Incidence and Prevalence of Hyperhidrosis

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KEYWORDS

• Hyperhidrosis • Primary hyperhidrosis • Secondary hyperhidrosis • Prevalence • Diagnosis

KEY POINTS

- Hyperhidrosis is a skin disorder that causes excessive sweating and is known to significantly impair quality of life.
- Primary hyperhidrosis is most often focal, affecting the palms, soles, and axillae; thighs, gluteal, and inguinal regions may also be involved. A patient may have one affected area or multiple focal sites of hyperhidrosis occurring simultaneously.
- Approximately 2.8% of the US population is affected by hyperhidrosis.
- Secondary hyperhidrosis is usually caused by an underlying medical condition or drug.
- Secondary hyperhidrosis must be ruled out before a diagnosis of primary hyperhidrosis is made.

INTRODUCTION

Hyperhidrosis is a skin disorder characterized by sweating in excess of what is required for thermoregulation. Hyperhidrosis can be primary or secondary in nature and may have general, regional, or focal manifestations.[1] Primary hyperhidrosis is most often focal and generally causes idiopathic, symmetrically bilateral excessive sweating of the axillae, palms, soles, or craniofacial region.[2,3] Secondary hyperhidrosis manifests most often as generalized excessive sweating that is related to an underlying medical condition or use of medication. Hyperhidrosis results in a decrease in quality of life, may cause impairment in the ability to carry out daily functions, and in some cases may increase risk of cutaneous infection.[4,5] Previous reports indicate that this condition affects 7.8 million people in the United States but this number may be conservative, because hyperhidrosis is both underreported by patients and underdiagnosed by health care professionals.[2] Understanding the epidemiology of the disorder is critical for screening, diagnosis, and treatment.

PRIMARY HYPERHIDROSIS

Primary focal hyperhidrosis is excessive sweating in a specific region of the body that is not caused by other medical conditions or medications.[2] The cause of primary hyperhidrosis is not well understood, but is thought to be due to an overactivity of the autonomic nervous system. Eccrine sweat glands, which are located in the deep dermal layer of the skin, are innervated by post-ganglionic sympathetic nerve fibers and are stimulated by the neurotransmitter acetylcholine.[6] In primary hyperhidrosis, it is believed that these sweat glands receive aberrant stimulation by the sympathetic fibers causing excessive sweat production, although this has not been proved. Histologic evaluation of the affected areas demonstrates normal appearing eccrine sweat glands with a normal number, size, and density of the glands. Quantity

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and function of acetylcholinesterase is also known to be normal, indicating an overabundance of neurotransmitter is not the cause.[3,6–8]

Focal Sites

Common focal sites for primary hyperhidrosis include palms, soles, axillae, craniofacial area, inguinal area, and gluteal region. Palmar, plantar, and axillary hyperhidrosis are the most comment manifestations of the disease.[2] Patients with primary hyperhidrosis may have one or multiple sites of involvement. For example, a patient may have palmar hyperhidrosis alone, palmar and axillary hyperhidrosis, or various other combinations of focal involvement.[2,9] This form of hyperhidrosis should be distinguished from generalized hyperhidrosis, and diagnostic criteria for focal hyperhidrosis can be helpful in accomplishing this. Whatever the manifestation of primary hyperhidrosis, the sweating is not related to another condition, but is itself the medical problem.[9]

Hexsel's Hyperhidrosis

Hexsel's hyperhidrosis is a type of regional primary hyperhidrosis that is characterized by chronic excessive sweating typically found in the inguinal region, including the medial surfaces of the upper thighs, suprapubic area, external genitalia, and at times the gluteal folds and gluteal cleft.[10] Patients with this condition have difficulty concealing the often-embarrassing sweat-drenched clothing in this area that typically results from having the disorder. Prevalence is largely unknown due to underreporting, but the condition appears less frequently than other forms of focal hyperhidrosis. Fifty percent of patients with Hexsel's hyperhidrosis have a positive family history of some form of hyperhidrosis, suggesting an inherited mechanism.[10]

Localized Unilateral Hyperhidrosis

Localized unilateral hyperhidrosis is usually seen as a sharply demarcated region of sweating on the forearm or forehead restricted to less than 10 cm by 10 cm. Most cases are idiopathic with no known triggering factors. The pathogenesis is unclear,[11] and one case report suggests that there is a hypohidrotic element to the disorder.[12] Less than 40 cases have been reported in the literature.[13]

Diagnostic Criteria for Primary Hyperhidrosis

Criteria for diagnosing primary focal hyperhidrosis include focal, visible, and excessive sweating for greater than 6 months without apparent cause with 2 or more of the following criteria: sweating that is bilateral and relatively symmetric, impairment of daily activities, frequency of at least one episode per week, age of onset less than 25 years, positive family history, and cessation of focal sweating during sleep.[14,15] A more recent analysis found that increasing the required criteria from 3 of 7 elements to 4 of 7 elements increases specificity (82% vs 21%) and positive predictive value (99% vs 95%). Increasing the specificity and positive predictive value further helps practitioners accurately delineate primary from secondary hyperhidrosis.[1] Primary hyperhidrosis that is truly generalized is rare, and the diagnosis should only be made after causes of secondary sweating are excluded. **Table 1** summarizes the criteria for diagnosis of hyperhidrosis.

Hyperhidrosis Prevalence

A major study seeking to determine the prevalence of hyperhidrosis in the United States was conducted wherein 150,000 US households were sent a survey inquiring about excessive sweating. Results from the survey projected that 2.8% of the US population is affected by hyperhidrosis.[2] Women and men were affected equally. Of those individuals affected, 50.8% have hyperhidrosis of the axillae, a third of whom described this condition as barely tolerable or intolerable and always or frequently interfering with daily activities.[2] The study also revealed that only 38% of respondents had ever discussed excessive sweating with a health care professional, with women being more likely than men to have discussed the problem (47.5% vs 28.6%). These findings demonstrate that although hyperhidrosis quite negatively affects quality of life, patients may be uncomfortable asking about this topic.[2] Hyperhidrosis is potentially underdiagnosed and undertreated; making this diagnosis necessitates inquiry during a routine review of systems regarding sweating and how it affects the patient's quality of life.

Although Strutton and colleagues[2] found a discrepancy between men and women in the reporting of sweating, no difference in incidence was found between the genders. Two studies of populations abroad, however, found that men had a higher incidence of hyperhidrosis: 16.66% versus 10.66% in Japan[16] and 18.1% versus 13.3% in Germany.[17] A study of Polish students found that men reported a higher intensity of hyperhidrosis symptoms than did women,[18] but a contradictory study in Canadian patients found that women reported being more severely affected.[19] Men are more likely to complain of craniofacial hyperhidrosis and to have "additional areas" involved (ie, back, chest, abdomen, forearm, genital, and lower

Table 1
Criteria for diagnosis

Primary Hyperhidrosis	1. Focal, visible, and excessive sweating of at least 6-mo duration without apparent cause 2. At least 2 of the following: • Bilateral and relatively symmetric • Impairs daily activities • At least one episode per week • Age of onset <25 y • Family history of hyperhidrosis • Cessation of focal sweating during sleep 3. Exclusion of secondary causes of excessive sweating
Secondary Generalized Hyperhidrosis	1. Generalized excessive sweating attributable to a definitive underlying medical cause; most commonly drugs, substance abuse, cardiovascular disorders, respira tory failure, infections, malignancies, endocrine/metabolic disorders, or neuro logic disease.
Secondary Regional Hyperhidrosis	1. Localized anhidrosis with compensatory excessive sweating in other areas. 2. Identification of a definitive underlying cause; most commonly stroke, peripheral nerve damage, spinal cord lesion, neuropathy, or Ross syndrome.
Secondary Focal Hyperhidrosis	1. Excessive sweating in typical anatomic sites (palms, soles, axillae, craniofacial) or in a well-defined anatomic distribution (trunk, inguinal folds, buttocks, legs, submammary folds, neck, or wrist). 2. Identification of a definitive underlying cause; most commonly Frey syndrome, eccrine nevus, social anxiety disorder, neurologic disorder, or tumor.

Adapted from Walling HW. Clinical differentiation of primary from secondary hyperhidrosis. J Am Acad Dermatol 2011;64(4):693.

extremities).[19] Women are more likely to experience axillary hyperhidrosis.[4]

Hyperhidrosis Epidemiology

A retrospective chart review in 2011 found that 93% of patients with hyperhidrosis had primary disease, as opposed to secondary hyperhidrosis. More than 90% of patients with primary hyperhidrosis had a typical distribution, involving the axillae, palms, soles, and craniofacial areas.[1] Of these, the majority had an isolated axillary distribution (29%) or palms and soles distribution (25%); other patterns were isolated soles (15.5%), axillae with palms and soles (11%), palms (6%), and craniofacial (5%). Atypical distributions included the trunk (3%), inguinal folds (1.3%), buttocks, legs, submammary folds, neck, and wrist (<1% each).[1]

The onset of primary hyperhidrosis is most commonly between 14 and 25 years of age. Eccrine sweat glands are fully functional at birth, however, so hyperhidrosis is also seen in infants and young children. When the condition is seen in prepubertal individuals, it is generally the palmar or plantar variety that manifests (88.9%), with less likely presentations in the axillary (15.5%), facial (6.6%), or abdominal and dorsal (4.4%) regions.[20] A post-pubertal onset is more frequently associated with an axillary distribution.[19] The low prevalence of hyperhidrosis among the elderly is thought to possibly represent regression of the disease over time.

There is a positive family history in 35% to 56% of patients with hyperhidrosis; the pattern of inheritance is most likely autosomal dominant with variable penetrance.[2,4,19] Recently it has been reported that there may be a genetic linkage to chromosome 14.[9] Much like the overall prevalence of hyperhidrosis, the incidence of a positive family history is likely underestimated, because patients may conceal the presence of hyperhidrosis from family members due to embarrassment. In fact, one study found an even higher correlation with positive family history in patients with primary palmar hyperhidrosis, with 65% of patients having a positive family history.[21] Earlier age of onset (<20 years old) was also shown to correlate with positive family history, but these data may be confounded by the fact that patients with more severe cases present earlier.[19] **Box 1** summarizes these epidemiologic findings.

SECONDARY GENERALIZED HYPERHIDROSIS

Secondary generalized hyperhidrosis is excessive sweating that is caused by a medical condition or medication. Underlying conditions that may cause secondary hyperhidrosis can be physiologic, such as pregnancy, menopause, fever, excessive

Box 1
A summary of epidemiologic findings

Summary of Epidemiologic Findings in Primary Hyperhidrosis

- 2.8% prevalence of hyperhidrosis, with 93% primary versus secondary
- Over 90% with typical distribution: axillae, palms, soles, craniofacial
- Onset between 14 and 25 years, with prepubertal onset associated with palmoplantar disease and post-pubertal onset associated with axillary disease
- Men may have higher incidence and higher intensity
- Men are more likely to have craniofacial hyperhidrosis or "additional areas" involved, whereas women are more likely to have axillary hyperhidrosis
- Autosomal dominant with greater than 35% of patients having positive family history; earlier age of onset may be associated with positive family history

heat, or pathologic, including malignancy, carcinoid syndrome, hyperthyroidism, pheochromocytoma, tuberculosis, HIV, endocarditis, and autonomic dysreflexia, among others.[1,22,23] Drugs that are known to cause secondary hyperhidrosis include antidepressants, hypoglycemic agents, triptans, antipyretics, cholinergics, sympathomimetic agents, and many others. Secondary causes of hyperhidrosis must be ruled out before diagnosing primary hyperhidrosis.[2,14,22] This is most easily accomplished with a thorough review of systems and additional work-up as appropriate based on the patient response. Psychiatric disorders can also present with hyperhidrosis. Secondary hyperhidrosis is a clinical feature of 32% of people with social anxiety disorder.[24,25] Some debate exists, however, over whether the relationship between these 2 entities is causal. [26]

Clinical characteristics that help distinguish between primary and secondary types of hyperhidrosis include onset of the disease, characteristics of the sweating, and associated symptoms.[1] Patients with secondary hyperhidrosis are more likely to have onset older than 25 years compared with patients with primary hyperhidrosis. Although patients with primary hyperhidrosis are much more likely to have sweating in a typical distribution, those with secondary hyperhidrosis are significantly more likely to exhibit unilateral or asymmetrical sweating, to be generalized rather than focal, and to have symptoms during sleep ("night sweats"). Secondary hyperhidrosis is less often associated with positive family history.[1] A middle-aged patient presenting with new onset generalized or asymmetrical sweating that also occurs while sleeping is highly suspicious for secondary hyperhidrosis. **Table 2** compares the characteristics of primary versus secondary hyperhidrosis.

SECONDARY FOCAL HYPERHIDROSIS

Although cases are considered rare, multiple types of focal secondary hyperhidrosis exist. For example, gustatory sweating is a condition wherein facial sweating occurs related to the consumption of foods. Gustatory sweating can be classified as either physiologic or nonphysiologic.[27,28] A physiologic type of gustatory sweating occurs as bilateral facial sweating that may occur in hot climates or after consumption of hot or spicy foods. Nonphysiologic types of gustatory sweating are caused by auriculotemporal nerve syndrome, diabetic neuropathy, infection, or sympathetic nerve damage from neoplasm or sympathectomy.[28] Frey syndrome is a focal facial sweating that occurs secondary to aberrant regeneration of damaged parasympathetic fibers that are destroyed by a parotid or salivary tumor or by surgical resection of a tumor. Frey syndrome may occur in up to 60% of patients after parotidectomy with facial nerve dissection.[28,29] Auriculotemporal nerve syndrome can also occur sporadically as a familial trait and in these cases, occurs without a preceding trauma to the nerve. Diabetic gustatory sweating may occur as a byproduct of sympathetic denervation, which is compensated for by innervation of aberrant parasympathetic fibers. These fibers stem from the minor petrous nerve and innervate the parotid gland, causing sweating when salivation is induced. This finding is seen in 69% of patients with diabetic nephropathy and 36% of patients with diabetic

Table 2
Characteristics of primary versus secondary hyperhidrosis

Comparison of Primary and Secondary Hyperhidrosis	
Primary Hyperhidrosis	**Secondary Hyperhidrosis**
• Sweating in a typical distribution • Positive family history	• Onset older than 25 y • Unilateral and/or symmetric • Generalized rather than focal • Presents nocturnally or during sleep

Table 3
Causes of secondary hyperhidrosis

Common Conditions	Nonneural Conditions
Acute febrile illness (eg, infection)	Arteriovenous fistula
Alcoholism	Blue rubber bleb nevus syndrome
Diabetes mellitus	Cold erythema
Gout	Drugs
Heart failure	Glomus tumors
Hyperthyroidism	Klippel-Trenaunay syndrome
Lymphoma	Local heat
Menopause	Maffucci syndrome
Obesity	Organoid and sudoriparous nevi
Parkinson disease	
Pregnancy	
Rheumatoid arthritis	

Nervous System–Mediated Conditions: A,B,C, D

Hypothalamic conditions (mediated by the hypothalamus)(A)
- Carcinoid syndrome
- Cardiac shock
- Chédiak-Higashi syndrome
- Chronic arsenic intoxication
- Cold injury
- Debility
- Chronic infection (eg, tuberculosis, malaria, brucellosis)
- Drugs
- Familial dysautonomia
- Erythrocyanosis
- Essential hyperhidrosis
- Exercise
- Hines-Bannick syndrome
- Hyperpituitarism
- Hypoglycemia
- Hypothalamic mass
- Idiopathic unilateral circumscribed hyperhidrosis
- Infantile scurvy
- Pheochromocytoma
- POEMS syndrome
- Porphyria
- Post-encephalitis
- Raynaud phenomenon or disease
- Reflex sympathetic dystrophy
- Rickets
- Stroke/cerebrovascular accident/transient ischemic attack (affecting hypothalamus)
- Symmetric lividity of the palms and soles
- Vitiligo

Peripheral-reflexive conditions (B)
- Drugs/medications
- Perilesional (eg, burn)

Cortical conditions (mediated by the cerebral cortex) (C)
- Congenital autonomic dysfunction with universal pain loss
- Congenital ichthyosiform erythroderma
- Epidermolysis bullosa simplex
- Familial dysautonomia
- Gorlin syndrome
- Palmoplantar keratoderma
- Pachyonychia congenita
- Pressure and postural hyperhidrosis

Medullary/Spinal conditions (mediated by the medulla oblongata or spinal nerves) (D)
- Auriculotemporal syndrome
- Granulosis rubra nasi
- Physiologic gustatory sweating
- Post-traumatic (spinal cord transaction or thoracic sympathetic chain injury)
- Encephalitis
- Sytingomyelia

Courtesy of International Hyperhidrosis Society, Quakertown, PA; with permission.

Box 2
Drugs that cause hyperhidrosis

Pain Medications
- Celebrex
- Hydrocodone/Vicodin
- Toradol/Ketoralac
- Morphine
- Relafen/Nabumetone
- Naproxen/Aleve
- Oxycodone/Roxicodone
- Ultram/Tramadol
- Duragesic/Fentanyl
- Marinol

Heart/Blood Pressure
- Norvasc/Amlodipine
- Lotensin/Benazepril
- Bumex/Bumetanide
- Coreg/Carvedilol
- Digoxin/Lanoxin
- Persantine/Dipyridamole
- Cardura/Doxazosin
- Vasotec/Enalapril
- Hydralazine
- Prinivil/Zestril/Lisinopril
- Cozaar/Losartan
- Lopressor/Metoprolol
- Nifedipine/Procardia
- Rythmol/Propafenone
- Altace/Ramipril
- Calan/Verapamil

Oncology/Cancer
- Arimidex/Anastrozole
- Lupron/Leuprolide
- Tamoxifen/Nolvadex

Gastrointestinal
- Lomotil/Diphenoxylate
- Anzemet/Dolasetron
- Asacol/Mesalamine
- Prilosec/Omeprazole
- Aciphex/Rabeprazole

Head/Neck Medications
- Aerobid/Nasarel
- Claritin/Loratadine
- Sudafed/Pseudoephedrine
- Aristocort/Azmacort
- Afrin/Neo-Synephrine
- Zinc tablets/Cold-EEZE

Hormonal/Endocrine
- Calcitonin/Fortical
- Glucotrol/Glipizide
- Insulin/Humulin
- Synthroid/Thyroid
- Depo-Provera
- Prednisolone/Orapred
- Evista/Raloxifene
- Genotropin/Somatropin
- Testosterone/Androgel
- Antibodies/Tositumomab
- Vasopressin/Pitressin

Skin Medications
- Topical steroids
- Accutane/Isotretinoin
- Lidocaine/Carbocaine
- Selsun/Selenium sulfide

Blood/Immune System
- Neoral/Cyclosporine
- Ferrous gluconate/Iron
- Remicade/Infliximab
- Cellcept/Mycophenolate
- Prograf/Tacrolimus

Antibiotics/Antivirals
- Acyclovir/Zovirax
- Rocephin/Ceftriaxone
- Cipro/Ciprofloxacin
- Sustiva/Efavirenz
- Foscavir/Foscarnet
- Tequin/Gatifloxacin
- Avelox/Moxifloxacin
- Ketek/Telithromycin
- Ribavirin/Copegus
- Retrovir/AZT

Psychiatric/Neuro Medications
- Elavil/Amitriptyline
- Buspar/Buspirone
- Tegretol/Carbamazepine
- Celexa/Citalopram
- Clozaril/Clozapine
- Norpramin/Desipramine
- Adderall/Amphetamine
- Migranal/Ergotamine
- Aricept/Donepezil
- Cymbalta/Duloxetine
- Lexapro/Escitalopram
- Lunesta/Eszopiclone
- Prozac/Fluoxetine
- Haldol/Haloperidol
- Sinemet/Levodopa
- Provigil/Modafinil

Eye Medications
- Phospholine Iodide
- Vascon/Naphazoline
- Alcaine/Vardenafil

Lung Medications
- Advair/Fluticasone
- Combivent/Ipratropium
- Xopenex/Levalbuterol
- Alupent/Metaproterenol

Genital/Urinary
- Cialis/Tadalafil
- Levitra/Vardenafil

Adapted from International Hyperhidrosis Society, Quakertown, PA; with permission.

neuropathy.[27] Gustatory sweating may also occur after infection, most commonly secondary to herpes zoster infection.[23]

Secondary focal hyperhidrosis may be seen in conjunction with a variety of cutaneous disorders, although a causal relationship is not established. Although uncommon, with only 20 reports in the literature, an eccrine nevus can cause localized hyperhidrosis in an area of skin with increased numbers of eccrine glands.[30] Associated hypertrichosis and comedones can be seen in the area. Another term used in the literature to describe an eccrine nevus is nevus sudoriferous.[31,32] A similar lesion, the eccrine angiomatous hamartoma,[33] shows an abundance of eccrine glands but is also accompanied by a proliferation of vascular channels. Fewer than 50 cases have been reported. Because of similarities in histologic appearance and make-up, these lesions may share a similar genetic pathway. Pachyonychia congenita is a rare autosomal dominant genodermatosis that is often associated with focal palmar and plantar hyperhidrosis. There have only been 450 reported cases of this since 1901. One study found hyperhidrosis in 51.5% of all patients with pachyonychia congenita and in 22.7% of children with the disorder.[34] Other associated disorders include palmoplantar keratodermas, glomus tumor, blue rubber bleb nevus syndrome, nevus sudoriferous, POEMS (polyneuropathy, organomegaly, endocrinopathy, M protein, skin changes) syndrome, speckled lentiginous nevus syndrome, Riley-Day syndrome, pachydermoperiostosis, Gopalan syndrome, causalgia, pretibial myxedema, Buerger disease, eccrine pilar angiomatous hamartoma, local injury, and increased size of eccrine glands.[13] **Table 3** summarizes the causes of secondary hyperhidrosis. **Box 2** lists drugs that can cause hyperhidrosis.

SECONDARY REGIONAL HYPERHIDROSIS

Secondary regional hyperhidrosis is often characterized by anhidrosis in one area with compensatory hyperhidrosis in another area. Most commonly the condition is iatrogenic, in the form of compensatory sweating following surgical treatment of primary focal hyperhidrosis. It may also manifest as part of Ross syndrome or in one of several neurologic conditions.[7,35,36]

Compensatory hyperhidrosis, sweating in areas remote from the original problem location, is a known potential complication of endoscopic thoracic sympathectomy (ETS). In 2011, expert consensus by the Society of Thoracic Surgeons reported that 3% to 98% of patients having had ETS develop iatrogenic compensatory hyperhidrosis.[37] One large-scale study found only 55% of patients developed compensatory sweating, with 2% considering the compensatory hyperhidrosis to be as bothersome as the original symptoms.[38]

Ross syndrome is a rare nervous system disorder characterized by a tonic pupil ("Adie pupil"), deep tendon hyporeflexia, and unilateral or bilateral anhidrosis.[36] It can present with associated segmental hyperhidrosis. Recent studies suggest that Ross syndrome may be autoimmune in etiology.[35] The disorder is rare, with about 50 case reports in the literature.[39]

Secondary regional hyperhidrosis may be related to stroke, spinal cord lesion, neoplasm, or peripheral neuropathies.[40,41] One pathophysiologic explanation for this phenomenon is that the primary lesion causes impairment of preganglionic neurons and subsequent anhidrosis, but bladder distension and other visceral stimuli enter the spinal cord distal to the lesion, causing a spinal dysreflexia that manifests as abnormal sweating. The phenomenon has also been called "perilesionary hyperhidrosis" or "border-zone sweating".[40] One study evaluated 633 strokes and found hemihyperhidrosis in 6 patients, whereas another evaluated 350 strokes and found hemihyperhidrosis in 5 patients, so incidence of hyperhidrosis in cerebral infarction may be estimated at 1% to 2%.[42] Hyperhidrosis can also be associated with syringomyelia and other central nervous system diseases.[43]

SUMMARY

Understanding the epidemiology of hyperhidrosis can improve the diagnosis, treatment, and ideally the prognosis of the disorder. Because of the social implications of excessive sweating, hyperhidrosis is likely underreported and therefore undertreated. Providers who are knowledgeable about hyperhidrosis may be more likely to identify those who are suffering from the disorder and may be more comfortable beginning the delicate dialogue about how excessive sweating might be affecting the patient.

Although most patients suffer from primary hyperhidrosis, an accurate assessment must be made to rule out secondary causes of hyperhidrosis to tailor treatment appropriately. With a modest improvement in the recognition of hyperhidrosis, a provider has the opportunity to make a major impact on a patient's quality of life.

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