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COMMENTARY

HERD IMMUNITY: BASIC CONCEPT AND RELEVANCE TO PUBLIC HEALTH IMMUNIZATION PRACTICES[1]

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Specific immunization has long been a basic tool in medical practice and in local, state, national and international public health. However, the protection afforded by such immunization has been largely limited to the persons immunized because: 1) the usual sources of infection are extra-human (tetanus, yellow fever); 2) spread is primarily by indirect means (typhoid); or 3) those immunized may remain at least partially susceptible to infection and, hence, continue as potential links in the future spread of agents transmitted by contact (diphtheria, pertussis, Salk-type inactivated polio vaccine). An important exception has been vaccination against smallpox which does induce solid though temporary resistance to infection. Presumably as the result of systematic vaccination, endemic smallpox has disappeared from large areas including the United States and major efforts are now in progress to eliminate the disease from developing countries as in West Africa (1).

The advent of live virus vaccines against poliomyelitis, measles and, most recently, rubella, has raised realistic hopes that, as with smallpox, these diseases also can be made to dissappear from large populations by means of large scale immunization programs. Whether in developing or developed countries, the planning of such programs, if they are to be of maximum effectiveness, requires full understanding of the principle of herd immunity and of all the factors which influence its operation.

That the factors influencing the operation of herd immunity are not fully understood by many people was well illustrated during the 1969 meetings of the American Public Health Association. A report on measles epidemics in West Africa (2), where over 90 per cent of the population is immune, including virtually all those over two years of age, was followed by a number of questions from the floor concerning the fact that epidemics continued to occur despite the large proportion of immunes. These questions stemmed from the currently prevailing concept that, as defined in a medical dictionary (3), herd

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immunity is "the resistance of a group to attack by a disease to which a large proportion of the members are immune, thus lessening the likelihood of a patient with a disease coming into contact with a susceptible individual."

This concept is directly applicable only to randomly mixing populations. However, truly random mixing can be assumed only for certain small closed populations and never occurs in open populations. In his treatise on the mathematical theory of epidemics, Bailey (4) states: "It is well known that epidemics in a large population can often be broken down into smaller epidemics occurring in separate regional subdivisions. These smaller epidemics are in general not in phase but interact with each other to some extent. We could hardly assume even a small town to be a single homogeneously mixing unit. Each individual is normally in close contact with only a small number of individuals, perhaps of the order of 10–50. The observed figures are therefore pooled data for several epidemics occurring simultaneously in small groups of associates. In reality such groups overlap and interact."

In his review article on epidemic theory (5), Serfling notes that as early as 1906 it was recognized that "the progress of an epidemic is regulated by the number of susceptibles and the rate of contact between infectious cases and susceptibles." While the literature on epidemic theory since 1906 contains excellent expositions of the concept of herd immunity, these are usually embedded in rather mathematical discussions of the validity of various epidemic models. Hence, it seems appropriate to offer a simple and minimally mathematical presentation which explicitly identifies and illustrates the factors which favor the spread of infection in populations containing many immunes and those which restrict such spread.

Crucial to our presentation is the recognition that open populations are made up of innumerable definable but often interlocking subgroups which differ in respect to proportions of immunes and intimacy of contact. In particular, we will try to show how variations of these population characteristics may affect "the rate of contact between infectious cases and susceptibles" and, hence, determine whether epidemic spread will occur.

Some basic assumptions

For the sake of simplicity, we will confine our discussion and illustrations to agents restricted to man and spread only by person-to-person contact. Also, we assume that the following postulates are generally applicable.

1) Individuals are either susceptible or fully immune, and immunity is durable.
2) The period of infectiousness is short, and is of approximately the same length for all who become infected.
3) There is no need to allow for "removals", that is for the withdrawal from circulation of infectives who become ill. (This may be because many infections are silent and those illnesses which do occur are mild, or because the period of greatest infectivity precedes the onset of illness as in measles.)

Since the purpose of an immunization program is to halt the spread of the agent, and our purpose here is to study herd immunity, we will confine ourselves entirely to infection and will not consider illness. The word "case" throughout will refer to a person who is infectious to others, or more simply, to an infective.

First we will consider the importance, or lack of importance, of the proportion immune in a randomly mixing population. We will, by considering this simplest of models, develop the tools which will enable us to tackle the more complex problems of the multiple and overlapping mixing groups which exist in all societies.

Randomly mixing populations—the Reed-Frost model

A randomly mixing population is one in which the probability of contact within any time interval is the same for every choice of two individuals in the population. Epidemic models based on random mixing have been used in connection with small closed populations such as those of orphanages, boarding schools or companies of military recruits (5–7).

One of the simplest models, and one with which most persons in public health work are acquainted, is that which Reed and Frost taught for many years at Johns Hopkins. For demonstration, they used a mechanical model in which random mixing was illustrated by stirring up a collection of colored balls. The Reed-Frost model involves consideration of discrete equal intervals of time (such as days or weeks) which, in our illustrations, we will take as equal to the length of the period of infectivity. Then, if a person becomes infected during the interval from t to $t + 1$ (the first week), he becomes infectious to others at $t + 1$ (the beginning of the second week), ceases to be infective and becomes permanently immune at $t + 2$ (the beginning of the third week).

The Reed-Frost model and its application in one extended example are described in some detail in the Appendix. Two definitions are particularly important to understanding this model: 1) *adequate contact* between two individuals is that sufficient to allow transmission of the infecting agent from an infectious person to a susceptible; and 2) the *contact rate*, p, is the probability that *any* two persons in the population will make adequate contact during any interval. This contact rate thus summarizes both the infectivity of the agent and the social habits of the population. It also is important to understand that the *expected number of contacts during any interval* for any member of the population is the product of the contact rate, p, and the number of potential contacts (the total population or N minus one) and that these contacts will be distributed between susceptibles, immunes and infective cases in the proportions in which each are present in the population during the specified time interval.

A simple, first example begins with a play group of 11 children of whom 10 are susceptibles and one is an infective and examines the consequences of increasing the size of the play group by adding five immunes. These are summarized in the following tabulation

Play group composition at beginning of the first interval				Contact rate	Expected number of contacts by case in first interval			Probability of no spread
Size	Cases	Susceptibles	Immunes		with susceptibles	with immunes	Total	
N	C	S	I	p	$m' = pS$	$w = pI$	$m = p(N-1)$	$(1-p)^S$
11	1	10	0	0.2	2	0	2	.107
16	1	10	5	0.2	2	1	3	.107
16	1	10	5	0.133	1.33	0.67	2	.239

It can be seen that, if the contact rate (p) is unchanged, addition of the immunes does not alter the probability of no spread. Although the expected total number of contacts by the case (m) is increased, the additional contacts are wasted on immunes. However, if m is held constant at 2 when the immunes are added, the contact rate decreases as does the expected number of contacts with susceptibles (m') and the probability of no spread increases.

A second example extends the Reed-Frost model to the introduction of a single infective case into larger populations ranging in size from 400 to 10,000 while one of the six pairs of specified characteristics are held constant and others are allowed to change. In practice the average number of contacts per person per interval (m) will be relatively constant as the total population size (N) increases. However, examples in which p is constant and m or $p(N-1)$ increases with N are included in example 2 for the sake of completeness and to stress the underlying principles. The results expected in each of these situations are shown in some detail in table 2 (Appendix) but our interest centers in the columns indicating number of susceptibles, average number of contacts with susceptibles, probability of no spread and epidemic size. The important relations shown can be summarized as follows:

1) The expected number of contacts by the case with susceptibles during the first interval, pS_0, completely determines the probability of no spread, $P(A)$;
   (a) if pS_0 is constant, so is $P(A)$ (sets 1 and 2),
   (b) if pS_0 increases, $P(A)$ declines (sets 3 and 4),
   (c) if pS_0 falls, $P(A)$ rises (sets 5 and 6).
2) Given constant pS_0 and $P(A)$, the median epidemic size increases with the number of susceptibles (set 2).
3) Population size, proportion immune and contact rate influence the probability of spread and median epidemic size *only* when, under the conditions postulated, their change necessarily causes changes in number of susceptibles (sets 2, 3 and 6) or in average number of contacts with susceptibles (sets 3, 4, 5 and 6).

In brief summary, application of the Reed-Frost model in the foregoing examples demonstrates that, over a wide range of variations, the number of susceptibles and the rate of contact between them determine epidemic potentials in randomly mixing populations. If these are held constant, changes in population size and, therefore, in the proportion immune do not influence the probability of spread.

Populations with complex mixing structure

As noted already, random mixing serves as an adequate approximation of contacts between individuals only in certain small closed populations. In this section we will consider the more complex mixing structures of real-life, free-living populations as typified by a large city.

In a typical city in the United States there may be a densely populated central area surrounded by various residential neighborhoods of differing population density and also differing in respect to economic, occupational and educational characteristics. Superimposed are school districts, the pediatrician's office, health department clinics, shopping areas, entertainment or recreational centers, transportation systems and occupational or business groups. Some of these may be heterogeneous with respect to cultural and socioeconomic factors and all may play a role in creating paths along which infection may spread from one otherwise isolated subgroup to another.

Important mixing groups

The *basic unit*, social and epidemiologic, is the *family or household*, within which the contact rate between members is high, whether they be of the same or quite different age groups. In some countries or cultures the effective household may be quite large, consisting of several related families living in one compound.

In general, the between-family contacts will be higher for households in the same *geographic neighborhood*. The role of neighborhoods in the spread of infection may be quite variable from one society to another. The habit common to American

children of running in and out of neighboring houses, having lunch or "sleeping over" with Johnny is obviously of real importance as a type of population mixing, as are the "coffee klatch" and neighborhood events such as picnics and Christmas parties. Also, common recreational interests, membership in the same religious group or social club and similarity of ages of children will result in the definition of *clusters of families* between which contact will be more intimate than that with other families in the same neighborhood.

The *schools* serve to bring together subgroups of the population defined by age, by broad area of residence and, in many cases, by socioeconomic status. They bring together children from different families and different immediate neighborhoods. Further, in rural and suburban areas, the school bus serves as a particularly effective exposure chamber for respiratory agent spread. Since school age children are characterized by high susceptibility rates for many infectious diseases, the schools provide potentially important paths of spread from one part of the population to another. Hence, whether or not the school is open may be of major importance in the occurrence of an epidemic (as in influenza).

The largest proportion of susceptibles is usually found among *preschool children.* The preschool child, according to the social habits of his society, will be exposed to the other members of his family, school age sibs and parents, and to the preschool children in other families through the naturally occurring neighborhood play groups. In addition, preschool children from birth on are apt to accompany their mothers to the market place (as in West Africa where the mothers carry their small children on their backs) and on other errands. In such societies, as is evident from the epidemic occurrence of measles in West Africa despite the high proportion of immunes, this practice alone may create high contact rates among large groups of susceptible infants and preschool children. In other societies the nursery school (or day care center), church school, or community playgrounds may serve this purpose.

The distribution of immunes

In general, the most important stratification of the community by immune status is that by age. The proportion immune increases with age and, for highly infectious agents, will approach unity at some point in life. In developed countries this may be in early adulthood, e.g. measles in the USA, while in developing countries this may be very early in childhood, e.g. measles in West Africa. However, within the population of a community, there may be pockets of susceptibles, either because prior epidemics have failed to spread into the group or because they have not accepted immunization. Current important examples in the USA relate to measles and poliomyelitis, both of which continue to occur in small outbreaks among unvaccinated groups characterized by low economic and educational status.

A community mixing model

In this example we will consider 100 susceptible children and one infected child whose opportunities for contact with each other depend on various types of social mixing groups. These may be considered as part of a much larger community of families. Although it is not necessary to specify the total community size or the proportion immune, important aspects of the distribution of these children are noted in the footnote to table 1.

The Reed-Frost model will apply here only as it pertains to within subgroup mixing, that is to a single family, a single nursery school, or a small neighborhood preschool play group. It will, however, provide a useful framework of terms and definitions.

Four situations will be considered. In the first there is no contact beyond the total community mixing at a very low level (a random mixing case); in the sec-

TABLE 1

*Distribution by size of epidemics among 100 susceptible children in a community of families, play groups and a nursery school**

Mixing groups	Within group contact rate	No. of epidemics with indicated Nos. of cases										Median epidemic size	Mean No. of cases	Maximum No. of cases
		1	2	3	4	5–9	10–19	20–29	30–39	40–59	60–79			
Total community	.002	82	15	2	1							1	1.2	4
Total community Families	.002 .500	22	18	34	8	17	1					3	3.3	16
Total community Families Play groups	.002 .500 .100	11	6	26	23	23	9	1	1			4	5.6	33
Total community Families Play groups Nursery school	.002 .500 .100 .100	23	4							28	45	58	45	73

* Observed distributions of epidemic size from computer simulation of an extension of the Reed-Frost model to allow for multiple mixing groups, based on 100 simulated epidemics per situation. The 100 susceptible children and the case were in 62 families containing 1 to 3 children (average 1.6) and in 24 playgroups containing up to 10 children (average 4.2). The case was in a 3-child family and a 5-child play group. Although the case did not attend nursery school, his two younger siblings did.

ond we allow also for high contact rates within the family or household; in the third we add small play groups in which children of neighboring families come in contact; and in the fourth we add a nursery school which is attended by 40 of the 100 susceptible children. The purpose is to show that, with a fixed number of susceptibles, as we allow more and more opportunity for contacts between susceptibles, the epidemic potential increases *whether or not* the proportion immune remains constant.

Table 1 presents the results in terms of the distribution by size of 100 computer-simulated epidemics per situation using a model (8) in which each separate group (family, play group, nursery school, total community) is randomly mixing but with differing contact rates prevailing in each. With contact depending entirely on total community mixing, 82 per cent of the trials resulted in no spread and the maximum epidemic size was four cases. When contacts within families were taken into account, only 22 per cent of the trials resulted in no spread but 74 per cent had three or fewer cases. With play group mixing added, 34 per cent of the epidemic trials resulted in more than five cases and in only 11 did spread fail to occur. Finally, when the nursery school of 40 children was opened, 73 per cent of the epidemics spread through more than one half of the susceptibles, the median epidemic size was 58, and the largest epidemic reached 73 of the 100 susceptibles. It also is of interest that the distribution of epidemic size has assumed the bimodality typical of contact rates high enough to permit large epidemics but not high enough to insure them. Here the introduction of a single case resulted in virtually no spread in 27 per cent of the trials, in large epidemics in the remaining 73 per cent and in no outbreaks of moderate size. The effective contact rate was high enough that, in every case in which three or more persons were infected, the epidemic caught fire and continued to more than 40 cases.

Discussion

The foregoing examples should have made it clear that for a given infectious agent, epidemic potential is determined by the number of susceptibles and the nature and frequency of their contacts with each other. If these characteristics of the population are held constant, other characteristics such as the size of the total population and the proportion immune have no influence on the epidemic potential. In this light, the question "what proportion of the population should be immunized to prevent an epidemic?", is not answerable in absolute terms. First, the question must be restated to allow for the element of chance, e.g. "What proportion of the population must be immunized to lower the probability of an epidemic of more than 10 cases below 5 per cent?" To reach even an approximate answer we must consider

1) The infectivity, method of spread and viability of the infectious agent.
2) The season, and what effect this has on population mixing (school open or closed) and the viability of the agent.
3) The number of susceptibles and their distribution by age, geographic area, economic status, etc.
4) The social habits of the society with respect to mixing groups which provide the type of contacts involved in the transfer of infection.
5) Some estimates of the subgroup contact rates among susceptibles.

Considerable information concerning items 1 and 2 ordinarily will exist and information concerning item 3 usually can be developed. Items 4 and 5 pose real difficulties. We can differentiate broadly between West Africa and the United States in terms of social habits, we can recognize important differences between urban and rural areas, and we can define populations linked by specific schools. Unfortunately, precise definition of the multitude of interlocking mixing groups in large populations and estimation of their respective contact rates are not possible to achieve. Nonetheless, recognition of the dominant influence of these factors on agent spread can help assure that an immunization program will be maximally effective. It is not enough to know how non-immunes are distributed in the total population by age alone. We also should know how they are distributed in population subgroups defined by such other attributes as place of residence, economic status, ethnic origin and religious affiliation.

This concept is hardly novel in that recurrent, usually small, outbreaks of such generally well controlled diseases as diphtheria, measles and poliomyelitis continue to occur in population subgroups characterized by their reluctance to accept immunization and which, typically, also can be defined in terms of race, low educational and economic level and even, as described in the following, religious preference. This latter is dramatically illustrated by a small outbreak of smallpox which lingered on for several months in the unusually well immunized community of Abakalike in Eastern Nigeria (9). Investigation revealed that it was confined entirely to members of a small religious sect who refused vaccination and who, despite dispersal throughout the community, maintained both close social ties within the group and relative segregation from the rest of the population. This experience, together with the theoretical considerations herein presented, suggest that, to reduce the probability of epidemic spread to the minimum, an immunization program should be preceded whenever possible by surveys to identify particularly susceptible population subgroups and be characterized by special efforts to reach groups so identified. In effect, this is a strategy to increase to the maximum the number of susceptibles immunized which, in view of the inescapable but important uncertainties described, must be the goal of every immunization program.

The foregoing considerations are most

relevant to programs of systematic immunization such as those directed against polio, measles and, more recently, rubella in the United States which have as their ultimate goal elimination of the causative agent from the country. A different strategy uses probability of exposure rather than of susceptibility to guide administration of vaccine. Thus, in countries previously freed of smallpox, reintroduction is followed by strenuous efforts to contain the disease, including intensive vaccination to build a wall of immunes about the newly recognized focus. This strategy also has been employed in West Africa since 1968 to supplement the program of general mass immunization now underway. Dubbed "eradication escalation", this new effort represents "a specific attack on transmission of smallpox when the disease is at its seasonal low ebb." As an intensive nationwide search leads to the recognition of new cases and epidemiologic investigations reveal their sources and contacts, intensive vaccination is employed to contain the focus. This strategy is believed to have greatly accelerated the very dramatic progress made in eradicating smallpox from this region (1).

Summary

Examples demonstrate that the potential for contact spread of an agent depends entirely on the number of susceptibles and their opportunities for contact with each other. The purpose of an immunization program is to reduce the supply of susceptibles to such an extent that the probability of spread is very small.

Free living populations of communities are made up of multiple and interlocking mixing groups, defined in such terms as families, family clusters, neighborhoods, playgroups, schools, places of work, ethnic and socioeconomic subgroups. These mixing groups are characterized by differing contact rates and by differing numbers of susceptibles. The optimum immunization program is one which will reduce the supply of susceptibles in all subgroups. No matter how large the proportion of immunes in the total population, if some pockets of the community, such as low economic neighborhoods, contain a large enough number of susceptibles among whom contacts are frequent, the epidemic potential in these neighborhoods will remain high.

Success of a systematic immunization program requires knowledge of the age and subgroup distribution of the susceptibles and maximum effort to reduce their concentration throughout the community, rather than aiming to reach any specified overall proportion of the population.

Addendum

Since this paper was submitted, a particularly appropriate illustration of the thesis presented has come to attention. Scott (10) has described epidemic measles in Rhode Island in 1968 which was virtually confined to an "ethnic island" (Portuguese, chiefly recent immigrants) in a highly vaccinated general population. In this episode, the agent was introduced from Portugal via a three-year-old child who was developing disease as he arrived.

Appendix

The Reed-Frost model

A full discussion of this model is given elsewhere (6, 7). The basic description and definitions are given below.

1) The population (of size N) is taken to be randomly-mixing.
2) The time scale is broken into equal intervals, the length of which equals the period of infectivity (during which an infected person may transmit the infection).
3) Adequate contact between two individuals is defined as that sufficient to result in transmission from an infectious person to a susceptible.
4) Contact rate, p = the probability that *any* two persons in the population

will, during any interval, make adequate contact.

Let N = the population size,
S_t = the number of susceptibles in the population at time t,
C_t = the number of cases (infectives) at time t (C_0 may be taken to be equal to one), and
I_t = the number of immunes at time t.

During the typical interval, time t to time $t + 1$:

1) The probability that any specified susceptible will escape contact with a given case is $q = 1 - p$:
2) The probability that this susceptible will escape contact with all C_t cases is q^{C_t}.
3) The probability that this susceptible will fail to escape contact with all C_t cases (and so become infected) is $1 - q^{C_t}$.

Thus, in this simple model, the expected number of new cases at time $t + 1$ (C_{t+1}) is simply the number of susceptibles (S_t) multiplied by the probability that any given susceptible will acquire infection ($1 - q^{C_t}$). Expressed mathematically, the expected

$$C_{t+1} = S_t(1 - q^{C_t}) \qquad \text{(equation 1)}$$

By repeated application of this equation for $t = 0, 1, 2, 3, \cdots$, the "expected epidemic" can be computed.

For each member of the population there are $N - 1$ potential contacts. The expected number of contacts per person per interval is

$$m = p(N - 1) \qquad \text{(equation 2)}$$

Note that these contacts may be with susceptibles, immunes, or cases and will be distributed in proportion to the values of S_t, I_t and C_t.

In the examples which follow it is our purpose to demonstrate that, given introduction of an agent into a community by the infection of one or more susceptibles, epidemic potential is completely determined by

S = the number of susceptibles and
p_s = the contact rate between susceptibles.

There is only one contact rate, p, in a randomly mixing population and, if both p and the number of susceptibles (S) are held constant, the contact rate between susceptibles (p_s) also will be constant. Given populations of differing size but with identical S and p, the number (and proportion) of immunes (I) will increase with population size. However, such increase in immunes will decrease the likelihood of an epidemic only if it reduces contacts between susceptibles. This was illustrated by the simple example described in the text and is more rigorously demonstrated in the example below.

In this example, we examine the operation of the foregoing simple rules, given the introduction of one infectious case into randomly mixing populations ranging in size from 400 to 10,000 while various pairs of specified characteristics are held constant and others are permitted to change. These characteristics are:

Characteristics	Symbol	Relationships
The population size	N	
The proportion immune	θ	$\theta = (1 - S/N) \times 100$
The contact rate	p	
The number of susceptibles	S	
The average number of contacts per person per interval	m	$m = p(N - 1)$ or approximately pN
The average number of contacts with susceptibles	m'	$m' = p(S - 1)$ or approximately pS

Because of the relationships between the above characteristics, there are only six ways in which we may choose two of them to hold constant. The results expected in each of these six situations or sets are presented in table 2 with respect to several parameters, some of which may interest only the statistically oriented reader. All readers, however,

TABLE 2

Probability that epidemic will abort with a single case and the median epidemic size under various conditions given one infectious case in a randomly mixing population

Set No.	Characteristics held constant	Population size	Proportion of population immune	No. of susceptibles	Contact rate	Average No. contacts (total)	Average No. contacts (with susceptibles)	Probability of no spread	No. of epidemics with one case	Median epidemic size	Mean No. of cases	Maximum No. of cases	Maximum % of susceptibles infected
			%					%	Observed distributions				
		N	θ	S_0	p	m	$m' = pS$	$P(A)$*	$C = 1$	Q_2†	$\bar{x}$		
1	No. of susceptibles, contact rate and therefore average No. contacts between susceptibles	400	0	400	.005	2	2 0	14	13	315	246	346	86
		5,000	92	400	.005	25	2.0						
		10,000	96	400	005	50	2.0						
2	Proportion immune, average No. contacts and therefore average No. contacts between susceptibles	2,000	96	80	.020	40	1.6	20	18	40	30	67	84
		5,000	96	200	008	40	1.6	20	24	106	74	157	78
		10,000	96	400	.004	40	1.6	20	17	244	183	307	77
3	Proportion immune, contact rate	2,000	96	80	.005	10	.4	67	64	1	1	6	8
		5,000	96	200	.005	25	1.0	37	39	2	9	79	40
		10,000	96	400	.005	50	2.0	14	13	315	246	346	86
4	Proportion immune, population size and therefore No. of susceptibles	5,000	96	200	.005	25	1.0	37	41	2	8	73	36
		5,000	96	200	008	40	1.6	20	24	106	74	157	78
		5,000	96	200	.010	50	2.0	14	12	157	131	181	91
5	No. of susceptibles, average No contacts	1,000	60	400	.004	4	1.6	20	17	244	183	307	77
		1,333	70	400	.003	4	1.2	30	36	3	38	193	48
		2,000	80	400	.002	4	.8	45	45	1	5	56	14
6	Contact rate, population size and therefore average No. contacts	2,000	80	400	.005	10	2.0	14	13	315	246	346	86
		2,000	90	200	.005	10	1 0	37	39	2	9	79	40
		2,000	96	80	.005	10	.4	67	64	1	1	6	8

Relationships among the parameters using the approximation N for $N - 1$ and S_0 for $S_0 - 1$ (S_0 = original number of susceptibles)

$$\theta = [1 - S_0/N] \times 100$$
$$m = pN \text{ and } m' = pS$$

Note that the expected number of cases at the beginning of the second interval is equal to m'

* $P(A)$ = *the probability that there will be no spread from the single case.*
$P(A) = (1 - p)^{S_0}$ was approximated here by $e - pS_0 = e - m'$.

† Q_2 is the observed median of the sizes of 100 epidemics simulated on the computer using the stochastic properties of the Reed-Frost model. See reference 8.

should pay attention to the columns indicating number of susceptibles (S_0), average number of contacts with susceptibles (m' or pS), probability of no spread and median epidemic size (Q_2). The important relations demonstrated are: 1) when the average number of contacts with susceptibles (pS) remains constant, so also does the probability of no spread (sets 1 and 2) although, naturally, the median epidemic size increases with the number of susceptibles (set 2); 2) the probability of no spread is inversely related to the average number of contacts with susceptibles, falling when pS rises (sets 3 and 4) and rising when pS falls (sets 5 and 6); and 3) population size, proportion immune and contact rate have no relation to the probability of spread or median epidemic size except when, under the conditions postulated, changes in these characteristics necessarily result in changes in S or number of susceptibles (sets 2, 3 and 6) or in pS, the average number of contacts with susceptibles (sets 3, 4, 5, and 6).

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