

It is possible therefore that the so-called universal programmes would be taken up by only a minority of the general population, and that the children in this minority were at low risk of subsequent psychopathology.

The third problem is lack of information about which universal interventions are most protective against later depression. We shall discuss later some examples of policies and interventions that might in theory be protective against later depression because they target modifiable risk factors for the disorder. However, few of these has yet been demonstrated to be effective in preventing depressive disorder even in the short term, let alone in the long term. It will be appreciated that this does not mean we should not support them. There are plenty of examples in public health where the decision to introduce a new policy did not await a lifetime of scientific investigation. The adequacy or otherwise of the evidence about a preventive intervention needs to be balanced against the uses to which that evidence will be put. For instance, low-cost policies to encourage continuity of parenting are unlikely to be harmful and would probably produce small benefits for many mental disorders. The costs of conducting preventive trials to evaluate the effects of such policies on any single problem such as depression are likely to be huge and so time-consuming that it would not be worth the effort.

Harmful effects of universal prevention programmes

Nevertheless, there is a case for conducting clinical trials on some universal preventive measures, if only because some of them could be harmful. It is often assumed that reasonable psychosocial interventions can only bring benefits and cannot do harm. This is not the case. To begin with, universal preventive interventions can be indirectly harmful to the extent that they cost money and this money could in theory be spent on some other form of intervention, such as a targeted intervention to those at higher risk. If the net benefits from the universal intervention are outweighed by those of the targeted intervention, then the universal intervention will in one sense be harmful because the overall burden of mental disorder in the community would be higher than if the money had been spent only on the targeted intervention.

Of greater concern is the direct harm that can occur from psychological interventions. Since a universal intervention offers only a small benefit to each participating individual, it would be all too easy for this small benefit to be eliminated by a small risk coming from the intervention itself. For example, in the 1980s there was in the United States a rapid growth of suicide prevention programmes (Garland et al. 1989; Shaffer et al. 1988), many of which aimed to increase awareness of the problem of adolescent suicide among all students. However, there is little evidence that these broad programmes do any good, and they may do harm by upsetting some students or even by normalizing suicidal behaviours (Shaffer et al. 1990).

Examples of universal preventive strategies

Clearly, there are reasons for caution in the development of universal preventive programmes. However, this does not mean that we should do nothing. We know enough about the mechanisms of early-onset depressive disorders to support some interventions that might be effective. For example, data from a variety of different sources show that young people who go in and out of foster homes or who are reared in institutions with multiple caretakers have an increased risk of depression and many other problems in later life (Brown et al. 1986; Rutter et al. 1990). Those from a similar background who are adopted or who receive long-term fostering in an ordinary family environment are more likely to develop normally (Hodges and Tizard 1989). For obvious reasons there cannot be controlled trials of different forms of parenting, but the indirect evidence is strong that better continuity of parenting would lead to important benefits. Policies that support permanency of placement should therefore be protective against later psychopathology, and would be safe.

It is important not only to reduce disadvantage suffered by children who grow up in adverse family environments, but also to reduce the risk of unwanted births. Children born to teenage mothers are at greatly increased risk of psychosocial problems. Moreover, teenage pregnancy appears to be an important mechanism linking childhood adversity to depression in early adult life. This is probably because early pregnancy increases the likelihood of other problems such as marriage to an abusive partner (Quinton et al. 1984). Measures to improve family planning could therefore have a useful preventive effect.

It may also be useful to include children in universal preventive programmes run through schools. The present evidence suggests that approaches directed at reducing depressive symptoms are not effective (Clarke et al. 1993). However, programmes that aim to increase the individual's competence and self-esteem may be more productive. There are theoretical reasons for thinking that increased social competence and positive attitudes may buffer an individual from the depressogenic effects of traumatic experiences (Cole 1990; Hammen 1991). A potential advantage of such approaches is that they should be acceptable to young people and their families since there is an immediate and obvious gain for the child. This

should help to ensure that they are widely available. Such programmes would in theory also prevent other problems such as behavioral difficulties, which would certainly make them attractive for schools.

Selective preventive strategies

One of the disadvantages of universal preventive strategies is that people who are not at special risk will be troubled unnecessarily. This is a special problem in the prevention of depressive disorder because as we saw in the previous section it is still not clear which universal measures will be effective. Moreover, because universal interventions must by definition be applicable to everyone, even if they are at low risk, they may be of relatively little benefit to those at greatest risk.

An alternative strategy is to target interventions towards high-risk groups, so-called selective interventions. In the context of the prevention of depression, two types of selective intervention can usefully be distinguished, vulnerability-focused interventions and event-centred interventions (Newton 1988).

Vulnerability-focused selective interventions

Vulnerability-focused interventions aim to improve the resilience of young people who are at risk for depression, either by direct work with the child, or indirectly by working with the child's family. Probably the best established risk factor for depression is having a family history of that disorder. This association is partly due to genetic factors but almost certainly is also a reflection of family environmental processes such as parenting problems and family discord (Harrington 1996). There are several examples of interventions that have targeted families in which at least one parent is depressed. For instance, Beardslee and coworkers (1993) described an educational programme for depressed parents that was designed to be administered by the clinician treating them. The programme aims to modify some of the psychosocial pathways that are thought to mediate the links between parental depression and psychiatric disorders in their offspring, such as poor communication. Preliminary data indicate that the clinician-facilitated programme was significantly better at changing the behaviour and attitudes of families than a lecture intervention (Beardsless et al. 1996). Another approach is based on befriending schemes such as Newpin and Homestart (Cox 1993; Cox et al. 1991). Volunteers are recruited from the local community, trained part-time for a few months, and then paired with client mothers, who are often depressed. There is also a centre with a creche that mothers can drop into when they want. The aim is to reduce maternal depression, child neglect and abuse.

Since early-onset depressive disorders are likely to have a significant genetic influence, it is possible that genetic counselling aimed at the avoidance of birth of high-risk individuals may eventually become a preventive measure. However, limited knowledge about the modes of inheritance, limited heritability and the absence of reliable genetic markers mean that in the short term genetic counselling is not likely to be a useful preventive tool. Depression at all ages has a complex inheritance and risk predictions are currently unreliable. Even in bipolar disorder average morbid risks to offspring are low (Harrington 1996), and since these disorders are treatable, it is hard to justify the avoidance of children.

A well-established vulnerability factor for depressive disorder is nondepressive psychiatric disorder. For example, around one fifth of children with depressive disorder also have a conduct disorder, and longitudinal studies suggest that the depression is often superimposed on the conduct problems (Harrington et al. 1991). Treatment of conduct disorder could therefore reduce the risk of subsequent depressive disorder.

Event-centred selective interventions

Event-centred interventions focus preventive activities on events that are thought to be linked to depression such as sexual abuse, bereavement or other traumatic experiences. The idea here is to help children through trauma or grief using psychological techniques such as debriefing. These techniques may require skilled therapists such as psychotherapists or family therapists (Harris-Hendricks et al. 1993), but there are now many organizations that offer self-help packages and advice for these vulnerable young people and their families.

As noted previously, some of these events are rare and carry only a relatively low risk of subsequent depression. Prevention of these events or treatment of their psychological consequences can therefore have only a limited impact on the total incidence of depression later in life. There are, however, some events that appear to have a substantially higher attributable risk for subsequent depressive disorder. For instance, retrospective studies of depressed adults have repeatedly shown a strong association between sexual abuse and depression in early adult life (Mullen et al. 1993), with an attributable risk of around 14% for major depression (Fergusson et al. 1996). This is much higher than the risk attributable to bereavement (D.M. Fergusson, per. commun.).

Advantages of selective interventions

Selective interventions have several advantages over universal programmes. Since they attempt to target only vulnerable individuals, the dangers of harming people who are not at risk are lower. This may be important in the prevention of problems such as deliberate self-harm, where there is concern that universal programmes may make matters worse because they normalize such behaviours (Shaffer 1994). In addition, it is possible for the intervention to be matched to the special needs of the young person and family. Thus, disputing parents can be urged to

stop arguing, the isolated child can be invited to attend extra-curricular activities and the depressed parent can be encouraged to attend a treatment programme. Advice that is appropriate to the needs of the child and family is likely to enhance motivation better than advice that is not personally relevant.

Problems of selective interventions

Selective interventions, like universal interventions, also have problems. The main problem is that data are lacking on whether or not selective psychological interventions are in fact protective against later depressive disorder. Thus, for instance, there have been no systematic large-scale randomized controlled trials of treatment for sexually abused children (Finkelhor and Berliner 1995), data from studies of bereavement programmes are contradictory (Schneiderman et al. 1994) and clinical trials of treatment for depressed parents are at much too early a stage to determine whether or not they will prevent depression in the children. This is a serious problem because the purpose of selective preventive strategies is not to pick out children at risk, but rather to pick out children whose risk is reversible. We could do more harm than good if we select children for interventions that turn out to be ineffective.

Indeed, there are several reasons for being concerned about the potential of selective interventions to do harm. Firstly, since children are selected out because they are high risk they may suffer unnecessary stigmatization. Secondly, there is the direct harm that could come from the intervention itself. There are plenty of examples from research on behavioural problems and delinquency in which well-intentioned selective interventions have been associated with a worse outcome than routine care (Berg et al. 1978; Cadman et al. 1987; McCord 1978; O'Donnell et al. 1979). For instance, Berg (1978) looked at the effectiveness of regular recall to court compared with counselling from a social worker in 96 children brought before the juvenile courts in Leeds, England, because of poor school attendance. Children were randomly allocated to these conditions and followed-up for 6 months. Children dealt with by regular recall to the court truanted less and also committed fewer offenses.

Much less is known about the adverse effects of selective preventive interventions for children who are at risk of emotional problems. However, recent research with adults in the UK warns us of the potential that these interventions have for doing harm to people at risk of emotional symptoms. Hobbs and colleagues (1996) in Oxford randomly allocated more than 100 adult victims of road traffic accidents, a group at high risk of anxiety and depression, either to psychological debriefing (PD) or to routine care. The intervention group had a worse outcome in terms of symptoms of post-traumatic stress. In another British study, Bisson and coworkers (Bisson et al. 1997) reported that adults who had PD after burns had a significantly worse psychosocial outcome than cases given no intervention.

Thirdly, there are the indirect negative effects that could occur if resources from other areas are diverted into selective preventive interventions of unproven benefit. For example, it has been suggested that all traumatically bereaved children should be seen as an emergency by mental health clinics (Black 1993). If research shows that interventions for such children do provide substantial protection from future psychopathology, then this could turn out to be a wise use of scarce clinical resources. In the meantime, however, for many mental health services in the UK this would mean that bereaved children, many of whom do not have a significant mental disorder, were given priority over non-bereaved children with established mental conditions.

It will be appreciated that this does not mean that no help should be offered to children who are at risk. If the child and family are asking for help and the child has symptoms, then it is good practice to offer advice and, perhaps, some ongoing support. However, on present evidence selecting out children only on the basis of risk factors, such as traumatic events, may not be an effective preventive strategy.

Another disadvantage of selective preventive strategies is that interventions confined to a high-risk group could not prevent the majority of cases of depressive disorder because no risk factor identified thus far is found in more than 50% of depressed cases. Thus, for instance, even strong risk factors, such as having a first-degree relative who has a depressive disorder, occur in less than one third of depressed adolescents (Harrington 1996).

Early interventions for depressive symptoms

There is evidence from studies of depressed adults that the earlier a depression is treated the better the outcome (Kupfer et al. 1989). Another approach to the prevention of early onset depressive disorder is therefore to intervene in the early stages of the process, when the adolescent has depressive symptoms but has not yet developed the disorder. The best-studied strategy has been to identify adolescents who have high scores on a depression questionnaire and to offer them a course of treatment. Preliminary data from studies of such interventions are encouraging. Jaycox et al. (1994) reported that in children identified as high risk because of depressive symptoms or maternal conflict, a school-based programme of 12 treatment sessions led to a significant reduction in depressive symptoms. Clarke and colleagues (1995) found that adolescents at risk of depressive disorder by virtue of depressive symptoms had a significantly lower risk of disorder after a group cognitive intervention than those who did not have treatment.

Early interventions have three main advantages over selective approaches. Firstly, there is evidence that at least in the short term they prevent the target outcome. Secondly, because symptoms of depression are usually better predictors of depressive disorder than other risk factors (Lewinsohn et al. 1994), early interventions have greater

potential to reduce the incidence of disorder. Thirdly, because most of the morbidity associated with depression comes from the large numbers of people with depressive symptoms, rather than from the small number of cases with depressive disorder (Fig. 1), they have the potential to produce large gains for the whole population.

Problems with early interventions

Early interventions also have disadvantages. The first problem is that some early preventive programmes do not fit well with the adolescent culture. Adolescents who attend group therapy for treatment of depressive symptoms would be regarded by many peer groups as a little cranky, and it is not surprising to find that in some studies compliance with treatment was poor. For instance, in the school-based study by Jaycox and colleagues (1994) less than 20% of cases who were eligible entered the trial. Compliance can be good in studies based on schools (Kolvin et al. 1981), and the school provides a good setting in which to screen young people for depression. Nevertheless, interventions that are not part of the adolescent culture will be difficult to set up.

The second problem concerns the related issue of sustaining programmes for preventing psychological problems within settings such as schools. For instance, it is notable that although the Newcastle studies showed many years ago that psychological treatments carried out within the education system worked (Kolvin et al. 1981), in the UK school-based mental health programmes have seldom lasted for more than a few years. Indeed, in the UK things seem to be going the other way, with an increasing number of problems that would in the past have been dealt with through the school psychology service being referred to mental health services (Parliamentary Select Committee 1997).

The third problem is that although depressive symptoms are better predictors of depressive disorder than other risk factors, their predictive power is still only modest and many cases with depressive symptoms would have to be treated to prevent one case of depressive disorder. Consider, for example, the results of the study by Clarke and colleagues (1995). After treatment, the risk of having an affective disorder was 14.5% for the intervention, compared with 25.7% in the control condition, a reduction of 11%. This means that nine high-risk cases would need to be treated to prevent one case of depression. [The "number needed to treat" can be calculated as 1/(absolute risk difference).] Of course, this ratio is not bad compared with other preventive interventions. A physician treating mildly hypertensive patients would need to treat something like 40 cases for 20 years to prevent one stroke (MRC Working Party 1985). Nevertheless, preventing depressive disorder by treating all adolescents with depressive symptoms would be a huge undertaking, particularly when one considers that most of them have at least one symptom (Fig. 1).

The hazards of screening

The fourth problem concerns the hazards that can occur from screening. Screening programmes generally affect a large number of people relative to the number who benefit. A small adverse effect of screening on the large numbers of people who are not helped by the intervention could therefore outweigh the gains achieved by the small number who have treatment. For instance, young people who are screen positive but who do not attend for treatment, a high proportion in some studies of juvenile depression (Jaycox et al. 1994), will probably suffer unnecessary anxiety as a result of knowing that they have a problem that requires treatment.

Screening programmes can also mean that depression becomes "professionalised", with young people who may previously have seen themselves as healthy having to see themselves instead as in need of treatment. Thus, an adolescent who gets spells of dysphoria which she and her parents had previously thought were a normal part of growing up is labelled as "depressive". This may be necessary to prevent the disorder, but for many people the issue of being labelled as having a "mental problem" is a significant stigma. This is a particularly difficult issue in school-based interventions, where the label may also become known to peers.

Even cases who are screen negative may be adversely affected by the screening process. We described previously the issue that screening for a problem, such as suicidal behaviour, may introduce this idea to young people who had not previously thought much about it. Research on screening for illnesses such cancer and heart disease reveals many other kinds of unexpected and potentially deleterious effects from screening. One of these is the "certificate of health effect" (Tymstra and Bielman 1987), which suggests that people who have received a negative result on screening may be more resistant to advice on healthy lifestyles. Similar kinds of problems could occur in screening for mental disorders. For instance, people who score just below the screening threshold on a depression screening questionnaire may be falsely reassured that their depressive symptoms are nothing to worry about and therefore less likely to seek treatment when more substantial psychological problems develop.

Treatment of established disorder

At first sight, it seems counter-intuitive that treatment of established depressive disorder could have any part to play in a prevention programme. After all, the whole point of a prevention programme is to prevent disorder. However, findings from recent research on the epidemiology of depressive disorder in adulthood suggest that vigorous treatment of early-onset disorder could have a significant impact on the prevalence of depression later in life. Depressive disorders tend to be recurrent conditions, which means at any given age a significant proportion of cases of depression will be old cases that have recurred. In the

Oregon study of mental disorder in late adolescence (average age 16.5 years), for example, around one third of incident cases during a year had had a previous episode (Lewinsohn et al. 1993), with an average age of onset of around 13 years. Looking forwards from age 13 years, we found that the relative risk of having an episode of depressive disorder in adult life was approximately six times higher in depressed cases than psychiatric controls (Harrington et al. 1990). If we assume the prevalence of depression at age 13 years is around 3%, and if we were able to treat all cases of depressive disorder at age 13 years, then we could in theory reduce the risk of depressive disorder at age 16 years by around 10%. Few other interventions have this potential.

Preventive strategies that are based on the treatment of established disorders have several other advantages. The main advantage is that there is evidence that in some cases depressive disorders in adolescents can be effectively treated, at least in the short term. Thus, controlled trials have shown that psychological treatments, particularly cognitive-behaviour therapy (CBT), are better than either no treatment or a comparison intervention (Fine et al. 1991; Lewinsohn et al. 1990; Wood et al. 1996). A recent trial with one of the "new" antidepressants has also produced encouraging results (Emslie et al. 1995).

Another advantage of treatment strategies is that they are easier to justify to politicians and the public than pure preventive strategies. Treatment strategies are based around individuals and as every politician knows, the public's perception of need is often conceived in personal terms. The personal stories of troubled children successfully treated are likely to be of greater public appeal than learned articles about the benefits to the general population of primary preventive strategies. After all, the main outcome in primary prevention is not developing a problem, and people without problems may not even known that they have been helped!

Problems with treatment as a strategy for prevention

Treatment strategies do, however, have several disadvantages. The most important is that even with intensive treatment a substantial minority of depressed adolescents fail to respond. For example, in a study of cognitive-behaviour therapy for adolescent outpatients with major depression Wood and colleagues (Wood et al. 1996) found that even after 6 months around one third of cases were still in episode. Follow-up studies of clinical samples of depressed cases, who would often have had intensive treatments of one kind or another, have found that around 10% are still depressed after one year (Harrington and Vostanis 1995).

The second problem is that since most of the extant treatment programmes are based on clinical services, it will be difficult to target that majority of depressed adolescents who never have contact with these services (Cooper and Goodyer 1993). Efforts are needed to increase the recognition of depressive disorders in high-risk groups, such as those attending general practitioners.

The third problem is the adverse effects of treatment. These are most obvious for pharmacological treatments, such as the tricyclic antidepressants, but can also occur with psychological interventions. For instance, in our studies of CBT with depressed adolescents (Kroll et al. 1996; Vostanis and Harrington 1994; Wood et al. 1996) we have usually found at least one or two adolescents who find the therapy too distressing and have to discontinue.

Maintenance strategies

Maintenance strategies (also known as tertiary prevention) aim to reduce disabilities arising as a consequence of disorder. Three kinds of maintenance strategies can be identified: (a) prevention of relapse and recurrence: (b) elimination of residual disabilities such as social impairment; and (c) prevention of complications such as deliberate self-harm or secondary disorders such as alcohol abuse.

Prevention of relapse and recurrence

It is becoming clear that there is a high risk of relapse in adolescents who have remitted from major depressive disorder. Short-term follow-up studies show relapse in 30% or more of outpatients. Long-term follow-ups also show a high risk of recurrence, with more than 50% of cases having another episode (Harrington et al. 1995).

In adults there is a lot of evidence that continuation of antidepressants for at least 6 months after remission will prevent relapse (Prien 1992) and longer-term maintenance has also been shown to be of value (Kupfer 1992). Much less is known about the value of such continuation treatments in adolescent major depression. However, preliminary data indicate that the continuation of CBT after remission from major depression may be effective in preventing relapse (Kroll et al. 1996).

Elimination of residual disabilities

Although juvenile depressive disorders tend to be relapsing and remitting disorders they are often associated with a variety of disabilities that persist after remission. For instance, Puig-Antich and colleagues (1985 a, b) found that impairments of peer relationships persisted for months after remission. Kandel and Davies (1986) reported that self-ratings of dysphoria in adolescence were associated with impairments in intimate relationships in early adult life.

Very little is known about either the origins or the treatment of these residual disabilities. Few treatment trials give much information on outcomes such as social functioning. In our research global functioning improved significantly more after treatment with CBT than after relaxation training, but there were no specific effects of treatment on comorbid symptoms such as conduct problems and anxiety (Wood et al. 1996).

Prevention of complications

The most serious complication of depression is of course suicide. Depressed young people very commonly have suicidal thoughts and some of them make suicidal attempts. Mitchell and colleagues (1988) reported that nearly 40% of depressed young people had made a suicidal attempt. This increased risk of suicidal behaviour seems to extend into adulthood. We found that nearly 30% of depressed children made a suicidal attempt in adult life (Harrington et al. 1994) and Rao and colleagues (1993) reported that the risk of completed suicide was significantly higher in depressed children than in anxious controls.

It is not clear how best to reduce the risk of suicidal behaviour in depressed children, but since suicidal feelings are an important part of the clinical syndrome of depression, it is reasonable to assume that active treatment of depressive disorder should reduce the risk of suicidal behaviour. It is also important to introduce measures that reduce the opportunity for serious self-harm. For example, in the longitudinal study by Rao and coworkers (1993) three of the seven suicides were by tricyclic antidepressant overdose. There is probably now a case for restricting the use of tricyclics to cases who have failed to respond to other forms of treatment.

Maintenance strategies share many of the same problems as treatment strategies. There is the additional problem that thus far they have not been properly evaluated.

Conclusion

It is evident that prevention of depression is a complex problem and that for many interventions we lack the scientific evidence on which to base our decisions. Nevertheless, it is possible to reach some tentative conclusions about what should be included or excluded from a strategy to prevent depression in early adult life.

The first point is that such a strategy needs to be concerned not only with the prevention of those severe forms of depression that clinicians call depressive disorders but also with milder forms of the problem. As Fig. 1 shows, most of the morbidity associated with depression arises from individuals who are depressed but who do not have the disorder. Reducing the average depression score of the whole population by a small amount is likely to produce greater overall gains than dramatic symptom reductions in a few highly symptomatic groups. It follows that universal programmes directed at the whole population will have an important role in the prevention of depression. At present, there is little empirical evidence to guide us in choosing between the various universal interventions that are available and each has weaknesses as well as strengths. Policies designed to improve quality and continuity of child care are likely to have a beneficial effect and should be safe, but have the disadvantage that their effects are very difficult to measure. School-based programmes to improve children's sense of competence and social skills may also be useful, but they are likely to be expensive and difficult to sustain in educational systems that at least in the UK are hard pressed even to meet the child's educational needs.

The second point, however, is that in the absence of good evidence that primary prevention of depression works it will continue to be important to recognize and treat children with established depressive disorders. In the past depressive disorders have been under-recognized in clinical samples (Harrington et al. 1990). This was probably because there was little evidence to support the validity of the concept both in terms of prediction and in terms of response to treatment. However, recent research suggests that at least in adolescents the concept of depressive disorder has substantial predictive validity (Harrington and Wood 1995), and controlled trials in clinical samples suggest that a variety of treatments are effective (Emslie et al. 1995; Fine et al. 1991; Wood et al. 1996). There is also a case for taking a longer-term view of the management of depressive disorders in young people (Harrington and Vostanis 1995) and preliminary findings suggest that long-term treatments are both feasible and effective (Kroll et al. 1996). There is therefore a case for spending more resources on recognizing and treating established cases of depressive disorder.

The third point is that much greater attention needs to be paid to the adverse effects of preventive interventions. All the interventions reviewed here could have adverse effects, but this potential for doing harm is seldom recognized. It is particularly important to investigate the safety and effectiveness of interventions for young people who are not actively seeking help for depression, but who have been selected on the basis either of risk factors such as bereavement or because of high scores on depression questionnaires. Even when randomized trials show that such interventions are effective, we need to know whether the gains are outweighed by the adverse effects of the interventions. It is not enough to know that an intervention works. There should be a balance sheet of gains and losses for each intervention. Table 1 shows an imaginary balance sheet for cognitive-behaviour therapy given to 1000 adolescents with high levels of depressive symp-

Table 1 Imaginary balance sheet comparing the gains and losses from giving cognitive-behaviour therapy to 1000 adolescents with high levels of depressive symptoms to prevent depressive disorder

Gains	Losses
100 fewer depressive episodes	1000 hours to screen schools
2 fewer suicides	15000 adolescent hours of group therapy
30 fewer suicide attempts	2000 therapist hours to run the groups
	90 cases upset by stigma of therapy
	30 extra cases of anxiety caused by screening

toms in order to prevent depressive disorder. It shows that to make a rational decision about the efficacy of a preventive intervention we need to know much more about the problems of these interventions. Unless we pay attention to these potential adverse effects of preventive interventions, we could end up doing more harm than good.

The final point concerns the balance between the various preventive strategies that have been outlined in this paper. At the moment primary prevention is being oversold in the UK. Amidst dramatic warnings of the "epidemic" of psychopathology among both children and adults, there are increasing demands for more resources to be spent on primary prevention. Indeed, the introduction of the joint purchasing of child and adolescent mental health services (CAMHS) by both health and social services has been accompanied by suggestions that resources should be diverted from traditional CAMHS towards community-based primary prevention programmes. The idea is that primary preventive approaches will reduce the need for treatment services. This view is mistaken. There are opportunities for primary prevention and CAMHS could make a contribution to the enterprise. However, we are a very long way from the point where preventive activities will reduce the incidence of mental disorders to a level where the work of treatment services will be altered significantly.

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