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PERSON-TO-PERSON TRANSMISSION OF *STAPHYLOCOCCUS AUREUS**

Quantitative Characterization of Nasal Carriers Spreading Infection

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The number of *Staphylococcus aureus* organisms disseminated by different carriers and the extent to which they contaminate their environment and transmit infection to their contacts have been shown to vary widely. Nasal carriers with purulent skin lesions or respiratory viral infections have been described as being especially apt to transmit staphylococci[1,2]; yet carriers lacking these conditions have also been observed to cause considerable environmental contamination and to spread infection to contacts.[3] Carriers whose nasal swabs yielded 10^5 or more *Staph. aureus* organisms in broth have been reported by White[4] to disperse more of these organisms to the air and onto their skin than those whose swabs yielded a smaller number. Whether the amount of a carrier's nasal infection, as estimated by counts from nasal swabs, reflects his ability to infect others is unknown. The ability of a few carriers of *Staph. aureus* to spread infection and to initiate hospital outbreaks is well documented[5-7]; the reasons for the unusual infectivity of such persons, however, remain to be established.

Person-to-person spread of experimentally inoculated *Staph. aureus* has recently been reported by Shinefield et al.,[8] who also demonstrated that colonization and disease due to 52/52A/80/81 strains of *Staph. aureus* were prevented when another strain was present in an infant's nose.[9] In preliminary studies in this laboratory, experimental inoculation of a tetracycline-resistant 52/52A/80/81 strain of *Staph. aureus* in the nose of both marmoset monkeys and healthy human subjects was found to be a safe procedure. One example of monkey-to-monkey transmission of the bacteria was observed among animals caged together and treated with tetracycline; however, this could not be demonstrated again in subsequent experiments. By contrast, under parallel conditions of proximity and tetracycline treatment, man-to-man transmission was repeatedly found.

The present investigation describes quantitative aspects of nasal carriage in 1 natural and 2 artificially induced human carriers in relation to their spread of 52/52A/80/81 *Staph. aureus* to various persons, including persistent carriers of other strains. This study gives evidence that spread of tetracycline-resistant *Staph. aureus* in man is enhanced by tetracycline treatment of the carrier.

Methods

Study families were selected as a result of an earlier community survey for tetracycline-resistant *Staph. aureus.* Each family was of middle-income circumstances and was composed of 2 adults and at least 5 children who lived in one-family houses in the Greater Miami area. In no families was there any person with recent skin or other infection or ill-health necessitating antibiotic therapy.

Nose cultures for staphylococci were carried out

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twice weekly by heavy swabbing of both anterior nares with a broth-moistened swab, which was then streaked on trypticase soy agar containing 7.5 per cent sodium chloride[10]; a second nasal swab was streaked on similar medium containing 25 microgm. of tetracycline per milliliter.[11] Ten colonies from each plate were tested for coagulase production on agar containing approximately 1 per cent bovine fibrinogen and 0.25 per cent fresh human plasma.[12] All coagulase-positive colonies were phage typed at routine test dilution with a set of 21 phages by a standard method,[13] modified only by the use of a special dispenser.* Nontypable strains and organisms of the "80/81" complex were retyped at a hundred times the routine test dilution for further identification.

Quantitative nose cultures for tetracycline-resistant organisms were performed by White's method with modifications.[14] A nasal swab was mechanically shaken in broth for ten minutes, five serial tenfold dilutions of the broth were made, and 0.1 ml. of each dilution was spread on tetracycline-fibrinogen agar for counts and coagulase testing. Ten colonies of *Staph. aureus* from the highest dilution were phage typed.

Air sampling was done with a TLD air-slit sampler† containing a tetracycline agar plate making 2 revolutions per minute for six hours; the rate of air-flow was calculated to be approximately 1 cubic foot per minute. All colonies present after forty-eight hours' incubation were tested for coagulase production, and all strains of *Staph. aureus* were phage typed.

The indicator *Staph. aureus,* a coagulase-positive, mannitol-positive, 52/52A/80/81 strain readily growing in medium containing tetracycline or penicillin in a concentration of 25 microgm. per milliliter, had repeatedly been recovered from 1 member (H.G.) of a study family for a year before these experiments. No organisms similar in phage type or antibiotic resistance were encountered in the other study families.

Tetracycline and alpha-phenoxyethyl penicillin (phenethicillin), obtained commercially, were administered in a dosage of 250 mg. four times daily for study purposes.

Results

Spread of the Indicator *Staph. aureus* in the G. family

Penicillin and tetracycline treatment of H.G., eight years of age, a persistent carrier of the indicator *Staph. aureus,* had different effects upon his dissemination of bacteria. With each of three courses of tetracycline he spread the indicator strain to 1 or more of his family contacts at risk‡ whereas after each course of penicillin he did not (Table 1). Infection in family contacts lasted no more than two months.

Table 1. *Effect of Consecutive Courses of Penicillin and Tetracycline on H.G.'s Spread of the Indicator* Staph. aureus *to Other Members of the G. Family.*

Treatment	Duration	Observation Period after Treatment	Contacts Infected/Contacts at Risk
	days	*days*	
Penicillin	7	14	0/5
Penicillin	12	21	0/4
Tetracycline	7	14	1/3
Penicillin	10	21	0/3
Tetracycline	7	14	2/5
Tetracycline	14	14	1/6

There was a difference in the effect of each antibiotic upon H.G.'s aerial dispersal of bacteria during sleep, as is evident in results of air sampling carried out at a distance of 6 feet from his bed from 11 p.m. to 5 a.m. each night (Fig. 1). For 32 nights during and after two courses of penicillin less than 400 tetracycline-resistant colonies of a variety of bacteria were recovered each night, and generally the number was less than 200. After tetracycline treatment, however, counts were 400 or more tetracycline-resistant colonies on 11 of 18 nights (p less than 0.001), and on 2 nights counts were as high as 850 and 1000. The indicator *Staph. aureus* itself was present on but 6 of 32 nights after penicillin treatment whereas after tetracycline treatment this strain occurred on 13 of 18 nights (p less than 0.001). After penicillin treatment 2 indicator *Staph. aureus* colonies occurred but once; after tetracycline treatment 2 to 6 colonies were present on 8 nights.

Tetracycline treatment of H.G. resulted in an increase of indicator *Staph. aureus* to 10^3 or more per nasal swab for several days to a week or more, and such an increase was associated with spread of infection to his contacts (Fig. 2). The increase to 10^3 colonies of indicator *Staph. aureus* per swab occurred within ten days of the start of tetracycline treatment; a similar increase did not take place as a consequence of penicillin treatment (Fig. 3).

Spread of the Indicator *Staph. aureus* in the K. Family

H.G. was moved into the K. household for one week to test the thesis that he could be made to spread the indicator *Staph. aureus* to persons outside his family. For a two-week period beginning with the week before his visit and lasting throughout his stay he was treated with tetracycline. During the week of H.G.'s visit to the K. home M.K., seven years of age, was moved to the G. home and slept in H.G.'s bed, with its wool blanket unchanged. On the sixth day of H.G.'s visit the indicator *Staph. aureus* was recovered from 2 K. family members, and a few days later this orga-

*In the form of Accu-Drop, Sylvania Corporation, New York City.
†Available from Elliot Engineering Company, Decatur, Georgia.
‡Family members were considered to be contacts at risk when their nose cultures failed to yield the indicator *Staph. aureus* for at least one month before the test period.

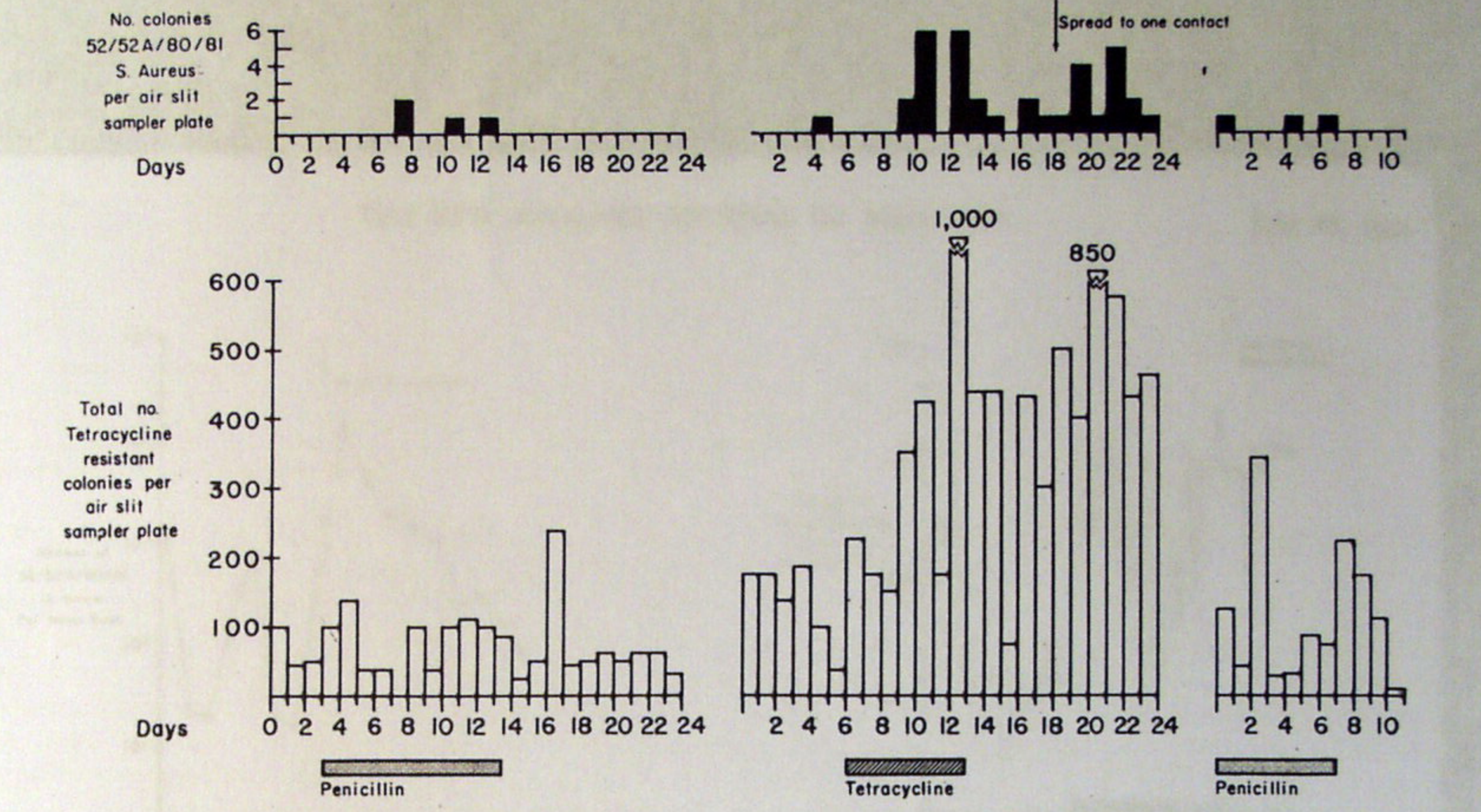

FIGURE 1. *Nightly Aerial Dispersal of a Variety of Tetracycline-Resistant Bacteria, and the Indicator* Staph. aureus *Specifically, by H.G. during Sleep, from 11 p.m. to 5 a.m. — Effects of Penicillin and Tetracycline Treatment in Consecutive Courses.*

nism was cultured from 2 additional members of the K. family. The indicator *Staph. aureus* was then recovered repeatedly from 1 member of the K. family over a six-week period, but no additional spread of infection was detected after H.G.'s departure. M.K. was not found to have acquired the indicator *Staph. aureus* during his stay in the G. home or afterward.

To study transmission of the indicator *Staph. aureus* in the K. family by an artificially induced carrier, M.K. was infected one year later by nasal inoculation of 4×10^6 organisms from an overnight culture of the indicator *Staph. aureus.* He received no antibiotics during the two months in which he was a carrier; nonetheless, his twice-weekly nasal counts of the indicator *Staph. aureus* were found to range to 10^3 or more per swab during a six-week

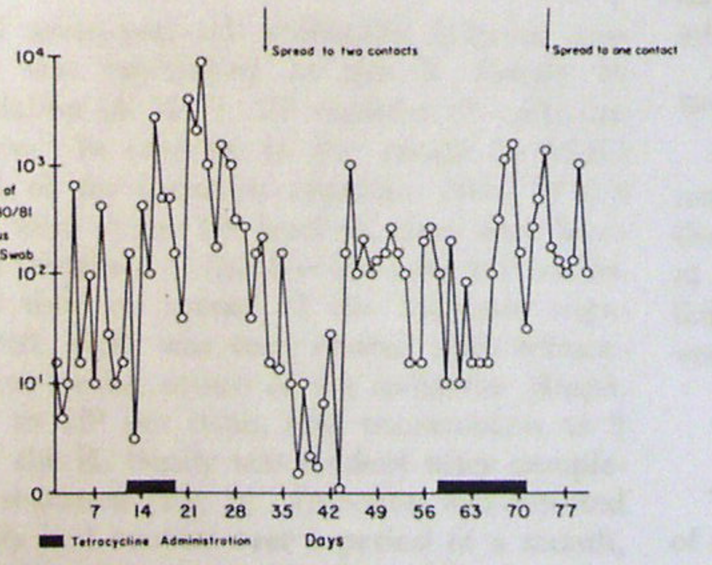

FIGURE 2. *Spread of the Indicator* Staph. aureus *by H.G. in the G. Family — Daily Counts of Nasal Swabs and Effects of Tetracycline Treatment.*

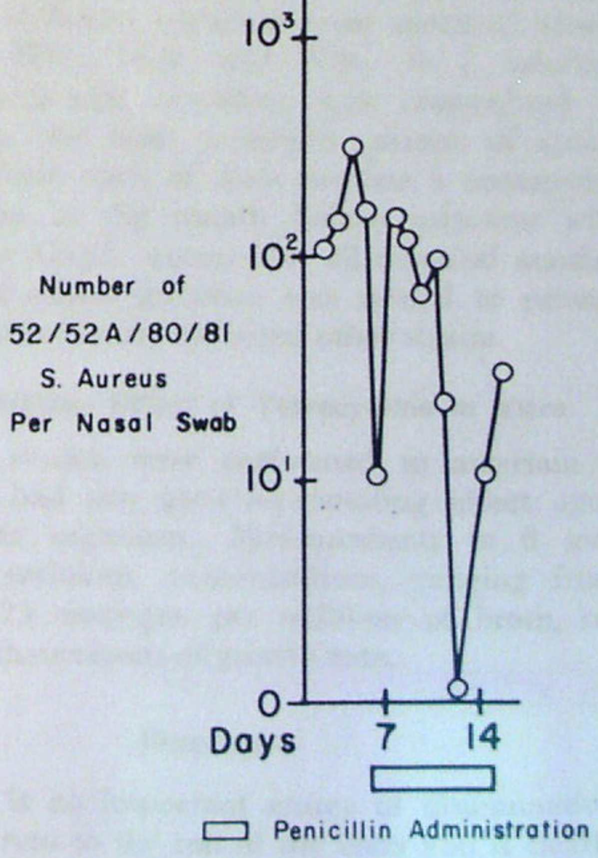

FIGURE 3. *Effect of Penicillin Treatment of H.G. on Daily Counts of Indicator* Staph. aureus *— Note the Lack of Increase to 10^3 per Nasal Swab.*

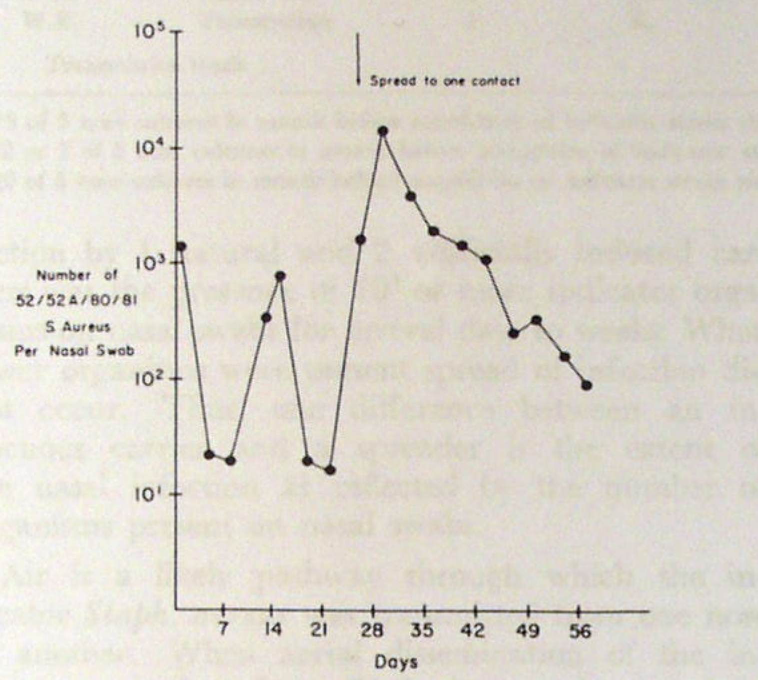

FIGURE 4. *Spread of the Indicator* Staph. aureus *by M.K. in the K. Family in the Absence of Treatment — Twice-Weekly Counts of Nasal Swabs.*

M.K. was inoculated with 4 × 10⁶ indicator organisms on the first day.

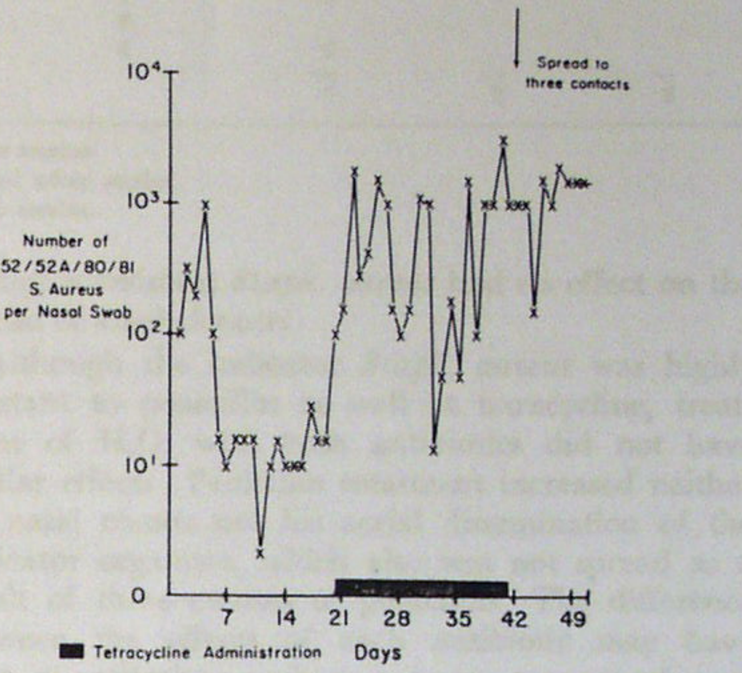

FIGURE 5. *Spread of the Indicator* Staph. aureus *by W.R. in the R. Family — Daily Counts of Nasal Swabs and Effects of Tetracycline Treatment.*

W.R. was inoculated with 16 × 10⁶ indicator organisms on the first day.

interval. As the counts were seen to reach their peak, spread of infection to 1 contact was observed (Fig. 4). Infection in the contact was noted over a three-week period. Toward the end of this time a nasal swab of the contact was found to yield 10^1 colonies of indicator *Staph. aureus*; no secondary spread of infection was detected.

Spread of the Indicator *Staph. aureus* in the R. Family

A second seven-year-old artificially induced carrier, W.R., was established in the R. family by nasal inoculation of. 16×10^6 colonies of indicator *Staph. aureus*. In contrast to the results in M.K., daily counts of the indicator organism from W.R.'s nasal swabs were at the 10^1 level six days after inoculation, and remained at this low level for two weeks. During this time no spread of the indicator organism occurred. W.R. was then treated with tetracycline for three weeks; counts of the indicator *Staph. aureus* rose to 10^3 per swab, and transmission to 3 members of the R. family was evident after completion of the treatment (Fig. 5). Infection was detected *intermittently* in 1 contact over a period of a month, but no secondary spread was observed. The duration of W.R.'s nasal infection after tetracycline treatment was two months.

Spread in Families Lacking the Indicator *Staph. aureus*

Two families with a total of 9 persistent carriers of strains not highly resistant to tetracycline were also studied. In this experiment tetracycline was administered for one week not only to the carriers but also to all contacts in both families. No person-to-person spread of any of the strains was observed.

Spread of the Indicator *Staph. aureus* to Carriers of Other Staphylococci

As summarized in Table 2, 12 cases of transmission of the indicator organism were observed among contacts of H.G., M.K. and W.R. In 7, infection with the indicator organism was transmitted to persons who had been persistent carriers of another strain. From each of these persons 5 consecutive nose cultures in the month before infection with the indicator *Staph. aureus* had all revealed another strain. In 2 others infection was spread to persons who had intermittently harbored other strains.

Growth-Promotion Effect of Tetracycline in Vitro

In vitro studies were performed to ascertain if tetracycline had any growth-promoting effect upon the indicator organism. Measurements in 6 serial tenfold antibiotic concentrations, ranging from 0.00025 to 25 microgm. per milliliter of broth, revealed no enhancement of growth rate.

Discussion

The nose is an important source of dissemination of *Staph. aureus* to the rest of the body and is clearly an origin of suppurative disease in those who have open wounds or who are otherwise susceptible.[15,16] Hence, a knowledge of how nasal infection* with *Staph. aureus* spreads from one person to another and the way an innocent carrier may be converted into an active spreader is fundamental in an understanding of the epidemiology of suppurative staphylococcal infections occurring sporadically and in outbreaks. In the present study, the one condition consistently associated with spread of in-

*Infection is used here in the manner employed by Dubos[17] to include the phenomena of the carrier state and latent infection.

TABLE 2. *Summary of Transmission Experiments with the Indicator* Staph. aureus.

Carrier	Antibiotic Treatment	No. of Courses	Contact Family	Contacts Acquiring Indicator Organism	Contacts at Risk	Pretreatment State of Contact Acquiring Indicator Organism		
						Persistent* Carrier of Other Strains	Intermittent† Carrier of Other Strains	Noncarrier‡
H.G.	Tetracycline	3	G.	4	14	3		1
H.G.	Penicillin	3	G.	0	12			
H.G.	Tetracycline	1	K.	4	6		2	2
M.K.	None	1	K.	1	7	1		
W.R.	None	1	R.	0	6			
W.R.	Tetracycline	1	R.	3	6	3		
Transmission totals				12		7	2	3

*5 of 5 nose cultures in month before acquisition of indicator strain yielded other strains.
†2 or 3 of 5 nose cultures in month before acquisition of indicator strain yielded other strains.
‡0 of 5 nose cultures in month before acquisition of indicator strain yielded other strains.

fection by 1 natural and 2 artificially induced carriers was the presence of 10^3 or more indicator organisms on nasal swabs for several days to weeks. When fewer organisms were present spread of infection did not occur. Thus, one difference between an innocuous carrier and a spreader is the extent of the nasal infection as reflected by the number of organisms present on nasal swabs.

Air is a likely pathway through which the indicator *Staph. aureus* was transmitted from one nose to another. When aerial dissemination of the indicator organism from H.G. increased, spread of infection occurred (Fig. 1). The absence of acquisition of the indicator organism by M.K. during his one-week stay in the G. house, while he slept in H.G.'s bed, indicates that in this case, intimate contact with a carrier's fomites was not sufficient for transmission of infection; it is to be emphasized that the fomites included the unaltered wool blanket previously used by H.G. and that such blankets are reported to serve as a reservoir of 80/81 *Staph. aureus*.[18]

The increase in number of tetracycline-resistant organisms in a carrier's nose after tetracycline treatment is most probably a consequence of reduction of competing or inhibiting bacteria.[19] In vitro studies gave no evidence that tetracycline directly stimulated growth of the indicator *Staph. aureus*. Furthermore, tetracycline treatment increased the aerial dissemination of other tetracycline-resistant organisms (Fig. 1). Suppression of nasal bacteria by tetracycline treatment is also a possible explanation for the increased rate of acquisition of nasal infection with tetracycline-resistant *Staph. aureus* in hospitalized patients.[20,21] Since exposure to tetracycline-resistant strains is not uncommon in present-day hospitals, tetracycline treatment in such an environment not only may cause a patient to become a nasal carrier but also may result in suppurative disease in him or his contacts, if the infection in the nose attains a spreading level. By contrast, as seen in the present study, tetracycline treatment at home of persons lacking close contact with carriers of highly resistant *Staph. aureus* had no effect on the spread of staphylococci.

Although the indicator *Staph. aureus* was highly resistant to penicillin as well as tetracycline, treatment of H.G. with both antibiotics did not have similar effects. Penicillin treatment increased neither his nasal counts nor his aerial dissemination of the indicator organism, which also was not spread as a result of three courses of penicillin. The difference between the effects of each antibiotic may have been quantitative; perhaps a larger amount of penicillin would have induced responses similar to those of tetracycline. Alternatively, the difference may have been qualitative by virtue of a difference in antibacterial action of each drug. Studies of hospital-acquired infection suggest that tetracycline changes the bacterial flora of the host to a greater extent than penicillin.[21,22] Other antibiotics might also induce spread of staphylococcal infection if the carrier's *Staph. aureus* was appropriately resistant and if his usual microflora was sufficiently altered.

Untreated after inoculation of the indicator *Staph. aureus,* M.K. experienced infection for two months. During this time counts of his nasal swabs exceeded 10^3 indicator organisms, which he transmitted to 1 contact (Fig. 4). By contrast, after his inoculation, W.R. had counts in excess of 10^3 indicator organisms per swab only after tetracycline treatment (Fig. 5). Infection in W.R. lasted for only two months after treatment; similarly, infection with the indicator *Staph. aureus* in all contacts persisted for no more than two months. The duration of natural infection in H.G. with the indicator organism was more than one year. In some persons nasal infection with 1 strain of *Staph. aureus* may last for several years.[23,24] The physiologic events in the host that govern both the amount and the duration of nasal infection remain to be determined.

Seven cases of nasal infection were detected among contacts who were persistent carriers of other strains of *Staph. aureus* (Table 2); this is noteworthy since Shinefield et al.[8] have reported that nasal colonization of infants precludes subsequent implantation of

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Nasal abnormality and the carrier rate of *Staphylococcus aureus*

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SYNOPSIS In a group of 178 hospital nurses minor nasal abnormalities were found to be associated with an increased rate of nasal carriage of *Staphylococcus aureus*.

The *Staphylococcus aureus* is found in the anterior nares of 30 to 50% and on the skin in 12 to 25% of healthy adults (Miles, Williams, and Clayton-Cooper, 1944; Williams, Blowers, Garrod, and Shooter, 1960). It is not known why some people carry the organism and others do not, although nasal carriage predisposes to skin carriage as the result of passive transfer. Despite this relationship, in about half the cases of skin carriage the organism is not found in the nose (Miles *et al.*, 1944), neither is there any correlation between the degree of nasal colonization and the intensity and extent of contamination of the skin (Hare and Ridley, 1958).

The incidence of staphylococcal carriage is apparently random and it has been stated that neither congenital anomalies of the upper respiratory tract (Cunliffe, 1949) nor non-staphylococcal disease of the nose (Williams *et al.*, 1960) predisposes to a higher nasal carrier rate. Although this may be true for the staphylococcus, McCartney and Harvey (1928) found that in the case of *Corynebacterium diphtheriae* nasal abnormality favoured the development of the carrier state. Subtle constitutional differences of the skin have been suggested as the reason for staphylococcal carriage on the integument (Tulloch, 1954) but this has not been demonstrated experimentally.

The paper records an attempt to determine some of the factors that may predispose to staphylococcal colonization of the nose and the skin in individuals without any obvious staphylococcal infection.

METHODS

One hundred and seventy-eight nurses, some of whom were in the preliminary training school and had not been in contact with a hospital environment, formed the group for study.

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CLINICAL ASSESSMENT An E.N.T. and dermatological history of each subject and her immediate family was taken. An aural surgeon examined the nose and throat in each individual and noted any clinically significant abnormality, and a dermatologist placed the subject into one of four skin types, namely, normal, dry, moist, or greasy. The presence of minor skin sepsis was also noted. A special note was made of recent treatment with chemotherapeutic agents.

BACTERIOLOGICAL SAMPLING Three swabs moistened with nutrient broth were taken from the following sites: the palmar surface of the left wrist, the nasal vestibule, and the mucosa immediately above the anterior end of the inferior turbinate. Under direct vision and with instrumentation an aural surgeon swabbed the inferior turbinate. A fourth swab was taken from the tonsillar area in the last 66 nurses.

The swabs were plated on to nutrient agar without delay. After 48 hours' incubation the plates were examined for coagulase-positive staphylococci. The phage and sensitivity patterns of these were determined. To determine sensitivity, Sentest (Evans) tablets were used on nutrient agar plates previously seeded with 1 ml. of a 1 in 100 dilution of an 18-hour broth culture.

RESULTS

The incidence of skin carriage was so low (7·25%) that any correlation with clinical skin types was unjustified.

The overall incidence of nasal carriage was 38% (68 out of 178). With one exception all were vestibular carriers, and in almost half (17%) carriage extended far enough to involve the mucosa above the inferior turbinate.

The staphylococci isolated were distributed amongst all phage groups (Table I). Table I also records their susceptibility to antibiotics. It can be seen that the populations studied, with respect to the character of the staphylococci present, were

TABLE I

CHARACTERISTICS OF STAPHYLOCOCCI ISOLATED FROM THE NOSES OF 178 NURSES

Sensitivity Patterns of Strains for *Penicillin*	*Streptomycin*	*Tetracycline*	*Erythromycin*	*No. of Ward Nurses Yielding Strains of Staphylococci* — *This Sensitivity Pattern*	*Phage Group* *I*	*II*	*III*	*Others*	*No. of Preliminary Training School Nurses Yielding Strains of Staphylococci* — *This Sensitivity Pattern*	*Phage Group* *I*	*II*	*III*	*Others*
S[1]	S	S	S	3	1	2	0	0	14	6	4	2	2
R[2]	S	S	S	35	19	1	3	12	6	3	2	1	0
R	R	S	S	1	0	0	0	1	0	0	0	0	0
R	R	R	S	4	0	0	3	1	0	0	0	0	0
R	R	R	R	5	0	0	5	0	0	0	0	0	0
Total No. of Nurses													
Yielding staphylococcus strains				48	20	3	11	14	20	9	6	3	2
Examined				107					71				
Percentage of nurses positive				45	19	2·7	10·3	13	28	12·6	8·5	4·2	2·7

[1]S = sensitive; R[2] = resistant
No strain was found to be resistant to either chloramphenicol or novobiocin.

TABLE II

PHAGE GROUP DISTRIBUTION OF STAPHYLOCOCCI ISOLATED FROM NURSES WITH NORMAL AND ABNORMAL NOSES

Nurses with	*Where Stationed*	*No. Carrying Staphylococci of Phage Group* *I*	*II*	*III*	*Others*	*No. of Non-carriers*	*Total*
Abnormal noses	Ward Preliminary training school	4 0 } 4	0 0 } 0	3 2 } 5	4 0 } 4	4 3 } 7	20
Normal noses	Ward Preliminary training school	16 9 } 25	3 6 } 9	8 1 } 9	10 2 } 12	55 48 } 103	158
Total		29	9	14	16	110	178

comparable to those of previous workers, *e.g.*, Barber and Burston (1955).

A clinically significant minor nasal abnormality, *i.e.*, a deviated septum or damaged turbinate, was found in 20 individuals and of these 13 (65%) carried a coagulase-positive staphylococcus in the nose. In the remainder, those having normal noses, 55 (29%) were nasal carriers. The incidence of staphylococcal carriage in the group with nasal anomalies was significantly higher than that in the group with normal noses ($\chi^2 = 6{\cdot}88$, n = $0{\cdot}01 > p > 0{\cdot}001$).

This relationship was further strengthened by the finding that staphylococcal carriage in the throat was almost exclusively confined to those individuals who showed some abnormality of the tonsil (Campbell, 1948). Thus, of 66 subjects examined, six had tonsillar disease and all of these carried *Staphylococcus aureus* in the throat. On the other hand, of the 60 subjects with normal throats only one carried a *Staphylococcus aureus* in this area. These findings are analogous to those of Hartley and Martin (1920), who found that the persistence of carriage of *Corynebacterium diphtheriae* was apparently related to the presence of diseased tonsils. In the present investigation no relationship was observed between mucosal carriage and tonsillar carriage (Dingle and Plummer, and others, 1949).

Nasal abnormality does not favour colonization of the nose by staphylococci of any particular phage group (Table II), nor does it predispose to involvement of the upper reaches of the nose (Table III).

TABLE III

EXTENT OF COLONIZATION OF NORMAL AND ABNORMAL NOSES BY STAPHYLOCOCCI

Nurses with	*No. Carrying Staphylococci on/in* *Mucosa Alone*	*Mucosa and Vestibule*	*Vestibule Alone*	*Total*
Abnormal noses	0	6	7	13
Normal noses	1	25	29	55
Total	1	31	36	68

DISCUSSION

It has been stated that nothing is gained in the estimation of staphylococcal carrier rates by swabbing the upper reaches of the nose, and indeed that misleadingly low carrier rates may be obtained by so doing (Williams *et al.*, 1960). Stratford *et al.* (1960), however, isolated *Staphylococcus aureus* from mucosal swabs in 51% of a group of 103 patients but from vestibular swabs taken from the same group in only 34%, a result that throws some doubt on the obvious criticism of mucosal sampling that the organisms isolated might be contaminants derived from the vestibule. The technique used by Stratford, Rubbo, Christie, and Dixson (1960) is not detailed; in our tests the vestibule was opened out with a Thudichum speculum and samples taken from the clearly visualized inferior turbinate region. In our experience the mucosa is rarely involved alone.

In view of the findings of Cunliffe (1949), who found no increase in the staphylococcal carriage rate of children with gross abnormality of the nasopharynx, our finding that there is a higher rate of nasal carriage in nurses with minor nasal abnormalities might appear surprising, However, this result for the staphylococcus is in accord with that of McCartney and Harvey (1928) for *Corynebacterium diphtheriae*, who found that 72% of diphtheria carriers showed nasal abnormalities, in contrast to patients who had recovered clinically from diphtheria and were also bacteriologically free in whom the abnormality rate was only 7%. Moreover in the discussion on the epidemiology of scarlet fever, Wilson and Miles (1955) suggest that abnormalities of the upper respiratory tract prolong carriage of *Streptococcus pyogenes*.

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