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FACTORS INFLUENCING THE DURATION OF COUGH AFTER ACUTE RESPIRATORY TRACT INFECTIONS

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ABSTRACT: Cough is one of the most persistent symptoms after an acute respiratory tract infection (ARTI), and its duration may extend beyond the period of fever, rhinorrhea, and other acute manifestations. A cough lasting three to eight weeks after an otherwise resolving respiratory infection is commonly described as postinfectious cough. Although it is usually self-limited, its persistence is influenced by interacting biological, clinical, behavioral, and environmental factors. This article reviews the main determinants of prolonged postinfectious cough, with particular attention to airway inflammation, transient bronchial hyperresponsiveness, impaired mucociliary clearance, mucus production, cough-reflex hypersensitivity, the type and severity of the initial infection, previous episodes of prolonged cough, smoking and other airway irritants, asthma and upper-airway disease, gastroesophageal reflux, and selected infectious causes such as Bordetella pertussis. Evidence indicates that the intensity of acute symptoms alone is an imperfect predictor of cough persistence, whereas previous postinfectious cough and increased cough sensitivity may identify individuals at greater risk. Recognition of these factors is important for appropriate reassurance, avoidance of unnecessary antibiotics, and identification of patients who require evaluation for asthma, sinus disease, reflux, pertussis, pneumonia, or another cause of chronic cough.

KEYWORDS: acute respiratory tract infection; postinfectious cough; cough duration; airway inflammation; bronchial hyperresponsiveness; cough hypersensitivity; mucociliary clearance; risk factors

INTRODUCTION

Acute respiratory tract infections are among the most frequent causes of cough in primary care. In otherwise healthy adults, cough associated with an uncomplicated respiratory infection commonly resolves within several weeks, but the patient's perception of an abnormally prolonged cough can lead to repeated consultations and unnecessary antimicrobial treatment. A systematic review of observational studies and untreated or placebo groups found a mean cough duration of approximately 17.8 days, illustrating that persistence for two or three weeks can still fall within the expected natural history of an acute respiratory illness. When cough continues for more than three weeks but no longer than eight weeks after the acute infection has improved, postinfectious cough becomes an important clinical consideration.

The persistence of cough is not explained by a single mechanism. Viral injury to the respiratory epithelium can maintain local inflammation after the pathogen has been cleared. Edema, inflammatory mediators, mucus hypersecretion, and temporary changes in airway smooth-muscle responsiveness can increase the sensitivity of the cough reflex. Upper-airway inflammation may contribute through rhinitis or rhinosinusitis, while asthma and gastroesophageal reflux can prolong or amplify symptoms.

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Consequently, the duration of postinfectious cough reflects an interaction between the initial infection, host susceptibility, recovery of airway function, and continuing environmental or comorbid triggers.

Understanding these determinants has practical importance. A persistent cough after an uncomplicated viral infection does not automatically indicate treatment failure or a continuing bacterial infection. At the same time, persistence beyond the usual postinfectious period should not simply be attributed to the preceding infection. The purpose of this article is to synthesize the principal factors that influence cough duration after acute respiratory tract infections and to explain their clinical significance within an evidence-based framework.

MATERIALS AND METHODS

A focused narrative review was conducted using peer-reviewed clinical studies, systematic reviews, and evidence-based clinical guidance concerning acute cough, postinfectious cough, respiratory viral infection, airway hyperresponsiveness, and factors associated with prolonged cough. Literature indexed in PubMed and evidence published in the CHEST clinical guideline literature were considered. Priority was given to systematic reviews, prospective observational studies, and clinical practice guidance. The reviewed evidence was organized around four domains: characteristics of the initial infection, host and airway factors, comorbid conditions, and environmental or behavioral exposures. Because studies used different definitions and follow-up intervals, the findings were synthesized qualitatively rather than combined in a quantitative meta-analysis.

RESULTS

1. Natural history and definition of postinfectious cough

The duration of cough after respiratory infection follows a continuum rather than an abrupt transition. Acute cough is generally defined as lasting less than three weeks, subacute cough as three to eight weeks, and chronic cough as more than eight weeks. In the subacute interval, postinfectious cough is a frequent explanation when the acute illness has resolved and chest imaging does not indicate pneumonia or another structural disease. The average duration reported for acute cough is close to 18 days, but substantial individual variation exists. Therefore, a cough persisting for several weeks should not be judged solely by comparison with an assumed one-week recovery period.

2. Airway inflammation and epithelial injury

The most important biological determinant is persistence of airway inflammation after the acute infection. Respiratory viruses can damage epithelial cells and disrupt the normal barrier function of the airway. Even after viral replication has declined, inflammatory signaling, epithelial repair, and altered local sensory pathways may continue. Inflammation can produce mucosal edema and increase stimulation of cough receptors. This explains why relatively minor exposures, such as cold air, talking, dust, or a small quantity of mucus, may trigger coughing during the recovery period. The degree and duration of epithelial disturbance therefore influence how rapidly cough sensitivity returns to baseline.

3. Transient bronchial hyperresponsiveness

Viral respiratory infections can temporarily increase airway responsiveness in people with and without asthma. Inflammatory edema, epithelial damage, altered smooth-muscle behavior, and mediator release can narrow the airway and amplify responses to inhaled stimuli. When bronchial hyperresponsiveness persists after the acute infection, cough may be prolonged and may occur with exertion, cold air, or nocturnal exposure. This mechanism is particularly relevant when postinfectious cough resembles an asthma phenotype. Individuals with previously unrecognized or established asthma may experience a more persistent cough because the infection acts as a trigger rather than the sole cause.

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4. Mucus hypersecretion and impaired mucociliary clearance

Respiratory infection can increase mucus production while simultaneously impairing the coordinated movement of mucus by airway cilia. Thickened or excessive secretions stimulate cough receptors and create a continuing mechanical reason for cough after systemic symptoms have disappeared. Recovery therefore depends not only on resolution of inflammation but also on restoration of effective mucociliary transport. Dehydration, dry air, tobacco smoke, and other irritants may further increase secretion viscosity or impair clearance, potentially extending symptoms.

5. Cough-reflex hypersensitivity and previous prolonged cough

An important host factor is the sensitivity of the cough reflex. Prospective observations in patients with H1N1 influenza found that those who developed postinfectious cough had greater cough sensitivity and were more likely to report a previous episode of postinfectious cough. This suggests that some individuals have a reproducible susceptibility to prolonged cough after viral infection. In such patients, the original inflammatory stimulus may be modest, while heightened sensory responsiveness allows coughing to persist after the primary airway injury has substantially improved.

6. Type and severity of the initial infection

The causative pathogen can influence the respiratory inflammatory response and subsequent symptom profile. Influenza and other viruses capable of producing substantial lower-airway inflammation may be followed by persistent cough in a subset of patients. However, severity of the acute clinical syndrome is not a reliable standalone predictor of persistence. Studies of H1N1 influenza indicate that acute symptoms, spirometry, bronchial responsiveness, and sputum cellular findings did not clearly distinguish patients who later developed postinfectious cough from those who did not. Thus, the duration of cough depends on host response as much as on the apparent severity of the initial illness.

7. Upper-airway disease and postnasal inflammatory mechanisms

Persistent inflammation of the nose and paranasal sinuses may maintain cough through upper-airway sensory stimulation. Rhinitis, rhinosinusitis, nasal obstruction, and retained secretions can contribute to an upper-airway cough syndrome. This mechanism is clinically important because a patient may believe that the infection itself is continuing when the dominant postinfectious process has shifted from the lower respiratory tract to the upper airway. Allergic rhinitis may also become more clinically apparent during or after an infection and can prolong coughing independently of the original virus.

8. Asthma, gastroesophageal reflux, and other comorbidities

Underlying diseases can transform a self-limited postinfectious cough into a prolonged or recurrent symptom. Asthma may be unmasked or exacerbated by respiratory infection, especially when cough is accompanied by wheezing, chest tightness, or variable airflow limitation. Gastroesophageal reflux can provoke cough through esophageal-bronchial reflex pathways and may also become more symptomatic because repeated forceful coughing increases reflux pressure. These conditions should be considered when the cough is unusually persistent, recurrent, nocturnal, or associated with other characteristic symptoms. If cough exceeds eight weeks, alternative causes of chronic cough should be actively investigated rather than continuing to label the symptom as postinfectious.

9. Smoking and environmental irritants

Tobacco smoke, occupational dusts, aerosols, strong odors, air pollution, and very dry or cold air can repeatedly stimulate an already sensitized cough reflex. Smoking may additionally impair mucociliary clearance and sustain airway inflammation. Consequently, exposure after infection can function as a prolonging factor even when the initial viral illness has resolved. Reducing exposure

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during recovery is therefore biologically plausible and clinically reasonable, particularly in people who already have airway hyperresponsiveness or chronic respiratory disease.

10. Specific infectious causes and diagnostic implications

Not every prolonged cough following an apparently viral illness is a simple postinfectious cough. Bordetella pertussis can produce a particularly prolonged cough, sometimes with paroxysms, post-tussive vomiting, or an inspiratory whoop. Mycoplasma pneumoniae and Chlamydophila pneumoniae have also been identified among patients with prolonged cough, although they account for only a minority of cases in available studies. The presence of prolonged cough in these settings has implications for diagnosis, infection control, and treatment. Therefore, duration should be interpreted together with epidemiology, vaccination history, exposure history, systemic symptoms, and characteristic cough features.

DISCUSSION

The evidence indicates that postinfectious cough duration is best understood as the result of a dynamic interaction between airway recovery and individual cough susceptibility. The initial infection initiates epithelial injury and inflammation, but the persistence of symptoms depends on how quickly inflammatory activity, mucus production, mucociliary function, bronchial responsiveness, and sensory nerve sensitivity normalize. This explains why two patients with similar respiratory infections can have markedly different cough durations.

A clinically important observation is that the intensity of acute symptoms does not consistently predict later cough persistence. Instead, previous prolonged cough and increased cough sensitivity may identify a subgroup with heightened susceptibility. This finding supports a patient-centered approach in which the clinician assesses previous episodes, asthma symptoms, rhinitis, reflux, smoking, environmental exposure, and cough triggers rather than relying only on the apparent severity of the original infection.

The distinction between expected postinfectious recovery and a new or unrecognized disease is particularly important after the third week. Most uncomplicated cases are self-limited, and routine antibiotics are not justified simply because cough persists. Antibiotic therapy has no role in ordinary postinfectious cough unless a specific bacterial condition is identified or suspected, such as early pertussis or bacterial sinusitis. Evidence for several pharmacological approaches remains limited, and systematic reviews have not established a universally effective medication for postinfectious cough. Treatment should therefore be guided by the dominant mechanism and by exclusion of alternative diagnoses.

The practical implication is that duration itself is a clinical signal. A cough of three to eight weeks after an improving respiratory infection may represent postinfectious cough, whereas cough beyond eight weeks requires reconsideration of the diagnosis. Red flags such as hemoptysis, significant dyspnea, persistent fever, hypoxemia, weight loss, abnormal chest findings, or suspected pneumonia require earlier evaluation. Future research should use standardized definitions, prospective follow-up, objective measures of cough sensitivity and airway inflammation, and pathogen-specific analyses to clarify which patients are most likely to develop prolonged symptoms.

CONCLUSION

The duration of cough after acute respiratory tract infection is determined by multiple interacting factors rather than by the infectious episode alone. Persistent airway inflammation, epithelial injury, transient bronchial hyperresponsiveness, mucus hypersecretion, impaired mucociliary clearance, cough-reflex hypersensitivity, previous postinfectious cough, upper-airway disease, asthma,

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gastroesophageal reflux, smoking, environmental irritants, and selected pathogens can all prolong symptoms. Most cases resolve spontaneously within the subacute period, but cough lasting more than eight weeks or accompanied by warning signs should prompt evaluation for other causes. Recognizing the factors that prolong cough can improve clinical decision-making, reduce unnecessary antibiotic use, and provide patients with a more accurate expectation of recovery.

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