



How Should Normal Flora, Colonization, and Carriage Identified in Throat/Nasopharyngeal Cultures be Interpreted Clinically?

Boğaz/Nazofaringeal Kültürde Normal Flora, Kolonizasyon ve Taşıyıcılık Klinik Açıdan Nasıl Yorumlanmalıdır?

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Question: How should normal flora, colonization, and carriage identified in throat/nasopharyngeal cultures be interpreted clinically? **Dr. Samed Çavdar**

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Answer (Sinem İrez Çetin, MD; Mustafa Kemal Hacımustafaoğlu, MD)

Introduction and general information: Before answering this question, it would be useful to briefly evaluate host-pathogen relationships in terms of commensalism, colonization/carriage, infection, and the development of disease. Infection is defined as the invasion of the body by a pathogenic agent with the potential to cause disease. An infection accompanied by overt clinical signs and symptoms is termed disease. Infection does not always lead to overt signs of disease; it may sometimes present with mild clinical findings or remain asymptomatic. In many cases, the terms infection and disease are used interchangeably. In this article, these terms are also used in similar senses.

The presence of an agent known to be pathogenic at non-sterile sites in the host, such as the nasopharynx/throat, skin, gastrointestinal tract, or vagina, does not necessarily

cause a pathological condition or infection. In some circumstances, it may even benefit the host. The combined effects of the anatomical site, host resistance, and external factors create a dynamic host-pathogen relationship that influences whether infection develops.

Definitions of concepts such as commensalism, colonization, and carriage may vary among different sources and can therefore be confusing. In this article, these concepts are addressed by the authors particularly within the framework of clinical significance, the epidemiological risk of transmission, and patient management. These relationships are evaluated primarily in the context of host-microbial agent interactions in the nasopharyngeal/throat region.

Blood, cerebrospinal fluid (CSF), urine, bile, and synovial fluid are considered fluids from sterile sites and do not contain microorganisms. As a rule, microbial growth in fluids from these sites is regarded as true growth. Occasionally, when

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appropriate culture procedures are not followed, normal-flora or environmental bacteria may be inoculated during the collection of specimens from sterile sites, such as blood cultures, tracheal aspirates, or urine cultures. This is regarded as contamination (1). Contamination is the detection, at a sterile site—and sometimes at a non-sterile site—of microorganisms transferred from another site or originating from the laboratory or environment (2). The sinuses and lower respiratory tract are generally considered sterile. Bacteria reaching these sites through contiguous spread or aspiration are rapidly cleared by normal local defense mechanisms, thereby potentially preventing the development of infection (1).

Normal flora is a term used to describe microorganisms, most commonly bacteria, which are present on and colonize the internal surfaces, such as the gastrointestinal tract and vagina, or external surfaces, such as the nose, nasopharynx, and mouth, of healthy individuals (1). The term normal flora primarily refers to bacterial agents. Viruses are not considered components of the normal respiratory flora; their detection at any time suggests infection. Such an infection may present with signs of disease, mild findings, or a subclinical course (2). Viral agents may remain in the nasopharynx/throat for some time before clinical findings appear, during the illness, or after the clinical findings have resolved. This should not be interpreted as indicating that the virus is part of the normal flora or represents colonization or carriage. The detection of *Candida* in a throat culture is generally regarded as part of the oral flora and is not considered evidence that it is the causative agent of infection.

Normal flora may vary among different age groups, between individuals, within the same individual over time, according to season, and with exposure to crowded environments, such as daycare centers, crowded classrooms, and dormitories (1). For example, the average prevalence of nasal *Staphylococcus aureus* colonization in the community is 20–35%; nasopharyngeal pneumococcal colonization is detected in approximately 40–50% of healthy children and 20–30% of healthy adults. Antibiotic treatment and vaccination may affect nasopharyngeal bacterial colonization (1).

The microbial agents constituting the normal flora may be termed commensal, colonizing, or endogenous microorganisms. They generally do not harm the host (1). Depending on their virulence characteristics, bacteria within the normal flora may be commensal, pathobiont, or pathogenic bacteria.

Normal flora consists predominantly of commensal bacteria. Commensalism is defined as a relationship in which a bacterium living in the same environment in the host benefits from that environment, while neither the host nor the other bacteria with which it coexists are harmed or affected—or de-

rive any additional benefit. Mutualism, by contrast, involves reciprocal benefit between two coexisting species (3). Commensal bacteria are defined as bacteria that, in principle, neither harm nor benefit the host or other coexisting bacteria and that share the nutrients available in the same environment—literally, “eating at the same table.” Their presence in the normal flora does not harm the host and may be considered beneficial because they competitively reduce the abundance of definite or potential pathogenic bacteria. In the gastrointestinal tract, for example, some commensal bacteria benefit the host by contributing to vitamin synthesis, releasing products that reduce colonization by pathogenic microorganisms, or competing with potentially pathogenic bacteria for nutrients. Similar host-protective effects may occur with commensal bacteria on the skin or in the throat/nasopharyngeal region. Commensal bacteria may release metabolites and nutrients that benefit the host and may also support other beneficial bacteria. Their presence in the normal flora is therefore considered favorable for the host (4). Commensal bacteria may have direct or indirect beneficial effects on the immune system; however, a conventional systemic antibody response to commensal bacteria generally does not develop (2,4,5).

Commensal bacteria generally have low virulence. In a normal host, they are not true pathogens in principle and do not cause infection. However, in the presence of substantial immunosuppression, they may act as opportunistic pathogens and cause infection (3). Furthermore, a commensal bacterium at one anatomical site may act as a pathogen at another. For example, *Escherichia coli*, which is generally considered a commensal and constitutes a major component of the normal gastrointestinal flora, may cause a urinary tract infection if it reaches the urinary tract in the same host. Likewise, *E. coli* that forms part of the maternal normal gastrointestinal flora may cause infection in a newborn infant.

Pathobionts are bacteria that normally live as part of the throat/nasopharyngeal flora without harming the host. However, following dysbiosis in the throat/nasopharynx caused by various factors or conditions, they may acquire a pathogenic phenotype and cause infection. Causes of such dysbiosis include impairment of local defenses, reduced host resistance, damage to the mucosa harboring the normal flora—for example, following a viral upper respiratory tract infection—or a marked reduction in the protective flora/commensals after inappropriate antibiotic use. Pathobionts belonging to the normal respiratory flora include *Streptococcus pneumoniae*, *Moraxella catarrhalis*, and *Haemophilus influenzae* (6–8). Definite bacterial pathogens, such as *Neisseria gonorrhoeae*, *Mycobacterium tuberculosis*, *Mycoplasma pneumoniae*, and *Bordetella pertussis*, are not components of the normal flora and, when detected in association with clinical findings—or in a

subclinical infection—should always be considered causative agents of disease.

Among bacteria that form part of the normal respiratory flora and exhibit pathobiont characteristics, pneumococci, non-typeable *H. influenzae*, *M. catarrhalis*, and *Neisseria meningitidis* predominantly colonize the nasopharynx, whereas group A streptococci mainly colonize the throat and oropharynx (2).

Bacteria colonizing the nasopharynx, such as pneumococci and *N. meningitidis*, are spontaneously eliminated over time and may be replaced by another serotype. Simultaneous colonization with multiple serotypes may also occur (2,4). Infection may develop in only a very small proportion of hosts carrying colonizing bacteria. Factors such as viral infections, including influenza, and smoking facilitate progression from colonization to infection. The risk of progression to infection is generally highest during the first days after colonization begins, usually within 1–10 days (2,9). However, the precise time at which colonization began usually cannot be determined.

At sites harboring normal flora, such as the skin, nose, throat, and gastrointestinal tract, host–pathogen interaction does not usually result in disease. Indeed, the presence within the normal flora of bacteria that are not ordinarily harmful supports the host. Nevertheless, during periods when regional host resistance is reduced for various reasons, such as after a viral infection, trauma, antibiotic use, or exposure to immunosuppressive drugs or conditions, potentially pathogenic bacteria—pathobionts—that are members of the normal flora may cause infection. In rarer circumstances, commensal bacteria, such as *Staphylococcus epidermidis* on the skin, may also cause opportunistic infection in immunosuppressive states (4). Consequently, the pathogenic potential of a bacterium within the normal flora is not fixed and does not remain stable over time. The harmlessness–infection–disease continuum in bacterium–host interactions is closely associated with the bacterium’s potential virulence characteristics and changes in the host’s systemic and local resistance.

Colonization is defined as the proliferation of a bacterium at a particular site in the host, such as the throat/nasopharynx or nose, without causing clinical findings or disease, for a certain period and to concentrations higher than would normally be expected (1).

Colonization is not accompanied by signs or symptoms of infection/disease. It may persist for days or months. It may resolve spontaneously under the influence of the host immune response or other factors—sometimes antibiotics prescribed for another reason or vaccination—or through competition with other microorganisms of the normal flora (2). Colonization may diminish and disappear over time. As colonization by

one species disappears, new colonization by different species or by different serotypes of the same species may develop. Neutralizing antibodies may develop against colonizing bacteria without clinical findings (2,4,5). Colonization may be terminated by the host immune response, as in the spontaneous resolution of nasopharyngeal *N. meningitidis* or *S. pneumoniae* colonization/carriage over time. A colonizing bacterium differs from a commensal in that it can cause infection in a normal host, without the presence of immunosuppression, when appropriate local conditions arise. An example is the development of acute otitis media following a viral infection due to *S. pneumoniae* colonizing the nasopharynx. It should nevertheless be remembered that many types of *N. meningitidis* may colonize the nasopharynx and that many of these have low virulence—particularly non-groupable, non-encapsulated *N. meningitidis* types—and are therefore unlikely to cause infection/disease. Consequently, colonization with these meningococcal types, which have low virulence and limited pathogenic potential, may indirectly have a more favorable effect on the host (10).

Carriage is generally regarded as the persistence of true pathogens at a particular site, such as the nasopharynx or gastrointestinal tract, usually for a longer period than colonization. Actually, the full set of characteristics associated with carriage applies primarily to carriage in the gastrointestinal tract. In carriage, there are no signs of active infection or disease, and the host is no longer at risk of infection because of the carrier state. A specific and protective immune response against the pathogen develops in the host; however, this response does not eliminate the proliferation responsible for carriage at that site. Carriage is clinically important from an epidemiological perspective. Although the carrier state does not pose a particular risk to the host, it creates a risk of transmitting the true pathogenic microorganism to others and causing infection in them. On the contrary From a public health perspective, the risk of transmitting colonizing bacteria to others is low in colonization. Carriers, however, pose a higher risk of transmission and should be taken into consideration (3). A specified duration is generally required to define carriage. For gastrointestinal *Salmonella* carriage, this period is ≥ 1 year. Treatment of the carrier state is more difficult and prolonged than treatment of an ordinary *Salmonella* infection.

Although colonization and carriage have distinct characteristics, particularly in the gastrointestinal tract, many sources use the terms interchangeably for the throat/nasopharyngeal region. Examples include nasal *S. aureus*; nasopharyngeal pneumococcal, meningococcal, non-typeable *H. influenzae*, and *Moraxella catarrhalis* colonization or carriage; and group A streptococcal colonization or carriage in the throat. No clear distinction is made between the two concepts (4,5,11,12).

In this respect, throat/nasopharyngeal and nasal carriage/colonization should be evaluated differently from the concept of gastrointestinal *Salmonella* carriage in terms of the development of infection in the host and transmission to others. No specific standard duration is defined for throat/nasopharyngeal carriage. For example, no standard duration has been established for nasal *S. aureus* carriage/colonization, nor is there a standardized definition specifying how many cultures should be obtained or at what intervals to establish carriage (12,13). *S. aureus* carriage may be persistent or non-persistent. Persistent carriage occurs in approximately 20% of the general population (12). Unlike gastrointestinal *Salmonella* carriage, *S. aureus* carriage increases the host's risk of staphylococcal infection. Most nosocomial *S. aureus* bacteremia (>80%) originate from an endogenously colonizing strain. Studies have shown, however, that although infection is more frequent among carriers, these infections are not more severe or fatal than staphylococcal infections in non-carriers (12). Some studies have even suggested that mortality attributable to *S. aureus* infection may be lower among carriers (12,14). This has been attributed to the higher titers of neutralizing antibodies produced by persistent nasal *S. aureus* carriers.

During respiratory tract infections, the causative bacterium is expected to be recovered from the throat/nasopharynx for a period before clinical findings emerge, during the illness, and for days to weeks after the illness. These periods may be shorter or longer depending on the host's age, immune status, vaccination history, and antibiotic treatment received. Therefore, microbial growth immediately before, during, and shortly after infection/disease should not be confused with colonization or carriage.

In conclusion, maintenance of the normal nasopharyngeal/throat flora contributes positively to host protection against respiratory tract infections. Disruption of this flora by viral infection or inappropriate antibiotic use may increase the risk of unfavorable colonization and infection within the existing flora. With regard to the throat and nasopharyngeal flora—somewhat differently from the gastrointestinal tract—colonization and carriage are generally considered synonymous. This differs from gastrointestinal *Salmonella* carriage, whether typhoidal or non-typhoidal. Although the risk is low, throat/nasopharyngeal colonization by certain bacteria may pose a risk of infection to the host, and this risk is greatest during the first days after colonization is initially acquired. In practice, however, it is not possible to determine when colonization developed or how long it has persisted. In the absence of clinical findings, treatment is not indicated for throat/nasopharyngeal colonization/carriage.

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