J. Child Psychol. Psychiat. Vol. 32, No. 7, pp. 1063–1080, 1991
Printed in Great Britain.

0021-9630/91 $3.00 + 0.00
Pergamon Press plc

Comorbidity in Child Psychopathology: Concepts, Issues and Research Strategies

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Abstract—Epidemiological data show that the co-occurrence of two or more supposedly separate child (and adult) psychiatric conditions far exceeds that expected by chance (clinic data cannot be used for this determination). The importance of comorbidity is shown and it is noted that it is not dealt with optimally in either DSM-III-R or ICD-9. Artifacts in the detection of comorbidity are considered in terms of referral and screening/surveillance biases. Apparent comorbidity may also arise from various nosological considerations; these include the use of categories where dimensions might be more appropriate, overlapping diagnostic criteria, artificial subdivision of syndromes, one disorder representing an early manifestation of the other, and one disorder being part of the other. Possible explanations of true comorbidity are discussed with respect to shared and overlapping risk factors, the comorbid pattern constituting a distinct meaningful syndrome, and one disorder creating an increased risk for the other. Some possible means of investigating each of these possibilities are noted.

Keywords: Comorbidity, child psychiatric disorder, epidemiology, classification

Introduction

Every medical student is taught that, whenever possible, a single diagnosis should be made (Jaspers, 1963; Kendell, 1975). Patients may present with multiple diseases but, when there is complex mixed symptomatology, an unusual presentation of a single disorder is more likely than the simultaneous occurrence of two or more unrelated conditions (comorbidity). Yet in child psychiatry, all epidemiological studies that have examined the issues have shown that comorbidity is extremely common (Anderson, Williams, McGee & Silva, 1987; Kashani *et al.*, 1987; Flament *et al.*, 1988; Szatmari, Boyle & Offord, 1989; Weissman *et al.*, 1987). The same has been found in adult psychiatry (Boyd *et al.*, 1984). In this paper, we consider the empirical findings, concepts, and research implications of this important issue.

Accepted manuscript received 28 *September* 1990

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Frequency of Comorbidity

The first question is whether the observed rate of comorbidity in epidemiological surveys exceeds that expected by chance alone. The expected rate is obtained by multiplying the base rates of each of the separate conditions involved in the comorbidity patterns studied. The epidemiological studies undertaken by Anderson *et al.* (1987) and Kashani *et al.* (1987) both presented their findings in a manner that allows this calculation. The data are summarized in Table 1a and 1b.

Table 1a. Base rates of disorders in three community studies that serve to calculate the expected number of comorbid cases

Author	*n*	Age	Disorders	Base rates
Anderson *et al.* (1987)	785	11	Oppos/Conduct d.	9.2%
			Anxiety d.	7.5%
			Attention deficit d.	6.7%
			Depressive d.	1.8%
Kashani *et al.* (1987)	150	14-16	Oppos/Conduct d.	14.7%
			Anxiety d.	8.7%
			Depressive d.	8.0%

d. = disorder.

Table 1b. Expected and observed number of comorbid cases in two community studies

Author	Number of disorders	Expected cases (E)	Observed cases (O)	O/E	95% C.I.
Anderson *et al.* (1987)	1	—	100		
	2	17.5	27		
	3	0.62	4		
	4	0.007	8		
	Total > 1	18.43	39	2.1	1.5–2.9
Kashani *et al.* (1987)	1	—	12		
	2	4.74	7		
	3	0.17	7		
	Total > 1	4.91	14	2.85	1.7–4.8

95% C.I. = 95% confidence interval.

Thus, in the Anderson *et al.* (1987) survey, 7.5% of 11-year-olds showed an anxiety disorder and 6.7% an attention deficit disorder; the expected comorbidity between these two disorders therefore is the product of 7.5% and 6.7%, namely 0.5%. By summing all possible combinations, the overall expected comorbidity rate for different numbers of conditions can be obtained (Table 1b). It is evident that the observed comorbidity rate was more than double that expected by chance; in the Kashani

et al. (1987) study the excess was even greater. The data used in these calculations (as presented in the original papers) were based on pooled diagnostic groupings. For example, all the various anxiety disorders were combined to form one grouping; the same applied to depressive disorders and to oppositional/conduct disorders. This means that comorbidity within these pooled categories was not taken into account. Accordingly, the true observed comorbidity rate must have been considerably higher, so that the calculated two- to three-fold excess over chance expectation constitutes a gross *under*estimate of the frequency of comorbidity. These two reports were chosen because they presented their findings in a fashion that made it easy to calculate observed and expected comorbidity rates. However, other epidemiological studies are agreed in showing a very high comorbidity rate and it may be concluded that the observed co-occurrence of supposedly separate child psychiatric disorders far exceeds that expected by chance alone.

The fact that the pooling of diagnoses greatly underestimates the true rate of comorbidity is evident in the adult epidemiological data presented by Boyd *et al.* (1984). For example, the population base rate for panic disorder was 67 out of 11,176, 0.6%. However, out of 266 individuals with major depression, 30 had a panic disorder, a rate of 11%, representing a huge increase over chance expectation.

In drawing this conclusion, we have restricted our attention to epidemiological data because general population base rates are essential for the calculation of expected comorbidity rates. Because much of the discussion of comorbidity in the literature is based on the findings of studies of clinic samples, it is necessary to note that clinic data alone cannot provide information on whether or not the observed comorbidity rate exceeds that expected by chance. A simple example serves to illustrate the point. Suppose that disorders A and B are both present in 10% of the general population and that there is a 5% rate for 'pure' A, 5% for 'pure' B and 5% for the comorbidity pattern of A + B. As the comorbidity expected by chance is 1% (i.e. 10% × 10%), this means that the observed comorbidity rate is five times the chance expectation.

Let us also suppose that all children with these disorders are referred to clinics without any referral bias (and that there are no other disorders). The clinic pattern will, therefore, be the same as that in affected members of the general population; namely 33% with disorder A, 33% with disorder B and 33% with A + B. If the clinic base rate (instead of the general population rate) is used to calculate the comorbidity expected by chance, the expectation is 67% × 67%, i.e. 45%. That is, instead of the true five-fold excess, the clinic data misleadingly appear to indicate that the observed rate is *less* than that expected by chance. Clinic data can be used to assess comorbidity only if the general population rates for each disorder are known *and* if data are available on the clinic referral rate and biases for each disorder.

In that connection, it should be noted that, whenever less than all subjects with disorder are referred, clinic samples will always contain a disproportionately large proportion of patients showing comorbidity (Berkson, 1946). That is because the referral likelihood for subjects with disorders A and B will be a function of the *combined* likelihood of referral for each disorder separately. That is so irrespective of referral biases. However, it is known that in practice there *are* various referral biases and these also need to be taken into account. For example, Shepherd, Oppenheim and

Mitchell (1971) found that children were more likely to be referred if there was also parental psychopathology or family problems.

For all these reasons, clinic data need to be used with considerable caution in studying comorbidity. However, provided that epidemiological data have already shown that there is a significantly raised comorbidity, and provided that enough is known on referral influences to consider their possible effects, clinic data may be used to examine possible explanations for comorbidity patterns.

Does Comorbidity Matter

Comorbidity has been largely ignored in the research literature for years and it is necessary to ask whether it matters sufficiently for detailed attention to be paid to it now. There are two main reasons why a failure to pay attention to comorbidity may lead researchers to draw quite misleading conclusions. First, a study of condition X may produce findings that in fact are largely a consequence of the ignored comorbid condition Y. For example, Anderson *et al.* (1987) found that of the 14 children with depressive disorders, no less than 11 had at least one other psychiatric condition as well. Indeed eight out of the 14 children showed depression *and* an anxiety disorder *and* a conduct disorder *and* an attention deficit disorder! It follows that the correlates, outcome or genetic features reported for childhood depression could in reality be those of attention deficit or conduct disorders.

The second reason is that, when comorbidity is ignored, the implicit assumption is made that the meaning of condition A is the same regardless of the presence or absence of condition B. As shown below, that is an unsafe assumption and in some circumstances it appears to be mistaken.

It might be thought that the solution would be to exclude comorbid cases in order to focus on "pure" groups. However, the extent of comorbidity is such that often this would result in the investigation of tiny atypical samples. For example, as already noted, only three out of 14 cases of depression were "pure" in the Anderson *et al.* (1987) study, and among the 12 cases of depression in the Kashani *et al.* (1987) investigation none was pure. Clearly, that cannot constitute a general solution; moreover, necessarily it involves a loss of the opportunity to determine the reasons for comorbidity, a search that could throw important light on aetiological mechanisms (see below).

DSM-III-R and ICD-9

The two major psychiatric classification systems, the American Psychiatric Association's (1987) DSM-III-R and the World Health Organization's (1978) ICD-9 (soon to be superceded by ICD-10—W.H.O., 1990), follow quite different approaches to diagnosis (Rutter & Gould, 1985; Rutter, 1988). While the W.H.O. system allows multiple diagnoses, it tends to discourage them by its adoption of a pattern approach to diagnosis. The clinician is expected to review the overall clinical picture made up of history, signs, symptoms and laboratory findings; and then to match this picture

with the prototypical diagnostic pattern that fits best, using a process of pattern recognition. It follows the long-standing medical tradition of recognizing that most disorders involve a complex admixture of specific and nonspecific symptomatology and that, in ordinary circumstances, it is more likely in practice that a patient will have one disease, rather than several. It provides the unifying explanation of protean symptomatology. Thus, there is an appreciation that there are many nonspecific symptoms—such as fever, tachycardia, fatigue, headache and skin rashes—that occur in many quite different somatic diseases. In much the same way, psychiatry has many nonspecific symptoms such as anxiety, depressed mood, poor concentration and restlessness. Of course, a dilemma arises from the fact that many of the nonspecific symptoms also constitute the hallmarks of specific diagnostic entities. The problem lies in the difficulty of deciding when, say, depression is an indicator of a major depressive disorder and when it is just an indication that psychopathologically something is the matter.

The strength of the W.H.O. approach is that the underlying concept is probably correct in many, perhaps most, instances. Thus, in reality, it is unlikely that a high proportion of patients truly have three, four or five entirely separate conditions. The outstanding weakness, however, is that, for many symptom patterns, the data are not available to determine when and how to give precedence to one diagnosis over another (Rutter, 1988). In some instances, this is dealt with by having a combination code. For example, it has long been recognized that there is a need to code schizoaffective disorder (that applies to DSM-III-R as well, although the details of criteria are not quite the same). More controversially, ICD-10 (W.H.O., 1989) has a code for depression combined with conduct disorder. A further limitation of the W.H.O. approach (at least as exemplified in ICD-9; ICD-10 has come closer to DSM-III-R) is that true patterns of comorbidity may be concealed and, therefore, neglected or that a wrong hierarchical principle may lead to invalid diagnoses.

The APA classification DSM-III-R works the other way round (although its predecessor, DSM-III, included more exclusionary hierarchies). Diagnoses are made on algorithms based on specified symptom constellations without regard to the presence or absence of accompanying symptomatology of a different kind (apart from a few exceptions). The consequence, perhaps the inevitable consequence, of this convention is that when a patient has any one diagnosis, there is usually at least one other diagnosis as well. The first obvious disadvantage of this system is that it contravenes common sense. Indeed, Weinstein, Stone, Noam, Grimes and Schwab-Stone (1989) found that, in a child psychiatric in-patient unit sample, strict adherence to DSM-III rules (freed of hierarchies) led to 78% of the patients showing comorbidity, but this fell to 20% when clinicians were allowed to use their own judgement. However, there are two other disadvantages. Although, in theory, a statistical system could be devised to present data on all possible patterns of comorbidity, no system has been used that way to date. Moreover, even if such data were produced, their complexity would make it extremely difficult, if not impossible, to make sense of the huge number of possible double, treble, and quadruple combinations.

The second disadvantage is that although DSM-III-R is supposedly free of diagnostic hierarchies that conceal comorbidity, in fact it includes a bewildering mix of inconsistencies on which combinations are, and which are not, allowed. For example,

in adults it is not allowed to diagnose overanxious disorder in the presence of generalized anxiety disorder, but it is allowed to diagnose both separation anxiety disorder and overanxious disorder in children. Or again, it is not allowed to diagnose both social phobia and avoidant disorder but it is possible to diagnose both social phobia and agoraphobia. Similarly, oppositional disorder cannot be diagnosed with conduct disorder but it can be diagnosed with attention deficit disorder. Evidently, there are more diagnostic hierarchies in DSM-III-R than is usually appreciated.

Detection Artifacts

Referral factors

Before considering possible reasons for different patterns of comorbidity, it is necessary to note the variety of ways in which it can be produced artifactually. As already discussed, there is the Berkson (1946) effect by which, for statistical reasons separate from referral biases, the comorbidity rate in clinic samples will always be greater than that in the general population whenever only a small proportion of the conditions making up the comorbidity pattern are referred to clinics—the state of affairs with the majority of child psychiatric disorders other than the most severe (Rutter, Tizard & Whitmore, 1970). In addition, of course, referral biases may further distort clinic data on comorbidity. For example, when a clinician is known to have a special interest in a particular pattern of comorbidity such cases are more likely to be referred to him or her. Similarly, tertiary referral centres seeing difficult and complicated cases are likely to have a disproportionately high level of comorbidity.

Screening and surveillance factors

It is important also to recognize that any general population epidemiological study that relies on screening or surveillance procedures is also open to possible detection artifacts. Thus, it is a common practice to use high scores on questionnaires designed to tap a broad range of behaviour as a means of picking out subjects with a high probability of disorder, who may then be studied individually in greater detail (Newman, Shrout & Bland, 1990; Rutter, 1989a,b). The procedure works well for many purposes but it will tend to miss monosymptomatic disorders and oversample children whose psychopathology include symptoms of many different types. A similar detection bias will apply whenever the diagnosis of a second disorder is dependent in part on subjects with one diagnosis being subjected to a closer degree of surveillance. This is particularly likely to operate in longitudinal studies investigating patterns of comorbidity over time (rather than concurrently).

These detection artifacts are all open to systematic quantified investigation by means of comparisons with total population unscreened samples.

Nosological Considerations

The usual concept of comorbidity implies the co-occurrence of two independent conditions or disorders. Even if the statistical data have not been distorted by detection

artifacts, the apparent overlap between supposedly different disorders may not represent comorbidity as usually conceptualized. This possibility arises because the basic nosological concepts may themselves be mistaken. As Bukstein, Brent and Kamliner (1989) pointed out, one of the major difficulties in studying psychiatric comorbidity is the lack of well validated diagnostic criteria.

Categories or dimensions?

The first possibility is that the concept of disorder (or disease) categories may itself be misconceived. Instead, as many psychologists have argued, it could be that psychopathology is best thought of as the end product of an admixture of extremes of personality dimensions. According to this view, disorders involve no qualitative discontinuity between abnormality and normality but rather a pattern resulting from quantitative variations on a range of behavioural dimensions. In so far as that is the case, apparent comorbidity is bound to arise from the inevitability of individuals with high scores on two or more dimensions; however, the extent of such apparent comorbidity will be much affected by the particular cut-off points used to define "disorder" and by the extent to which the definitions of disorder involve truncation of dimensions. The same circumstances will arise if the behavioural dimensions operate as risk factors for disorder. For example, numerous studies have shown the strong overlap between attention deficit (hyperactivity) and conduct disorders (Szatmari *et al.*, 1989; Taylor, 1988). Does this imply comorbidity between two different disorders or rather does it mean that both inattention/overactivity and aggressivity are risk factors for disruptive behaviour?

There has been surprisingly little research that has set out to contrast and compare the validity of dimensional and categorical approaches. However, one test would be to determine if behavioural dimensions related to one diagnostic category functioned as a risk factor for the second condition at levels below the diagnostic threshold. This was the approach followed by Robins and McEvoy (1990) in examining whether conduct problems before the age of 15 years predicted substance abuse. Their results showed that they did so at all levels of severity, indicating that conduct problems functioned as a dimensional predictor. Thus, of those with no conduct problems, 38% exhibited substance abuse; of those with one conduct disorder problem, 52% did so; of those with two conduct problems 66% did so; and so on. At least in this population, with these two "disorders", a dimensional approach seemed to account for the findings better than comorbidity between two separate disorders. This research strategy warrants greater usage.

Overlapping diagnostic criteria

A second type of nosological confusion arrives from the fact that the *same* item of behaviour appears in the list of diagnostic criteria for several *different* diagnostic categories—a problem highlighted in previous considerations of comorbidity (Pfeffer & Plutchik, 1989) and by the DSM-III Child and Adolescent Psychiatry Working Party (Shaffer *et al.*, 1989). For example, not surprisingly, anxiety/worrying appears in some form in the lists of criteria for all the various supposedly separate anxiety

disorders; just as depressed mood forms part of the criteria for dysthymia as well as major depressive disorder. In addition, agitation is one of the criteria for anxiety, depression, and attention deficit/hyperactivity disorder. This is not unreasonable as so many of the behaviours that define specific disorders are also nonspecific indicators of psychopathology. Nevertheless, the fact that this is so will lead to a degree of artifactual comorbidity. A related problem arises from the fact that so many mood states involve mixed emotions. Thus, it is well known that anxiety and depression very commonly occur together regardless of the diagnosis (Grayson, Bridges, Cook & Goldberg, 1990). In so far as an increasing severity of disorder is likely often to involve an increasing number of nonspecific indices of psychopathology (Costello *et al.*, 1988; Bird *et al.*, 1988; Weissman, Warner & Fendrich, 1990), and in so far as these form part of the sets of criteria for different disorders, there is some danger that there will be an artifactual association between severity and extent of comorbidity. That is simply because severe disorders with many symptoms are likely to have a greater chance of fulfilling the criteria for more than one disorder. It is possible to provide a partial check on whether any severity–comorbidity association is real or artifactual by determining whether the association holds when severity is defined in terms of degree of social impairment rather than number of symptoms. A partial test of the influence on comorbidity of nonspecific indices of psychopathology is also afforded by determining whether the correlates (with respect to features such as family history, prognosis, treatment response etc.) of the behavioural item (e.g. depression or anxiety or restlessness) are similar when it occurs as part of a mixed clinical picture to when it occurs as part of the pure syndrome it defines.

Artificial subdivision of syndromes

A somewhat similar problem may arise from the tendency to subdivide disorders defined in terms of one main symptom complex into various subcategories according to particular elements or facets of that complex. For example, anxiety disorders are subdivided into some dozen different syndromes characterized by the generality of the anxiety (e.g. "overanxious disorder" or "generalized anxiety disorder") or its specific focus (e.g. "separation anxiety disorder" or "social phobia") or the presence/absence of some particular feature (e.g. agoraphobia with or without panic), or its association with some stresses (e.g. "post-traumatic stress disorder" or "adjustment disorder with anxious mood"). So far as children are concerned, there is the additional problem that sometimes there are two disorders defined in clearly similar terms, one of which is intended to apply to all age groups and one of which is supposedly particular to childhood, but which are *not* mutually exclusive under the age of 18 years (e.g. "overanxious disorder" and "generalized anxiety disorder"). Not surprisingly, numerous studies (see Barlow, 1988) have shown extensive comorbidity between these various anxiety disorders. Most published investigations have concerned adult patients but the same situation seems to apply in childhood (Last, Hersen, Kazdin, Finkelstein & Strauss, 1987). Thus, in the study cited, half the children with separation anxiety disorder also had overanxious disorder and 95% of this comorbid group had at least one other diagnosis as well! There have been some attempts to test the discriminant validity of these different anxiety disorders

(see Barlow, 1988) with some modest success with respect to at least some of the differentiations. For example, Mannuzza, Fyer, Liebowitz and Klein (1990) have reviewed the evidence suggesting that social phobia is meaningfully distinctive from panic disorder and agoraphobia in adults. However, they also point out that the meaningful interpretation of symptoms requires that they can be considered in the individual context (something that is difficult with a symptom check list approach to diagnosis). Less is known about the validity of differentiations among anxiety disorders in childhood. Last *et al.* (1987) found that children with separation anxiety disorders tended to be somewhat younger than those with overanxious disorders but this could reflect age effects on patterns of manifestation rather than a difference between two distinct disorders. Nothing is known on whether there is any meaningful distinction between "overanxious disorder" and "generalized anxiety disorder" and the comorbidity between them seems to have received no attention, perhaps because child psychiatrists tend to choose the category in the children's section of DSM-III-R without checking to see whether the all-ages diagnosis might equally apply.

However, the issue of comorbidity cannot be dealt with merely by testing the discriminant validity of the separate anxiety categories; it is necessary to go on to examine the characteristics of the comorbid groups as they relate to the "pure" diagnoses. For example, it could be that it is useful to separate simple phobias that arise in the absence of generalized anxiety from generalized anxiety disorder but, equally, it might well be the case that high levels of general anxiety tend also to lead to various focused phobias as well. At least, the possibility needs testing. The very high level of comorbidity between different anxiety disorders suggests that some of it represents nosological confusion.

It may well be clinically useful to be able to note that an anxiety disorder has several different facets but, if these do indeed represent varied aspects of the same basic disorder, it seems misleading to view them as examples of comorbidity. Clearly, further research is needed to bring better order into the confusing nosological territory of anxiety disorders.

One disorder represents an early manifestation of the other

A further possibility is that one disorder constitutes an early manifestation of the other. When that is the explanation, it may be desirable to code these manifestations separately because the stage of the disorder has important clinical implications. But it would make no sense to regard transitional phases with both early and later manifestations as representing comorbidity. There are well known examples in medicine where distinct stages of a disease are recognized; for example, primary, secondary and tertiary syphilis or the differentiation between cervical carcinoma and precancerous cervical dyskaryosis or dysplasia. In child psychiatry, there are several disorders in which it has been suggested that they represent early manifestations of some other diagnosis. Thus, oppositional disorder is a syndrome mainly diagnosed in younger children and which often seems to be a precursor of conduct disorder (Loeber & Lahey, 1989). Similarly, separation anxiety tends to be diagnosed in younger children and it has been suggested that it may represent an early manifestation of overanxious

disorder (Last *et al.*, 1987). Or again, conduct disorder in childhood is an established precursor of antisocial personality disorder in adult life (Robins, 1978).

To test this type of disorder progression hypothesis it is crucial to have longitudinal data (Loeber, 1990). Thus, if condition A is a precursor of B, it must be the case that the presence of A at time 1 increases the likelihood of B at time 2; and that B never precedes A. However, equally, it is to be expected that only some cases of A will develop into B and, if there is more than one precursor of B, there may be instances of B that have not been preceded by A. To date, although there are some data suggesting the plausibility of hypotheses on progression, decisive testing has yet to be undertaken.

One disorder is part of the other

It may also be suggested that one disorder is part of or a secondary manifestation of the other conditions. For example, DSM-III-R specifies that if there is a pervasive developmental disorder, neither attention-deficit hyperactivity disorder, nor pica, nor overanxious disorder can be diagnosed as well. Presumably this is on the basis of the fact that the symptoms characteristic of these other disorders are so very frequently part of the symptomatology of autism. A similar exclusion applies to generalized anxiety that occurs only during the course of a mood disorder. There are several ways in which this hypothesis may be tested. For example, if the comorbid disorders are episodic in nature, it is possible to determine whether the remission and recurrence of the one are associated in time with those of the other and, especially, if the administration of a treatment that is specific to the one leads to the loss of the symptoms of the other disorder. This approach has been followed in the case of the co-occurrence of nocturnal enuresis and other child psychiatric disorders—with largely negative findings (Shaffer, 1973, 1985). It has also been adopted to examine the comorbidity of depression and conduct disorder (Puig-Antich, 1982; Puig-Antich, Lukens, Davies, Goetz & Todak, 1985), with inconclusive mixed findings. Unfortunately, this strategy is weakened by the weakness and/or nonspecifity of so many treatments; thus, antidepressant medication is not particularly effective in the treatment of depression in children and adolescents (Puig-Antich *et al.*, 1987; Ryan *et al.*, 1986) and tricyclic drugs have a wide range of actions that extend far beyond those on mood. A further limitation is that the two constellations of symptoms may represent *alternative* manifestations of one disorder; variable expression is a well recognized feature of many genetic disorders (as illustrated, for example, by neurofibromatosis). This possibility may be examined through the use of genetic research strategies. There are many examples of the use of family studies for this purpose; for example to examine the association between anorexia nervosa and depression (Strober, Lampert, Morrell, Burroughs & Jacobs, in press) or that between alcoholism and depression (Merikangas *et al.*, 1985, 1988)—in both cases with evidence suggesting independent transmission. However, on their own, family data cannot make a satisfactory differentiation between genetic and environmental mechanisms. Adoptee and twin designs of various kinds (Rutter *et al.*, 1990) are effective for this purpose and may be used to examine comorbidity. For example, Holland, Hall, Murray, Russell and Crisp (1984) found no tendency for affective disorders to occur in the cotwins of subjects with anorexia

nervosa and Cadoret, Troughton, Moreno and Whitters (1990) found no direct genetic connection between alcohol and antisocial problems in biological relatives and depressive symptomatology in adopted-away subjects. It would be helpful to make greater use of genetic research strategies in the study of comorbidity in child psychiatry.

Possible Explanations of True Comorbidity

The explanations considered this far all involve a mechanism by which the apparent comorbidity represents some type of artifact so that the real situation does not involve the co-occurrence of two or more truly separate and independent conditions. As discussed, that does not mean that the findings should be dismissed; quite often elucidation of the meaning of the apparent comorbidity should throw light on the nature of the disorders involved. However, that is even more the case when there is true comorbidity. Several rather different underlying processes need to be considered.

Shared risk factors

One possible reason for overlap between two disorders is that they share the same risk factor or factors. This possibility arises from the fact that many psychiatric disorders are multifactorial in origin and that many causal factors are not diagnosis-specific. For example, there are various extreme temperamental traits or constellations of traits that are associated with a range of psychiatric disorders as well as with learning difficulties (Kohnstamm, Bates & Rothbart, 1990). Thus, it has been suggested that temperamental variables such as overactivity, short attention span and impulsivity might account for the well established comorbidity between conduct disorders and reading difficulties (Yule & Rutter, 1985). Family adversity might also operate in the same way (Offord, Poushinksy & Sullivan 1978; Richman, Stevenson & Graham, 1982), because large family size and social disadvantage carry an increased risk for both conditions (Rutter & Giller, 1983; Sturge, 1982). However, it is not known whether the shared risk factors mechanism does in fact account for this (or any other) pattern of comorbidity. What are needed are investigations in which comorbidity is examined before and after partialling out, or statistically taking account of, shared risk factors.

Overlap between risk factors

A variant of this mechanism is provided by the possibility that, even when the risk factors for two disorders are distinct and different, there may be comorbidity because the risk factors themselves are associated. When that is the case, the individual may be at risk for two separate conditions with the risk mechanisms for each independent, but co-occurring. For example, parental depression constitutes a risk factor for a range of different child psychiatric disorders (Rutter, 1989b). Particular attention has been focused on major depressive disorders in the offspring with the suggestion that they are genetically mediated (Weissman *et al.*, 1987; Weissman, 1988). However, there is also an increased risk of conduct disorder that seems to be a function of family discord, which is much more frequent when one or both parents are depressed

(Rutter & Quinton, 1984). Thus, it might be suggested that the comorbidity between depression and conduct disorder could arise, at least in part, because parental depression is associated with a genetic risk for depression in the offspring and an environmentally mediated risk (when there is associated discord) for conduct disorders. In order to test this hypothesis, it would be necessary to examine comorbidity after taking account of these two routes to disorder in the child; thus there should be no comorbidity when there is family harmony and cohesion if the postulated mechanisms are operating in the way hypothesized.

Chiles, Miller and Cox (1980) provided data that are consistent with the risk factor overlap concept. Delinquent adolescents with and without comorbid depression were compared. The two groups could not be differentiated on the basis of risk factors for delinquency (a family history of divorce, parental death, child abuse and incest) but the depressed delinquents did differ in being more likely to have factors thought to constitute risk variables for depression (depression or alcoholism in a first degree relative). Thus, it could be argued that depressed delinquents had the risk factors for both disorders whereas the nondepressed delinquents had them for only one. It is of importance to notice here that assortative mating may also be another mechanism by which two disorders are transmitted to the offspring.

The comorbid pattern constitutes a meaningful syndrome

The basic assumption that appears to underly the DSM-III-R policy of making diagnoses on the presence of particular constellations of symptoms irrespective of the presence or absence of other constellations is that comorbidity does not alter the meaning of any of the diagnoses involved in the comorbidity pattern. That assumption may be tested by comparing comorbid and "pure" diagnoses on features such as course or family history or responses to treatment that might reflect diagnostic meaning. This strategy has been used relatively infrequently but there are two examples of syndromes where the few available data seem to negate the assumption. There is a large body of data demonstrating that stimulant drugs usually bring about major, sometimes dramatic, short-term benefits in children with attention deficit hyperactivity disorders (ADHD) (Klein, 1987). Taylor *et al.* (1987) showed that the benefits were *not* evident when the system pattern included marked anxiety; and Pliszka (1989) found that children with ADHD comorbid with anxiety had *no* significant benefit from methylphenidate in a double blind control trial (whereas those without anxiety showed the usual good drug response). The findings from both these studies are striking for two separate reasons. First, as they showed, comorbidity with conduct disorder had no effect on drug response, so that this is not a nonspecific effect of comorbidity of any type. Second, the co-occurrence of anxiety disorder *removed* one of the most characteristic features of one of the two disorders in the comorbid pattern. The implication seems to be either that the comorbid pattern constitutes a variety of anxiety disorder (systematic comparisons with "pure" anxiety disorders are needed to test the hypothesis) or that it constitutes a meaningfully distinctive syndrome in its own right. Either way, the implication is that the comorbidity has altered the meaning to be attached to ADHD and, therefore, that it warrants separate coding in the classification system.

A second example is provided by the comorbid pattern of depression and conduct disorder in childhood. The follow-up into adult life undertaken by Harrington, Fudge, Rutter, Pickles & Hill (in press) showed that the risk of adult criminality associated with conduct disorder was unaffected by the presence or absence of comorbid depression. However, the risk of major depressive disorder in adult life associated with childhood depression *was* affected by comorbid conduct disorder; the low risk in the comorbid group did not differ from that in the group without depression. Puig-Antich *et al.* (1989) also found that the comorbid group differed from children with a "pure" depressive disorder with respect to family history of major depressive disorder. This difference in familiality applied only to the comorbidity with conduct disorder and not to that with separation anxiety. The findings to date are too sparse to warrant firm conclusions and in any case do not differentiate between the alternative hypotheses that comorbid depression and conduct disorder constitute a meaningfully different syndrome and that the depression in the comorbid group is secondary to or part of the conduct disorder. Again, however, it seems that the comorbid pattern may change the meaning of the depressive disorder; it needs to be identified as a separate group and studied further.

One disorder creates an increased risk for the other

A further possible explanation for comorbid patterns is that one disorder creates an increased risk for the other. For example, Cadoret *et al.* (1990), using an adoptee design, showed that an adult diagnosis of antisocial personality or substance abuse was associated with a four-fold increase in risk for depressive symptomatology even though the genetic origins of the two disorders were distinct. Robins and McEvoy (1990), using retrospective data from the Epidemiological Catchment Area Study, showed that conduct disorder in childhood was a powerful predictor of later substance abuse. The data were not such as to allow determination of the extent to which these were truly separate disorders. However, the evidence suggested that the mechanisms involved both duration and timing of exposure to illicit drugs, plus, probably, the generation of psychosocial stressors and adversity that serve as risk factors for heavy use of psychoactive substances.

There is an extensive literature on the effects of acute and life stressors as risk factors for psychiatric disorder in both childhood and adult life (Brown & Harris, 1989; Goodyer, 1990). However, less attention has been paid to the origins of these stressors (Champion, 1990; Rutter, 1986). There is increasing evidence that people shape and select their own environments and that various psychiatric disorders play a part in generating stress and adversity. For example, follow-up studies of children with conduct disorder have shown markedly increased rates of unemployment and marital breakdown in adult life (Robins, 1966, 1986), both well established risk factors for depressive disorders. Neuroticism has also been found to be associated with an increased likelihood of marital breakdown (Kelly & Conley, 1987).

It will be appreciated that an adequate testing of this causal chain hypothesis requires several steps. Clearly, a start is provided by longitudinal time sequence data showing that condition A both precedes and is associated with an increased risk for condition B; whereas the reverse does not apply. However, as clearly noted, this could simply

mean that A and B constitute different stages in the progression of a single disorder. Data showing that comorbid A + B has correlates that approximate those of "pure" A, but not those of "pure" B, indicate that B cannot have caused A (a strategy used to examine the comorbidity of conduct disorder and reading retardation—Rutter *et al.*, 1970) but, although consistent with the hypothesis that A caused B the findings are open to other interpretations.

In particular, this pattern of results indicates that the meaning of "pure" B and comorbid A + B must be somewhat different. What are needed are data showing that the origins of "pure" B and comorbid A + B involve the same risk factors but that condition A generates those risk factors. In other words, it is necessary to test the several links in the postulated causal chain. This full research strategy has yet to be pursued in the investigation of comorbidity in child psychiatric disorders.

Conclusions

Many studies document the pervasiveness of patterns of comorbidity in child, as well as adult, psychiatry. However, it is clear that the extent of comorbidity can be assessed only through the use of fully representive epidemiological data. That is primarily because calculation of the chance expectation of comorbidity requires data on the general population base rates of each of the conditions involved in the comorbidity. In addition, the Berkson effect and a variety of referral biases make the use of clinic data hazardous and potentially misleading.

Until relatively recently, comorbidity has received little attention in the research literature and considerations of psychiatric classification issues have generally ignored the extreme frequency with which psychiatric disorders seem to co-occur. This neglect has had two unfortunate consequences. First, the findings of many studies of one specified psychiatric condition are likely to be misleading because the correlates of the disorder being investigated may represent the correlates of some unspecified comorbid condition. Second, there is the unsafe assumption that the meaning of any given disorder is exactly the same regardless of the presence or absence of other disorders. It is sometimes claimed that the DSM-III-R classification system avoids the difficulties associated with diagnostic hierarchies jut because it allows any level of comorbidity. However, it is evident that it includes more hierarchies than generally appreciated and that the encouragement of comorbid diagnoses introduces further problems.

In this paper we have outlined some of the detection artifacts that may produce a false picture of comorbidity and have gone on to discuss some of the nosological considerations that apply to comorbidity. These include: the concepts of disorders as categories or dimensions; overlapping diagnostic criteria; artificial subdivisions of syndromes; one disorder representing an early manifestation of the other; and one disorder being part of the other. Finally, we have considered some of the possible explanations of true comorbidity in terms of: shared risk factors; overlapping risk factors; comorbid patterns constituting a meaningful syndrome; and one disorder creating an increased risk for the other. In each case, we have put forward some general guidelines for testing alternative hypotheses. It is evident that it may be quite difficult

to differentiate between competing explanations but equally it is clear that an improved understanding of the varied mechanisms underlying comorbidity should shed important light on the processes involved in the genesis and contribution of psychiatric disorders.

Acknowledgement—Dr Caron was supported by Fellowship No. 891069 from Fonds de la Recherche en Santé du Québec.

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