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2. G. T. Allison, *Amer. Polit. Sci. Rev.* **63**, 689 (1969).
3. The relevance of mathematical modeling to "real-world" decision-making is often tenuous. No attempt will be made in this article to discuss the relevance or implications of the methodology used herein.
4. Senate Committee on Appropriations and Senate Committee on Armed Service, *Department of Defense Appropriations* (Government Printing Office, Washington, D.C., 1965), part 1.
5. The term "deterrence" has broad ramifications. This article deals with a restricted usage of the term, namely, for policies of "assured destruction" or "warfighting," or both. Other conceivable usages of deterrence are not discussed in this article, for example, for a first-strike capability or an extensive civil defense posture.
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12. It is well known that the optimum attack deployment is to target all missile sites as uniformly as possible [for example, see Afheldt and Sonntag (*11*)].
13. Reliability and other statistical effects can be handled by appropriate adjustment of the parameters. For example, see (*7*) for a discussion of the increased force requirements to maintain mutual deterrence at any confidence level.
14. It suffices to choose α such that

$$\alpha \gtrsim \max_i \left\{ \gamma_i \left(\bar{M}_i \Pi_j (1 - p_{ij})^{[\mu_j \bar{M}_j / \bar{M}_i]} \cdot (1 - p_{ij} \langle \mu_j \bar{M}_j / \bar{M}_i \rangle) \right)^{-1} \right\}$$

15. In the case of two equal parties maintaining mutual deterrence with the introduction of a weaker third party not attempting to maintain such a relationship, the number of additional missiles required by each of the major powers is proportional to the third power's forces.
16. The effects of countermeasures to ABM, such as radar attacks or decoys, should be treated by game-theoretic methods. However, these effects can be reflected by appropriate adjustment of the intercept probability q. Costs β and σ are overall program costs (in current dollars) of the total weapons systems, if coincident program time-frames for both systems and sizable procurements are assumed.
17. These results are reinforced by the effects of ABM countermeasures.
18. A complete analysis of the triad of forces—missiles, bombers, and submarines—leading to the optimal mix involves nonlinear mixed-integer optimization problems. The solution of these problems can involve detailed simulations and were not treated.
19. The views expressed in this article are the authors' and do not necessarily reflect the views of the United States government. We are grateful to Drs. Lee R. Abramson and Felix E. Ginsberg and Col. Alfred C. Herrera for their critical reading of this article. We also thank George A. Lincoln for his critical review and thoughtful observations.

Population Density and Pathology: What Are the Relations for Man?

Evidence from one city suggests that high population density may be linked to "pathological" behavior.

Omer R. Galle, Walter R. Gove, and J. Miller McPherson

Studies of various animal populations suggest that high levels of population density frequently produce "pathological" behavior. The results of these studies, coupled with an increased concern about high rates of growth in the human population, have led to speculations about the implications of high levels of density for human populations. We begin this article with a review of some of these studies, noting the implications of possible animal-human similarities, and then take the animal studies as a serious model for human populations and devise a test case.

In 1962, Calhoun published an article detailing the ways in which overcrowd-

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ing affects the behavior of rats. In his experiment, he gave the rats sufficient food and water, but the density of the population was substantially higher than it is in the rats' natural habitat. Calhoun observed the following "pathological behaviors" under these conditions: increased mortality, especially among the very young; lowered fertility rates; neglect of the young by their mothers; overly aggressive and conflict-oriented behavior; almost total withdrawal from the community (the "somnambulists"); and sexual aberrations and other "psychotic" behavior (*1*). It should be noted that these aberrations were much more common in the central pens, where the rats *voluntarily* congregated.

In recent years it has become clear that rats are not alone in being adverse-

ly affected by high density (*2, 3*). A study by Susiyama (*4*) of wild monkeys indicated that high density led to a general breakdown in the monkeys' social order and resulted in extremely aggressive behavior, hypersexuality, the killing of young, and so on. High density appears to cause death in hares (*5*) and shrews (*6*). Morris (*7*) has found that high density causes homosexuality in fish. Probably the most frequently demonstrated effect of density is in the area of natality. For example, under conditions of high density the clutch size of the great tit decreases (*8*), as does the number of young carried by shrews (*6*). It appears likely that high density reduces the fertility of elephants (*9*). Female house mice abort if they smell a strange male mouse (*10*), as do shrews (*11*).

In sum, high population density appears to have a serious inhibiting effect on many animals. It must be noted, however, that the effect of density is not uniform among different species; different species react to density in different ways. It is probably inevitable that increasing knowledge of the effect of density on animal behavior leads to concern about the effect density may have on human behavior. By now, the idea that density has, or at least may have, serious consequences for man appears to have fairly wide acceptance. Such acceptance is obvious in much popular writing (*12*) as well as in work specifically aimed at behavioral scientists (*13*).

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Density and Pathology in Human Populations

Although many people have written about the effect overcrowding has on human behavior, there is a paucity of good research. A detailed and careful review of the existing literature by Schorr led him to believe that the effect of poor housing (overcrowding) has been understated. Schorr concluded that poor housing (overcrowding) had the following effects (*14*, pp. 31–32).

A perception of one's self that leads to pessimism and passivity, stress to which the individual cannot adapt, poor health, and a state of dissatisfaction; pleasure in company but not in solitude, cynicism about people and organizations, a high degree of sexual stimulation without legitimate outlet, and difficulty in household management and child rearing . . . .

Other authors interpret the existing data differently and feel that such relations have not, in general, been clearly established (*15*).

The evidence on the relations of pathological behavior and high population density is ambiguous; before the issue is decided, a number of studies of different populations in different settings will have to be undertaken. If, as Hall (*3*) has suggested, different cultures and different ethnic groups have different spatial requirements, the issue becomes quite complex. A recent and important interview study in Hong Kong suggested that within that culture and in that setting, where virtually everyone lives in an overcrowded environment, variations in crowding are not related to severe emotional strain, but are related to a lack of control over children (*16*).

We will look at the relation between population density and a variety of pathological behaviors as they vary over the community areas of Chicago (*17*). Even if we use the animal studies as a guide, it is not obvious what effects we should look for in humans because, as noted before, density appears to affect different species in different ways. Our analysis will thus, of necessity, be exploratory. Since Calhoun's study has received more attention than others, we use his results as a starting point. There are several practical reasons for doing so. First, he covers a wider range of "pathologies" than do most other researchers. Second, there are a number of indices in the Chicago data that will serve as surrogate measures of Calhoun's "pathologies." In particular, there are indices of (i) fertility, (ii) mortality, (iii) ineffectual care of the young, (iv) asocial, aggressive behavior, and (v) psychiatric disorder. The following are operational definitions of the measures that we use in the statistical analysis.

For each of the 75 community areas of Chicago, the *Local Community Fact Book for Chicago* (*18*) provides information on the number of persons residing in that area. This, combined with the size of the land area included in each community area (*19*), gives a measure of population density—the number of persons per acre.

The first two measures we use for indices of "social" pathology are distinctly biological in nature—mortality and fertility. The immediate cause of mortality will generally be specific diseases, although mortality rates will also be affected by such variables as malnutrition, accidents, and suicide. Variations in fertility are due to differences in conception, gestation, parturition, and the factors involved in these processes. However, as Calhoun noted, the factors involved in determining variations in mortality and fertility are largely social in nature. Thus, although mortality is largely the consequence of disease, we are interested in variations in mortality as social phenomena because such variations appear to be indirectly caused by, and certainly are associated with, such variables as social class, ethnicity, and, possibly, population density. The same may be said for the factors involved in the determination of variations in fertility. Let us define, then, the first measure of social pathology as the "standardized mortality ratio." This measure is the age-adjusted death rate of a given community area, expressed as a ratio to the death rate for the total population of Chicago in 1960. Our second measure of social pathology will be the "general fertility rate," which is simply the number of births in a community area per 1000 women ages 15 to 44 in the same area.

As a measure of ineffectual parental care of the young, we will use the number of recipients of public assistance under 18 years old in May 1962 per 100 persons under 18 years old in April 1960. Although this is not an ideal measure of ineffectual parental care, families receiving such assistance are typically disrupted, having only one parent in residence, and the family is not providing for the children in the normal societal manner. We shall call this the "public assistance rate," but it should be remembered that the rate refers only to the *young* persons of the community area. Our measure of asocial, aggressive behavior will be the "number of male individuals brought before the Family Court of Cook County on delinquency petitions during the years 1958–61 per 100 male population 12–16 years of age in 1960" (*18*). We refer to the measure simply as the "juvenile delinquency rate." Finally, as an indication of withdrawal and other psychotic behavior, the fact book reports age-adjusted rates of admissions

Table 1. Zero-order, multiple, and partial correlation coefficients for social pathology, population density, ethnicity, and social class (Chicago, 1960).

Parameter	Social pathologies				
	Standard mortality ratio	General fertility rate	Public assistance rate	Juvenile delinquency rate	Admissions to mental hospitals
Population density and social pathology					
Zero-order correlation coefficient of each pathology with population density*	0.283	0.373	0.337	0.492	0.349
Partial correlation coefficient of each pathology with population density, controlling for social class and ethnicity	−0.177†	−0.023†	−0.118†	0.227†	0.142†
Social class, ethnicity, and social pathology					
Multiple correlation coefficient of each pathology with social class and ethnicity	0.828	0.853	0.885	0.927	0.546
Multiple-partial correlation coefficient of each pathology with ethnicity and social class, controlling for population density	0.817	0.827	0.871	0.907	0.466

* The measure of density, persons per acre is transformed into natural logarithms. † Not significantly different from zero at *P* = .05.

to mental hospitals for 1960–1961 per 100,000 persons in the community area in 1960. This we shall call the rate of "admission to mental hospitals" (20).

Variations in the five social pathologies we have just defined are normally explained by social structure factors, such as social class and ethnic (or racial) status. For example, it is assumed that variations in the mortality rate arise from such factors as exposure to disease, access to medical assistance, and knowledge about effective preventive measures and that such factors are mediated by one's social class and ethnic status. Similar arguments are made regarding the other pathologies. The precise explanations of the way in which class and ethnicity relate to each pathology would probably differ—in fact, there may be more than one explanation of how class and ethnicity relate to a particular pathology. Nevertheless, most sociologists see these social structure variables as the primary factors determining the variations in the rates of these pathologies. The case for the population density argument will be substantially strengthened if we can demonstrate not only that variations in population density make a significant contribution to the amount of variance explained in selected social pathologies, but that this contribution remains significant even after taking into account (or controlling for) the traditional sociological variables, social class and ethnic status.

We have chosen three measures as indicators of social class: the percentage of employed males in the community area who have white-collar occupations; the median number of years of school completed by all persons 25 years of age and older in a community area; and the median family income for all families residing in that community area. We have combined these measures into an index of social class (21). This index was developed in a blatantly post hoc fashion in which we maximized the degree to which class is associated with variations in the different pathologies. Our index of ethnicity is also based on three measures: the percentage of Negroes in the community area, the percentage of Puerto Ricans in the community area, and the percentage of foreign-born in the community area. Again, this index was developed in a post hoc fashion, in which we maximized the degree to which ethnicity is associated with variations in the different pathologies (22).

Preliminary Results

Table 1 exhibits, for each of the measures of social pathology, four different correlation coefficients. The relation between population density and social pathology is given, as is the more traditional problem of the relation between social structure and social pathology.

The causal model implicit in an argument like Calhoun's is simply

$$\text{Density} \xrightarrow{\quad u \quad} \text{Pathology}$$
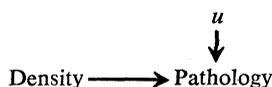
(The u in this model indicates unmeasured variables not taken into account that impinge on pathological behavior.) For this model, a relevant measure is the set of zero-order correlations between density and each of the five pathologies (23). These are presented in Table 1. For each social pathology, the relation with density is significantly different from zero, but it is relatively small. Furthermore, one of the five coefficients, though significant, is in the wrong direction. That is, the animal studies consistently indicate that the higher the population density, the lower the level of fertility. Here, the relationship is positive: the higher the density, the higher the fertility. However, some

Density as a spurious relation

Density as an intervening variable

Fig. 1. Models of density as a spurious relation and as an intervening variable; u_a and u_b represent all the unmeasured variables impinging on density and pathology that are not taken into account in the models.

investigators might argue that high rates of fertility are pathological for urban populations (24). Thus, one might conclude that population density has a small but significant effect on social pathology: the higher the density, the higher the pathology.

We know, however, that the lower one's social class and ethnic status, the more likely one is to live in areas with a high population density. Thus, it may be that class and ethnicity account for the variations both in population density and in pathology, and that there is no *causal* relation between density and pathology. Alternatively, class and ethnicity may affect density, and density may, in turn, affect the pathologies. In this case, density partially "interprets" the way in which class and ethnicity relate to the pathologies. We assume that, in this latter instance, class and ethnicity also affect the pathologies in ways unrelated to density. These two possibilities are presented in Fig. 1.

If the relation between density and pathology is spurious, then when we control for class and ethnicity, the partial correlation between density and pathology should approach zero. In contrast, if density is an intervening variable that only partially mediates the effects of class and ethnicity, the partial correlation between density and pathology will not go to zero when class and ethnicity are used as controls, although it may be reduced. Furthermore, if density is a major intervening variable, the partial correlation between the social structure variables and the pathologies would be noticeably reduced when density is used as a control.

As is apparent from Table 1, when class and ethnicity are used as controls, the correlations between density and the pathologies are not significantly different from zero. Furthermore, Table 1 shows that controlling for density has virtually no effect on the correlation between the social structure variables and the pathologies. One may assert that these data indicate that the relation between density and the pathologies is spurious (25). These results are similar to those of Winsborough, who used 1950 data for Chicago (26).
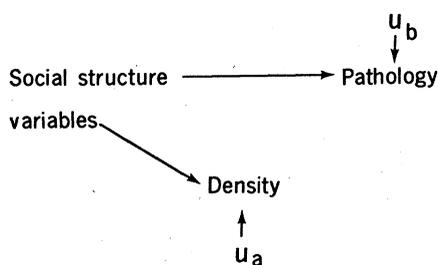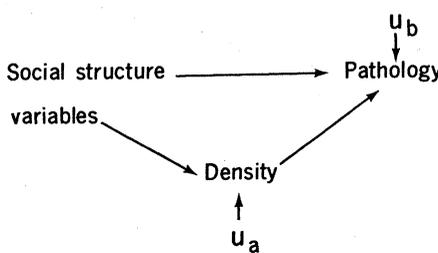
Dimensions of Population Density

However, before we accept such a conclusion, a reappraisal of our measure of density (persons per acre) may be in order. When the animal ecologists

Table 2. The interrelations among the components of population density (Chicago, 1960). (All measures of density are transformed into natural logarithms. For this reason, the multiple regression analysis of the four components of density on persons per acre yields a multiple r of 1.00, and the unstandardized regression coefficients are also 1.00.)

Measures of the interrelations	Components of population density			
	Persons per room	Rooms per housing unit	Housing units per structure	Structures per acre
Zero-order correlations with persons per acre	0.146	−0.560	0.741	0.717
Standardized regression coefficients from a multiple regression analysis of the four components of population density on persons per acre	0.226	0.242	0.811	0.699

refer to overpopulation of a particular species, they generally indicate the number of animals per some unit of area, such as an acre. However, in the case of human populations, the situation is substantially more complex, especially in an urban setting. On the one hand, there is what might be called overcrowding at the personal, or individual, level. That is, is it possible for an individual to have privacy in the particular housing unit in which he resides, or is he constantly in contact with others? We refer to this type of overcrowding as "interpersonal press." As we have developed the concept, interpersonal press is composed of two distinct factors: the number of persons per room and the number of rooms per housing unit (27).

Population density may also be affected by more "structural" factors. In the urban setting there is considerable

variation in the kinds of structures persons live in and in the spacing of these structures. If each individual housing unit is a single, detached structure, then there must be many individual structures per acre to achieve a high level of population density. Alternatively, if there are many high-rise apartment buildings in the area, then the number of housing units per structure will increase dramatically, while another measure, the number of residential structures per acre, may stay relatively low.

A given level of population density in a community area can be achieved by different combinations of four components of density: (i) the number of persons per room; (ii) the number of rooms per housing unit; (iii) the number of housing units per structure; and (iv) the number of residential structures per acre.

Table 2 shows the interrelations of

the various components of population density for Chicago (28). The first row shows the zero-order correlations between the overall measure of population density (persons per acre) and each of the four components of this overall level. The next row shows the results of a multiple regression analysis of each of the four components of population density on the general measure of population density (persons per acre). Both rows indicate that it is the structural measures of density (housing units per structure and structures per acre) which account for most of the variance in persons per acre, while the measures of interpersonal press (persons per room and rooms per housing unit) have only a modest relation to persons per acre.

These data thus suggest that the preceding analysis of the relation between density and pathology may have yielded misleading conclusions. This is particularly obvious if the effect of density on pathology is primarily a consequence of interpersonal press. Therefore, we reanalyzed the relation between density and pathology by breaking down population density into its four component parts.

We are still essentially testing the two models outlined in Fig. 1, with the one difference that, as density has been broken down into four components, our measure of density is now represented by multiple components; the relation between density and each pathology will therefore be represented by a multiple correlation coefficient. As before, if the relation between the components of density and the pathologies is spurious, the multiple-partial correlation between density and the pathologies should approach zero when we control for class and ethnicity; if density is an intervening variable, the multiple-partial correlation should not go to zero, although it may be reduced (29). The importance of density as an intervening variable should be directly related to the reduction of the multiple-partial correlation between the social structure variables and the pathologies when density is used as a control.

As is shown in Table 3, the results of the analysis when population density is broken down into its four components are strikingly different from the results of the original analysis shown in Table 1. Density is now related to each of the pathologies, and in each case a significant relation between the components of density and the pathologies

Table 3. Social pathology, density, ethnicity, and social class reexamined.

Parameter	Social pathologies				
	Standard mortality ratio	General fertility rate	Public assistance rate	Juvenile delinquency rate	Admissions to mental hospitals
Population densities and social pathology					
Multiple correlation coefficients of the four components of density* on each of the social pathologies	0.867	0.856	0.887	0.917	0.689
Multiple-partial correlation coefficient of each pathology with the four components of density, controlling for ethnicity and social class	0.476	0.371	0.584	0.498	0.508
Social class, ethnicity, and social pathology					
Multiple correlation coefficient of each pathology with social class and ethnicity	0.828	0.853	0.885	0.927	0.546
Multiple-parital correlation coefficient of each pathology with ethnicity and social class, controlling for the four components of population density*	0.143†	0.351	0.574	0.574	0.086†

* All measures of density are transformed into natural logarithms.   † Not significantly different from zero at $P = .05$.

Table 4. The proportion of variance explained by the four components of density and by class and ethnicity.

Manner of partitioning the explained variance between the major variables	Social pathologies				
	Standard mortality ratio	General fertility rate	Public assistance rate	Juvenile delinquency rate	Admissions to mental hospitals
Working backward from effect to cause					
Total "effect" of the four components of density	75.2	73.3	78.7	84.1	47.5
Increment added by class and ethnicity	0.4	3.2	7.0	5.3	0.4
Total variance explained	75.6	76.5	85.7	89.4	47.9
Working forward from prior cause to effect					
Total "effect" of class and ethnicity	68.5	72.8	78.3	85.9	29.8
Increment added by the components of density	7.1	3.7	7.4	3.5	18.1
Total variance explained	75.6	76.5	85.7	89.4	47.9

remains when class and ethnicity are used as controls. Furthermore, the relation between the social structure variables and the pathologies is markedly reduced when the components of density are used as a control. From this revised analysis it appears that at least some of the components intervene between class and ethnicity and the various pathologies, thereby partially interpreting that relationship. We will assume that this is correct, although we emphasize that we have not proved it. For example, we are simply assuming that class and ethnicity "cause" density and thereby ignore the possibility that density (through selective migration) "causes" class and ethnicity.

With the posited model in mind, let us attempt to evaluate the contributions made by class, ethnicity, and the four components of density. Following Duncan (30), we can do this in two different ways. First, we can work back from effect to cause. In this case, the multiple correlation between the components of density and pathology represents the total "effect" of density, including both its "unique" contribution to the variance of the pathology in question and the contribution it "transmits" from the social structure variables (class and ethnicity). The increment added by class and ethnicity that is not "routed" through density can be calculated by

subtracting the variance explained by density from the variance explained by density, ethnicity, and class. Alternatively, we can go from earliest cause to effect. In this case, the multiple correlation of ethnicity and class with the pathologies represents the total effect of these social structure variables, including the effect routed through density. We can then calculate the independent effect of density (the effect that is unrelated to ethnicity and class) by subtracting the variance explained by ethnicity and class from the variance explained by density, ethnicity, and class.

The results of these analyses are presented in Table 4. If we work back from effect to cause, density appears to "account" for most of the variance, with the social structure variables having relatively little effect on the pathologies except through their effect on the components of density. On the other hand, if we go from earliest cause to effect, we see that class and ethnicity do, at least indirectly, account for most of the variance of the pathologies. It is noteworthy that in most cases the independent increment of explained variance added by either the social structure variables or by the components of density is fairly small. These findings are consistent with the second model proposed in Fig. 1; that is, the results are compatible with the assumption that

the components of density interpret the relation between the social structure variables and the pathologies.

As a step toward identifying the relative importance of each of the four components of population density, a multiple regression analysis was run for each of the five social pathologies. In four of the five cases, the standardized regression coefficients indicated that the number of persons per room is the most important determinant of the effect of density on pathology. The exception is admissions to mental hospitals, in which case the most important component of density is the other measure of interpersonal press—rooms per housing unit. Next, we found that in four of the five cases the second most important component is housing units per structure. When an analysis such as that outlined in Table 4 is performed on a comparison between the effect of persons per room and rooms per housing unit when class and ethnicity are taken into account, the results are strikingly similar. That is, the values differ only slightly from those in Table 4, in which all four components of density are considered.

Table 5 presents a similar analysis, but with only one component of density considered—persons per room. Because we already suspected that persons per room is not strongly related to

Table 5. The proportion of variance explained by persons per room and by class and ethnicity.

Manner of partitioning the explained variance between the major variables	Social pathologies				
	Standard mortality ratio	General fertility rate	Public assistance rate	Juvenile delinquency rate	Admissions to mental hospitals*
Working forward from prior cause to effect					
Total "effect" of persons per room	60.5	65.4	73.3	61.5	15.8(46.8)
Increment added by class and ethnicity	9.8	9.5	10.1	24.4	15.6(0.2)
Total variance explained	70.3	74.9	83.4	85.9	31.4(47.0)
Working forward from prior cause to effect					
Total "effect" of class and ethnicity	68.5	72.8	78.3	85.9	29.8(29.8)
Increment added by persons per room	1.8	2.1	5.1	0.0	1.6(17.2)
Total variance explained	70.3	74.9	83.4	85.9	31.4(47.0)

* The numbers in parentheses indicate the values that occur when rooms per housing unit are used instead of persons per room.

admission to mental hospitals, we first focused our attention on the other four pathologies. For these pathologies, the total amount of explained variance dropped relatively slightly. As we move from effect to cause, we find that persons per room accounts for most of the explained variance, although the relation is not as strong as when we used all four components of density. However, compared to our earlier analysis, there is a noticeable increase in the independent increment added by class and ethnicity. Most of this increase can be attributed to the fact that housing units per structure are no longer treated as part of density.

This analysis suggests that, for mortality, fertility, public assistance, and juvenile delinquency, the most important component of density is persons per room. Next, but considerably less important, is the number of housing units per structure. For these four pathologies, the other two components of density—rooms per housing unit and structures per acre—appear to be relatively unimportant.

The pattern is quite different for admissions to mental hospitals. When Table 5 is compared with Table 4, one can easily see the marked decline in the total amount of variance explained, when the only component of density considered is persons per room. This is not surprising, since the standardized regression coefficients indicate that rooms per housing unit is the most important component of density as a predictor of admissions to mental hospitals. In Table 5 we have put in parentheses the variance associated with rooms per housing unit. In comparing these with those obtained when the four components of density are used, it is apparent that rooms per housing unit can account for virtually all of the variance in hospital admissions associated with density.

If our assumptions are correct, these data indicate that density—particularly persons per room (except in the case of admissions to mental hospitals)—may be an important factor in the development of various pathologies.

How Density May Relate to Pathology

Before considering each pathology separately, let us make some general observations. First, as the number of persons in a dwelling increases, so will the number of social obligations, as well as the need to inhibit individual desires. This escalation of both social demands and the need to inhibit desires would become particularly problematic when people are crowded together in a dwelling with a high ratio of persons per room. Second, crowding will bring with it a marked increase in stimuli that are difficult to ignore. Third, if human beings, like many animals, have a need for territory or privacy, then overcrowding may, in fact, conflict with a basic (biological?) characteristic of man (31).

It would seem reasonable to expect that people would react to the incessant demands, stimulation, and lack of privacy resulting from overcrowding with irritability, weariness, and withdrawal. Furthermore, people are likely to be so completely involved in reacting to their environment that it becomes extremely difficult for them to step back, look at themselves, and plan ahead (32). It would certainly seem that in an overcrowded situation it would be difficult for them to follow through on their plans. Thus, we might expect the behavior of human beings in an overcrowded environment to be primarily a response to their immediate situation and to reflect relatively little regard for the long-range consequences of their acts.

It seems from the above discussion that the most important component of density, as far as the pathologies are concerned, would be persons per room. This, of course, is the component that our analysis has indicated to be most important. Furthermore, it would seem that, to the degree persons in different dwelling units are involved with each other because of spatial arrangements (that is, could hear arguments, television, and so on), many of the reactions that occur on the interpersonal level (such as irritation and withdrawal) might also occur at this interunit level of interaction. Probably the most significant indicator of overcrowding at the interunit level of interaction is housing units per structure—and this, in our analysis, was the second most important component of density.

We now turn to a brief discussion of the possible effect of density (overcrowding) on each of the five pathologies under consideration.

Mortality. There are at least four possible ways in which overcrowding may be related to mortality. First, increased contact with others increases one's chances of contracting various infectious diseases. Such contact would presumably be related to both the number of persons per room and the number of housing units per structure. Second, if persons do become tired and run-down because of overcrowding (33), overcrowding would increase their susceptibility to disease. Third, sick persons in an overcrowded situation are likely to be constantly disturbed by the activity of others and thus will often not get the rest and relaxation that is important to treatment. And fourth, if overcrowding is associated with irritability, withdrawal, and ineffectual behavior, the treatment the sick person receives (from family members) will not be as effective in an overcrowded situation. Regarding the above points, we would note that investigations of overcrowding do indicate that it is related to poor health (34) and that controlled studies confirm that improved housing reduces the incidence of illness and death (35).

Fertility. Animal studies indicate that overcrowding leads to a drop in natality. However, we found the exact opposite —namely, the greater the density, the greater the fertility. If we are to consider the animal studies as being relevant to human beings, we must reconcile this difference. We reiterate that, although density has a significant impact on many animals, both the effects of density and the mechanisms involved differ widely from species to species. Second, we note that a frequent effect of overcrowding among animals is the development of hypersexuality (1, 4). Among human beings, an increase in sexual intercourse is likely to lead to increased natality, for women are receptive and able to conceive for 12 months of the year. In contrast, most female animals are receptive and able to conceive during a very specific and limited period of time, and at this time they typically have sexual intercourse. Therefore there is no reason to believe that increased rates of sexual intercourse among animals would typically lead to increased natality, whereas it would among human beings. Third, we note that many factors that would appear to limit natality in animals, such as lack of territory (36) or intense social competition (37), do not appear to be major factors in human populations. Fourth, because overcrowding appears to make it difficult to step back, look at one's situation, and plan ahead, it may be that persons in overcrowded situations are less likely to perceive the

long-range consequences of having more children and are thus less likely to want to use birth-control techniques. And finally, because overcrowding makes it difficult to follow through on plans, birth control, even if desired, may be ineffectually practiced.

Ineffectual parental care (public assistance). Overcrowding may lead to tensions and irritations in the home. Potentially, this could cause the break-up of the family, which might also mean the loss of financial support. Even if the family does not break up, children may receive less effective care in the home because overcrowding leads to ineffectual performance and withdrawal on the part of the parents. Furthermore, in overcrowded situations parents may be less likely to support their children in the usual manner, through gainful employment, because of weariness, poor health, and ineffectual ways of behaving that affect their performance in the larger community.

Juvenile delinquency. As noted above, in an overcrowded environment parents are likely to be irritable, weary, harassed, inefficient. Children, in turn, are apt to find the home a relatively unattractive place, full of constant noise and irritation, with no privacy, no place to study, and so on. They are thus inclined to seek relief by getting out of the home. In fact, their disappearance may be partially welcomed by the parents, for it removes, temporarily, a source of irritation. Studies of low-income (overcrowded) families indicate, as our analysis would suggest, a strikingly early cutoff point in parental will and ability to contain children (*38*).

An important factor in the development of delinquent gangs appears to be a high degree of autonomy. We have suggested that such autonomy is probably greater in dwellings with a high persons-per-room ratio. It may also be that autonomy is greater where there are a large number of housing units per structure, which, as we have already argued, may lead to a decrease in communication between persons in different dwelling units. At any rate, the Chicago data indicate that housing units per structure has more "impact" on delinquency than it does on the other pathologies.

Psychiatric disorder. From the above discussion, it would seem reasonable to anticipate a fairly strong relation between persons per room and admissions to mental hospitals. However, persons per room has a much weaker rela-

tion to admissions to mental hospitals than it does upon the other pathologies. In fact, the density component with by far the strongest relation to admissions to mental hospitals is rooms per housing unit, a finding that does not fit readily into our framework.

Admissions to mental hospitals is highly correlated with the percentage of persons living alone ($r = .72$) (*39*). It may be that isolation is a contributing factor in the development of mental illness (that is, too little interaction instead of too much). Furthermore, disturbed persons living by themselves are more likely to require hospitalization when they can no longer care for themselves than are persons living with and assisted by others. We suspect, however, that the correlation between rooms per housing unit (or persons living alone) involves primarily a self-selection factor. That is, people who have a history of difficulty in getting along with others are likely to move to small apartments where they live by themselves, and these are the persons who are most likely to be admitted to mental hospitals. If this is the case, then it is the kind of housing that has drawn disturbed persons into particular community areas. This would involve a process that falls completely outside the posited model. It may, of course, be that overcrowding played a role in the creation of the person's initial disorders, which in turn led to his living alone, but these data, while not denying that possibility, do not support it.

Conclusion

Our study suggests that overcrowding may have a serious impact on human behavior and that social scientists should consider overcrowding when attempting to explain a wide range of pathological behaviors. Having made this point, we end on a note of caution. We have been using cross-sectional ecological data. Thus, not only have we not proved that there is a causal relation between density and the various pathologies, but the relations that appear at the ecological level may not appear at the individual level. We would also note that, although social structure variables and density are analytically very distinct, they are so highly intercorrelated, at least for these data, that it is difficult to accurately identify their independent effects. Even assuming that the data on Chicago do reflect the im-

portance of density, more research is needed. At the moment, we may speculate about how overcrowding relates to various pathologies, but specific knowledge about causal links, if there are any, is lacking.

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17. These are ecological data, and relationships that occur at this level of analysis do not necessarily occur at the individual level. However, it seems to us that ecological measures are appropriate and meaningful when dealing with phenomena such as density. That is, characteristics of areal units may have a significant effect on rates of human behavior.
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20. As noted, there are 75 community areas in Chicago. However, the central business district (community area 32—known as the Loop) is a unique area with regard to various social, economic, and other kinds of indicators. In our case, the measures of pathology are dramatically changed if the central business district is included. Perhaps the most marked case is the rate of admissions to mental hospitals. The city-wide rate is 297.6; the rate for the Loop is 3757.2, and the next highest rate is 851.1. While the elimination of the Loop does not transform the distribution of admissions to mental hospitals into a normal distribution, it does substantially reduce its deviation from this ideal: skewness is reduced from 7.56 to 2.88, and kurtosis is reduced from 62.32 to 15.14 [for a discussion of skewness and kurtosis, see J. Freund, *Modern Elementary Statistics* (Prentice-Hall, Englewood Cliffs, N.J., 1960), pp. 99–105]. Other measures, especially the standardized mortality ratio, are affected in similar, although somewhat less drastic, fashion. For this reason our analysis is based on 74 rather than 75 community areas in Chicago around 1960.
21. A regression analysis of income, education, and occupation was run on each of the pathologies. These five regression equations wer° then used as a basis for constructing the weighted sum of the three measures as a general index. The equation for the index of social class is as follows: index of social class = 0.1 × (median family income) + 10.0 × (median years of school completed) + (percentage of employed males in white-collar occupations) − 550.0. Median family income is by far the most important component of the social class index.
22. A regression analysis of percentage of Negroes, percentage of Puerto Ricans, and percentage of foreign-born was run on each of the pathologies. As with social class, these five regression equations were then used as a basis for constructing the weighted sum of the three measures as a general index. The equation for the index of ethnicity is as follows: index of enthnicity = 25.0 × (percentage of Negroes) + 10 × (percentage of Puerto Ricans) + 0.1 × (percentage of foreign-born). The percentage of Negroes is by far the most important component of the ethnicity index.
23. As Blalock notes, grouping by proximity may partially control for independent variables associated with "error" in the dependent variable. Thus to some extent, the size of the correlation between density and the pathologies, and between the social structure variables and the pathologies may be determined by the fact that the community areas, like all ecological variables, involved data grouped by proximity [H. Blalock, *Casual Inferences in Nonexperimental Research* (Univ. of North Carolina Press, Chapel Hill, 1964), pp. 102–114.
24. In a subsequent section of this article we will discuss the possibility that in human populations high rates of fertility might be a consequence of population density.
25. The same conclusion is reached if one uses regression coefficients. We would note that there are advantages and disadvantages to using either regression coefficients or partial correlations. Although multiple partial correlations are not strict estimates of the parameters of the causal model, we consider them to be sufficient for our purpose, and using them simplifies the analysis in the second part of the article.
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27. Holding the number of persons per room constant, it is probable that an increase in the number of rooms will increase the likelihood that a person will be able, at least occasionally, to be alone in a room.
28. The number of persons in each community area is reported directly in the *Local Community Fact Book*, as is the number of housing units. The number of rooms per community area and the number of residential structures per community area are, however, based on estimates from open-ended interval data. The fact book reports the number of housing units with 1, 2, 3, 4, 5, 6, 7, and 8 or more rooms in them. To get an estimate of the number of rooms per community area, we multiplied the number of housing units at each level by the appropriate number of rooms. The highest interval was multiplied by 8, even though it was an open-ended interval. The fact book reports the number of housing units in 1-unit structures, 2-unit structures, 3- and 4-unit structures, 5- to 9-unit structures, and 10- or more unit structures. Data from the 1940 fact book suggest that, for that year, slightly over half of the housing units located in the over 10 category were in the over 20 category. To estimate the number of residential structures in the area, we set the midinterval points for these data at 1, 2, 3, 5, 7, and 20. We divided the number of housing units in each category by these midinterval points and added the resulting figures to get the estimate of the number of residential structures for the community area. The four measures of density were then calculated by division: number of persons divided by the number of rooms, the number of rooms divided by the number of housing units, and so on.
29. The cogency of the multiple-partial correlation coefficient as an estimate of the relation is based upon the assumption that all indicators are related to the pathologies in the predicted direction. This assumption is, in general, supported by an examination of a table of the partial regression coefficients relating the four dimensions of density to each of the pathologies, although the general fertility rate increases with density. This table is available from the authors upon request.
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39. The relation between the percentage of persons living alone and admissions to mental hospitals remains fairly strong, even after class and ethnicity are used as controls ($r = .59$). The percentage of persons living alone also has a high negative correlation with rooms per housing unit $r = -.91$).
40. The research for this paper was supported in part by the Urban and Regional Development Center, Vanderbilt University. We thank H. Costner, L. Rigsby, A. Gove, and O. D. Duncan for their comments on an earlier draft of this article.

NEWS AND COMMENT

National Environmental Policy Act: Signs of Backlash Are Evident

It is as much the duty of government to render prompt justice against itself, in favor of citizens, as it is to administer the same between individuals.—ABRAHAM LINCOLN

It's a rare occasion when Congress produces a piece of legislation that measures up to the lofty purpose of governmental self-control that Lincoln had in mind. Having done so, the chances are great that Congress will soon break out in a rash of second thoughts once the practical difficulties of enforcing self-control hit home.

This, at least, would seem to be the main lesson to be drawn from a bitter fight currently shaping up in Washington over the future of the 2-year-old National Environmental Policy Act (NEPA), a law that President Nixon symbolically chose to sign as his first official act of the new decade and one that his chief environmental adviser, Russell E. Train, has called "one of the most significant policy reforms in recent history."

History notwithstanding, the signs of a backlash against NEPA are becoming evident. A few high government officials like John A. Carver, Jr., a member of the Federal Power Commission (FPC), consider it a "paper monster . . . of great potential harm" and suggest that perhaps "Congress should take another look." And federal agencies, ranging from the Atomic Energy Commission (AEC) to the Department of Transportation, are pressing for new

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