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# Occurrence rates of von Willebrand disease among people receiving care in specialized treatment centres in the United States

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## Abstract

**Introduction:** In the network of U.S. comprehensive haemophilia treatment centres (HTCs), von Willebrand disease (VWD) is the most common bleeding disorder other than haemophilia. Estimates of the size and characteristics of the VWD population receiving treatment are useful for healthcare planning.

**Aim:** Estimate the prevalence and incidence of VWD among males and females receiving care at U.S. HTCs (HTC-treated prevalence and incidence).

**Methods:** During the period 2012–2019, de-identified surveillance data were collected on all VWD patients who visited an HTC including year of birth, sex, race, Hispanic ethnicity, VWD type, and laboratory findings and used to calculate period HTC-treated prevalence by VWD type and sex. Data from patients born 1995–1999 were used to estimate HTC-treated incidence rates.

**Results:** During the period, 24,238 patients with a diagnosis of VWD attended HTCs; for 23,479 (96.9%), VWD type was reported or could be assigned. Age-adjusted HTC-treated prevalence was 8.6 cases/100,000 (7.2/100,000 for Type 1, 1.2/100,000 for Type 2 and 1.7/million for Type 3) and was twice as high in women as men (4.8 vs. 2.4 cases/100,000) for Type 1 and similar by sex for Type 2 and Type 3. HTC-treated Type 1 incidence increased over the period, averaging nearly threefold higher for women than men (26.2 vs. 9.9/100,000 live births). Sex differences were less for Type 2 (2.2 vs. 1.4 cases/100,000 births) and slight in Type 3.

**Conclusion:** Prevalence and incidence of HTC-treated VWD differ by sex and type and are likely strongly influenced by differences in rates of diagnosis.

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**Keywords**

incidence; prevalence; von Willebrand disease

## 1 | INTRODUCTION

Among hereditary bleeding disorders, von Willebrand disease (VWD) is considered the most frequent.[1] A study conducted in a three-state region of the U.S. among children of multi-racial and -ethnic origins during routine grade school examinations revealed an overall prevalence of 1.3%.[2] This was consistent with a previous estimate of 0.8% from Italian schoolchildren.[3] Prevalence based on groups seeking medical care, however, has been estimated at between 1:1000 in primary care[4] and 1:10,000 in tertiary care settings.[1] Estimates of the size and characteristics of the VWD population receiving treatment are useful for healthcare planning.

Among the three major diagnostic categories of VWD, Type 1 is the most commonly diagnosed followed by Type 2. Both are characterized by mucocutaneous bleeding. Type 3 VWD is the rarest and most severe form and causes severe mucocutaneous bleeding, as well as complications similar to those seen in moderate to severe haemophilia A, including hemarthrosis.[5,6] A study based on a survey of haematology departments in the United States, Canada and Europe found the incidence of 'severe' VWD in the United States to be 1.38 per million population.[7]

Estimates of the size and characteristics of the U.S. VWD populations are needed for health care planning and resource needs assessment. Since 1975, a system of comprehensive care clinics, termed haemophilia treatment centres (HTCs), has received federal support to provide diagnosis and care for people with bleeding disorders. In 1998, the Centers for Disease Control and Prevention (CDC) established surveillance in the U.S. HTC Network (USHTCN) to monitor treatment and outcomes of care for these populations. The most recent version of this surveillance system, called Community Counts, includes an HTC Population Profile component (HTC PP) that collects minimal data on all people with bleeding and clotting disorders who visit the USHTCN each year.[8] Not all people with VWD receive care in a network HTC, as the majority exhibit mild clinical course, and others may be treated by non-network haematologists. Nonetheless, the wide distribution of network HTCs in nearly every state across the country provides the opportunity to estimate occurrence rates of VWD among patients referred to the USHTCN for diagnosis and/or medical care.

## 2 | MATERIALS AND METHODS

The data reported were collected through collaboration of American Thrombosis and Hemostasis Network (ATHN), the Centers for Disease Control and Prevention (CDC) and the Hemophilia Treatment Center Network (HTCN) using ATHN Study Manager as part of the Community Counts surveillance program. In January 2012, HTC staff began collecting HTC PP data on all people with bleeding disorders who visited an HTC for diagnosis or care using standardized forms.[8] Demographic and clinical data were de-identified using a unique

identification code known only to HTC staff prior to transmission to CDC. Data elements pertinent to this study include year of birth, sex, race, Hispanic ethnicity, 3-digit zip code of residence, VWD type and subtype and lowest VWF activity level measured along with concurrent VWF antigen and factor VIII levels.

We used data from all individuals with a VWD diagnosis who visited any of the 139 USHTCN centres located in the continental United States and Hawaii during the period 2012–2019. Patients with Type 2 VWD subtypes A, B, M and N as well as those with Type 2 of unknown subtype were included in the category Type 2. Patients with Type reported as other or unknown and having VWF and factor VIII levels were assigned a VWD Type using a published algorithm.[9] Patients with VWF antigen, VWF activity or factor VIII level below 50 units/decilitre were classified as Type 3 if VWF antigen was undetectable, Type 2 if VWF activity/VWF antigen or factor VIII/VWF antigen ratio was ≤0.6, or Type 1 if both ratios were >0.6.

HTC-treated VWD prevalence was calculated by dividing the number of individuals with VWD who received diagnosis or care in the USHTCN during the period 2012–2019 by the average U.S. population over the same time period[10] and expressed as cases per 100,000 (100 K). Subject age was calculated by subtracting the year of birth from the year of the most recent clinic visit during the surveillance period. Age-adjusted prevalence was calculated by direct standardization to the age distribution of the U.S. population in 2000.[11] Prevalence by race and ethnicity was calculated using census estimates in 2015 as an approximation for the average general population during the surveillance period. State-specific prevalence was calculated by determining the number of subjects living in each state based on their 3-digit zip code of residence and dividing by the state populations from the U.S. Census Bureau.[12]

Using surveillance data on year of birth, HTC-treated VWD incidence rates for 1995–1999 were calculated by dividing the number of VWD births by the number of national births in that year using the National Center for Health Statistics natality tables.[13] These birth years were used so that the youngest individuals would be at least 13 years old during the surveillance period since the diagnosis of VWD is often delayed, especially in women, until after bleeding symptoms occur and, in some cases, later due to diagnostic difficulties. Annual incidence rates, both sex-specific and total, were calculated for each of the 5 years and expressed as the number of infants with VWD born per 100 K live infants born in that year. In addition, rates were calculated separately by VWD type due to known variations in incidence between types.

## 3 | RESULTS

A total of 24,238 patients with a VWD diagnosis received care in an HTC during the period 2012 through 2019. The VWD type was reported for 22,093 (91%) cases. Of the remaining cases, the type could be assigned for an additional 1386 cases based upon laboratory results provided by HTC staff. Therefore, complete VWD data were available for a total of 23,479 (96.9%) unique patients with a VWD diagnosis during the period and formed the analysis dataset.

The characteristics of the study population are shown in Table 1. As expected, most of the subjects had Type 1, and Type 3 was the least common form of VWD. Nearly two-thirds (65%) were women. Compared to the general U.S. population, the proportions of Blacks (7.3% vs. 13.4%), Asian (2.6% vs. 5.9%) and patients of Hispanic ethnicity (16% vs. 18.5%) were lower in the HTC-treated VWD population.

The age distribution of the VWD patients is compared with that of the U.S. population separately by sex in Figure 1. The age distributions of the populations of men and women with VWD seen in the HTCs were both different than that of the general population and varied by sex with the male prevalence peaking at ages 5–14 years while the female prevalence peaked at ages 15–24 years.

The HTC-treated prevalence estimates of VWD by sex and type are shown in Table 2. The age-adjusted HTC-treated prevalence of Type 1 VWD was twice as high in women than men (4.8 vs. 2.4 cases per 100 K, respectively). In contrast, the HTC-treated prevalence of Type 2 and Type 3 VWD was similar in men and women and much lower than that of Type 1 VWD. The overall HTC-treated prevalence of Type 2 was 1.2 cases per 100 K and that of Type 3 was 2 cases per million (Table 2). Considering all types and both sexes, the overall age-adjusted HTC-treated prevalence of treated VWD was 8.5 cases per 100 K.

There were differences by sex across the VWD types in patterns of age-specific HTC-treated prevalence as shown in Figure 2A-C. The prevalence of Type 1 VWD in males peaked in the age group 5–9 years and was somewhat greater than that among females prior to age 5 years and then declined after age 19 years (Figure 2A). The HTC-treated prevalence peaked for females among those aged 15–19 years then declined but remained higher at every age group than males. The overall HTC-treated prevalence of Type 1 VWD peaked at 25 cases per 100 K for subjects in the age group 15–19 years. The same-sex pattern was seen for Type 2 VWD; however, the difference in prevalence between the sexes was not nearly as great with an overall peak prevalence of 2.6 cases per 100 K (Figure 2B). The age-specific HTC-treated prevalence pattern was similar for males and females in Type 3 VWD and was relatively consistent from ages 5–34 years (Figure 2C).

The state-specific HTC-treated prevalence varied from 0.7 cases per 100 K in Delaware to 22.7 cases per 100 K in Connecticut (Figure 3). Except for a somewhat higher prevalence in the upper Midwest, there does not appear to be a very strong regional pattern suggestive of founder or other effects.

Of the 23,479 study subjects, 3890 (17.0%) were born during the years 1995–1999 and formed the incident cohort. These subjects ranged in age from 13 to 24 years with an average age of 18.8 (median = 19) years. Compared to the full study group, a larger proportion of subjects in the incident cohort had Type 1 VWD and was female while the racial and ethnic composition of the two groups was similar (Table 1).

HTC-treated VWD incidence rates based on the incident cohort by year of birth, sex and VWD type are shown in Table 3. The numbers of subjects with Type 1 VWD increased in each of the 5 years studied resulting in increasing HTC-treated incidence over the period. The average HTC-treated incidence over the period was nearly threefold higher for women

than men (26.2 vs. 9.9 per 100 K live births). The sex difference was far less for Type 2 VWD with an incidence of 2.2 vs. 1.4 cases per 100 K births for women and men, respectively. There was no appreciable sex difference in Type 3 with an overall HTC-treated incidence of 2.4 per million live births.

## 4 | DISCUSSION

Estimates of prevalence and incidence of VWD will be influenced by two key characteristics of the disorder: its genetic heterogeneity and its diagnostic ambiguity.[14-16] Unlike many rarer coagulopathies, VWD is most often seen in the heterozygous form as an autosomal dominant disorder, due to the presence of a single disease allele and a non-pathogenic allele; the disease exhibits not only reduced penetrance, meaning that not all heterozygotes show its effects, but also variable expressivity, since those affected within the same family may show varying degrees of symptoms.[17] Patients have intermediate levels of VWF often associated with blood type O[18,19]; these levels may overlap with the ranges seen in unaffected individuals and may vary over time with age-related normalization in approximately a third of patients over a period of 5–20 years.[20-23] Some individuals who transmit the gene to offspring show no effects of it or have laboratory features but no symptoms.[17] Difficulties in diagnosing heterozygous VWD have been well documented over many years,[14-16] and somewhat arbitrary diagnostic schemes with variable diagnostic thresholds have been developed. Changes in these diagnostic schemes over time have influenced which patients were given a diagnosis of VWD and how they were classified for clinical purposes.[24] The classification scheme used in this study was first published in 1994 by the International Society on Thrombosis and Haemostasis (ISTH)[14] and updated in 2006[15]; it includes three major categories: partial quantitative deficiency of VWF (Type 1), qualitative abnormality of VWF (Type 2) and total deficiency of VWF (Type 3). Type 1 is the most common form and the most difficult to diagnose. Type 2 VWD is more easily identified, because most subtypes have qualitative abnormalities of VWF that can be detected by specialized tests. Type 3 is the homozygous form of the disease and the rarest, requiring two abnormal alleles; it is easy to diagnose, because it results in almost complete absence of VWF, although it can be misdiagnosed as haemophilia A due to its low levels of factor VIII, if complete testing is not done.[25] The ISTH classification scheme does not include the category of ‘low VWF’, which has been proposed for levels of VWF activity and antigen between 30 and 50 units/decilitre.[16] Choices for diagnosis on study forms followed ISTH criteria and did not include ‘low VWF’ as an option.

Genetic testing cannot be used for assessing prevalence of VWD, because only 50–70% of patients with Type 1 VWD have a defect in the structural gene for VWF[26,27] with the likelihood of a gene defect increasing with decreasing VWF level. The cause for a significant proportion of Type 1 VWD cases has not been identified and may have only an indirect effect on the measurements used for diagnosis. A portion of these may represent the chance concurrence of blood group O and bleeding symptoms and might be more properly classified as ‘low VWF’ rather than VWD if newer classification schemes were used.[16] Previous prevalence studies have assessed the frequencies of laboratory abnormalities in VWF in unselected populations, such as school children, based on laboratory findings and bleeding symptoms. These studies have resulted in the high-frequency figures of 1% of the

population having VWD.[2,3] Follow-up to one of these studies, however, found that fewer than one-half of the subjects identified as having VWD had subsequent bleeding episodes over a 13-year period.[28] The authors concluded that clinically relevant disease was less common than 1% and suggested that focus be placed on identifying those with significant bleeding. The current study is limited in that it assessed only patients seen for diagnosis or care in the USHTCN. Because there are no figures available on the proportion of VWD patients seen outside the HTCs, prevalence and incidence could be calculated only for HTC-treated VWD patients, unlike haemophilia, for which the numbers could be extrapolated to the entire U.S. population.[29] Our estimate of the overall HTC-treated prevalence of VWD of 8.6 per 100,000 falls within the range of 2.3–11.3 per 100,000 cited from a variety of studies of treated patients conducted worldwide.[1] It is not surprising that numbers of patients attending tertiary care centres are lower than those found in population-based studies, due to the proportion of VWD patients with minimal symptoms[1] and the late age of diagnosis, which often does not occur until adulthood and after multiple tests.[30] From these data, it is not possible to determine whether the wide discrepancy between population and treatment frequencies represents individuals not receiving appropriate treatment, those receiving care outside specialized centres, or those not needing care for VWD. A further limitation of this study is the use of diagnoses reported by the HTCs, which were not independently verified either by centralized testing or analysis of comprehensive diagnostic laboratory data. The limited data available represented lowest levels recorded at the reporting HTC and may not have been those used for diagnosis, particularly for older patients; thus, they were not analysed except when a diagnosis was reported as ‘other’ or ‘unknown’. Patients who might be classified as ‘low VWF’ may have been included in these categories, which made up 5.9% of the study group, or in the VWD Type 1 category, depending on the classification scheme used at the time of diagnosis.

The finding of a higher proportion of females in the HTC-treated population of Type 1 VWD is consistent with those of smaller studies.[4,20] Prevalence in a treatment population is dependent on both frequency of the trait and rate of diagnosis. Age at diagnosis determines age of appearance in the treatment population and may be highly variable for less severe bleeding disorders. Age at diagnosis for haemophilia, which requires treatment early in life, is reported to be 36 months for mild, 8 months for moderate and 1 month for severe cases.[31] In contrast, the most common form of VWD, Type 1, is often not diagnosed until adulthood. Analysis of Community Counts Registry data from 2712 children ages 2–12 showed that mean age at diagnosis for males and females were 4.2 and 5.0 years for Type 1 VWD, 2.6 and 3.3 years for Type 2 VWD, and 0.8 and 1.5 years for Type 3 VWD.[32] In studies with broader age ranges, however, males with Type 1 VWD were reported to have a mean age at diagnosis of 9.6 years[33] and females a median age at diagnosis of 16.0 years.[34] These sex differences are reflected in the age distributions seen in the current study, with male HTC-treated prevalence peaking at 5–14 and female at 15–24 years. The treatment prevalence of Type 1 VWD was twice as high in females as in males. For Type 2 VWD, the ratio was 1.4, and for Type 3, there was equivalence. The equal numbers of males and females seen in the more severe Type 3 group suggest that the sex differences seen in Type 1 are not genetically based but depend on diagnostic rates and perhaps inclusion of patients with ‘low VWF’. Low VWF cohorts described in previous reports have included

predominately women and manifest bleeding phenotypes similar to those of patients with Type 1 VWD.[20,35] However, this designation has not been recognized as a separate category by the ISTH.[15]

The apparent increasing HTC-treated incidence of VWD is most likely due to the increased availability and quality of diagnostic testing, as well as increased incorporation of milder bleeding disorders and women into the U.S. HTC system.[36] This increase mirrors the succession of committee opinions from 2001–2013 by the American College of Obstetricians and Gynecologists advising VWD testing in women with heavy menses. It is also likely that there is no true difference in population incidence between males and females. The difference seen is more likely due to the additional burden of obstetric and gynaecologic symptoms among women leading to increased diagnostic testing.[37] Neither increases in rates over time nor differences by sex were seen in Type 3 VWD, which usually becomes apparent at an early age due to its severe symptoms.

The HTC-treated incidence for Type 3 VWD of 2.4 per million observed in this study is lower than the 3.1 per million reported in 1982.[7] That study, however, specified only ‘severe’ VWD, without assessing levels. The earlier study also occurred before the contamination of FVIII treatment products with human immunodeficiency virus (HIV). The lower prevalence of Type 3 patients in the 35–44 and 45–54 age groups could be due to a disproportionate loss of Type 3 patients to HIV infection, who were more likely to be treated with FVIII treatment products due to their decreased FVIII levels and hemarthroses.

For both Type 1 and Type 2 VWD, HTC-treated prevalence decreased above age 35 years. This finding may be the result of poor diagnostic methods in the past or to the reported changes in patient levels of VWF with ageing. Longitudinal studies [21-23] have found that 25–58% of patients reach normal VWF levels in adulthood and would not meet diagnostic criteria if they had presented later in life. However, this age-related increase in VWF was not accompanied by decreased bleeding.[21] Either explanation could result in the absence of a proportion of adults from the treatment population.

## 5 | CONCLUSIONS

The HTC-treated prevalence and incidence of VWD differ by sex and type and are likely strongly influenced by differences in rates of diagnosis and referral. The female preponderance reflects the likelihood of women being more symptomatic then men from the additional haemostatic challenges of menstruation and childbirth and is a call for further study and care at the public health level for women presenting with heavy menses and/or post-partum haemorrhage.

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CHM and ABP designed the study, conducted the research, analysed the data and wrote the paper. VRB, KA, RFS and PAK conducted the research and wrote the paper.

## DATA AVAILABILITY STATEMENT

The Community Counts surveillance data are available via a data visualization tool that displays de-identified data on patients with bleeding disorders who are enrolled in Community Counts in an interactive, visual format (https://www.cdc.gov/ncbddd/hemophilia/communitycounts/data-viz.html). However, due to ethical restrictions related to protecting patient confidentiality, additional individual-level data generated from the Community Counts surveillance system cannot be made publicly available.

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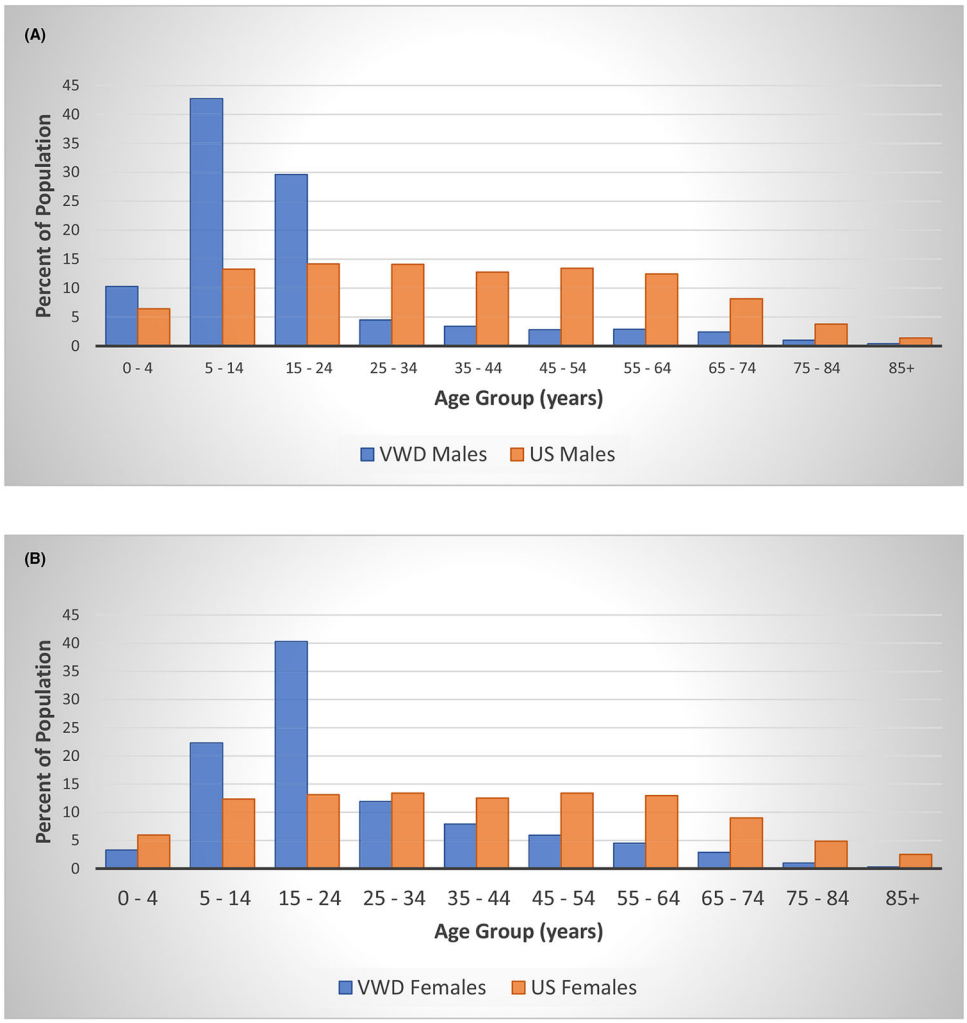


**FIGURE 1.**

Age distributions of males (A) and females (B) with von Willebrand disease (VWD) receiving care in the U.S. Hemophilia Treatment Centers Network compared to those of the general US populations

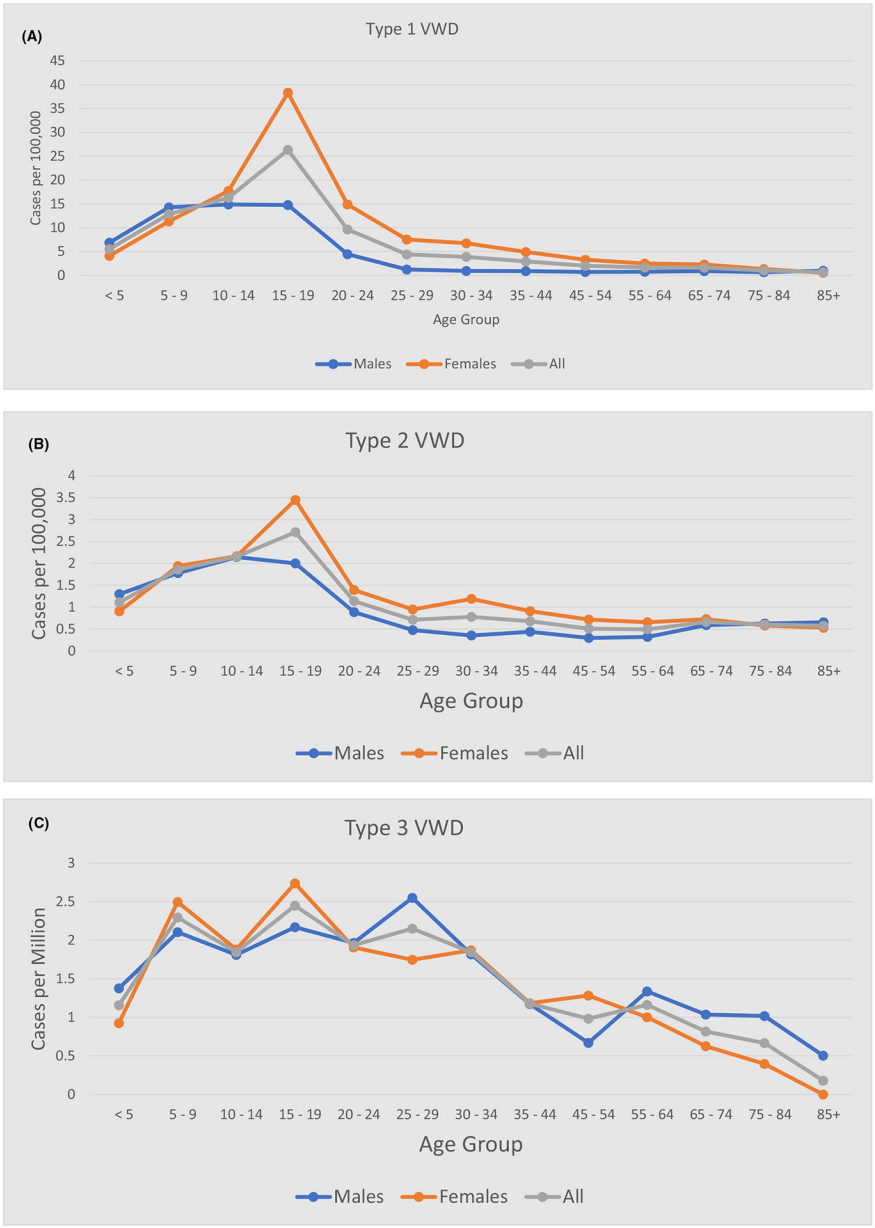


**FIGURE 2.**

Age-specific prevalence by sex for subjects with Type 1 (A), Type 2 (B) and Type 3 (C) von Willebrand disease (VWD) receiving care in the U.S. Hemophilia Treatment Centers Network

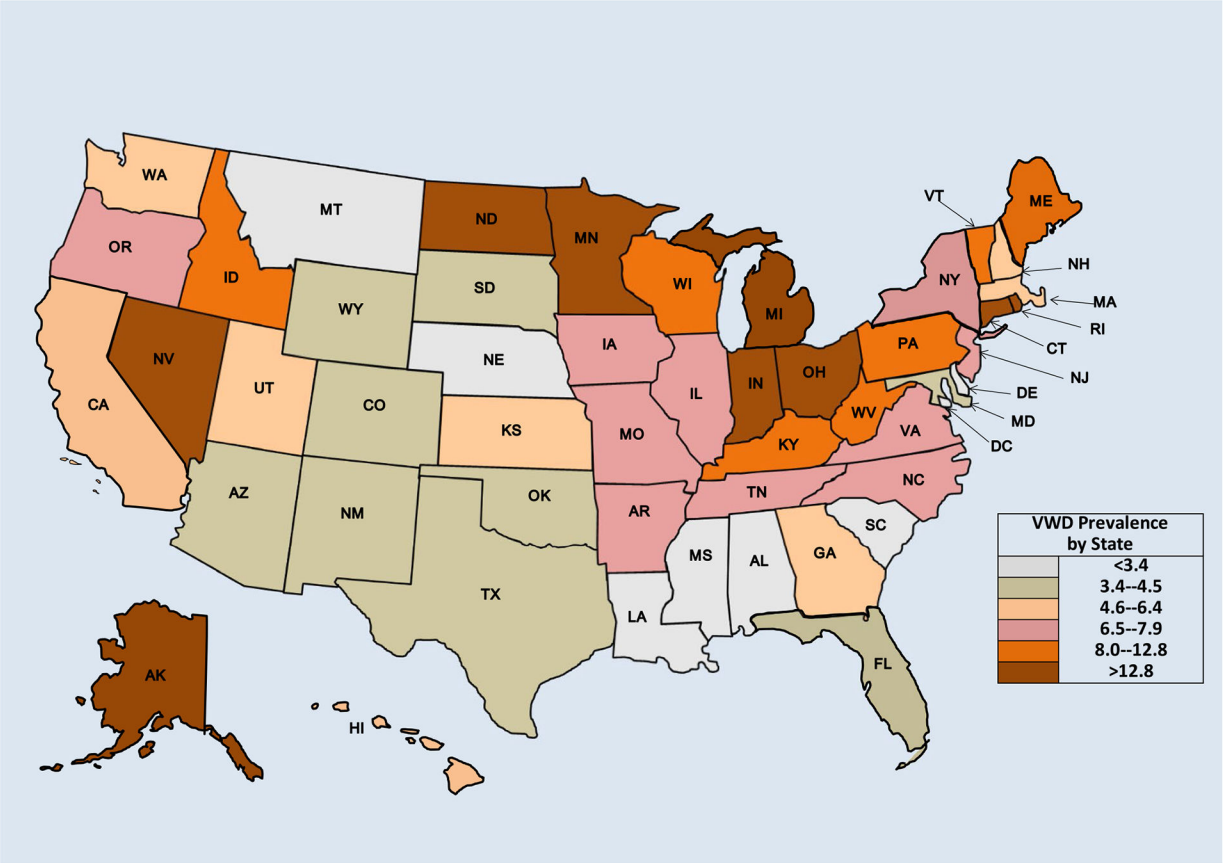


**FIGURE 3.**

Map of state-specific prevalence (cases per 100,000) of HTC-treated von Willebrand disease (VWD) in the United States

## TABLE 1

Characteristics of 23,434 subjects with von Willebrand disease (VWD) receiving care in the U.S. Hemophilia Treatment Centers Network, 2012–2019

| | All | | Born 1995–1999 | |
|---|---|---|---|---|
| **Characteristic** | **N** | **%** | **N** | **%** |
| VWD type | | | | |
| Type 1 | 19,845 | 84.5 | 3497 | 89.9 |
| Type 2 | 3178 | 13.5 | 347 | 8.9 |
| Type 3 | 456 | 1.9 | 46 | 1.2 |
| Sex | | | | |
| Female | 15,245 | 65 | 2736 | 70.3 |
| Male | 8234 | 35 | 1154 | 29.7 |
| Age (years) | | | | |
| Under 1 | 55 | 0.2 | – | |
| 1–5 | 1857 | 7.9 | – | |
| 6–11 | 3830 | 16.3 | – | |
| 12–19 | 8635 | 36.8 | 2350 | 60.4 |
| 20–29 | 3611 | 15.4 | 1540 | 39.6 |
| 30–39 | 1845 | 7.9 | – | |
| 40–49 | 1255 | 5.4 | – | |
| 50–59 | 1021 | 4.4 | – | |
| 60–69 | 800 | 3.4 | – | |
| 70+ | 570 | 2.4 | – | |
| Race | | | | |
| White | 19,616 | 83.6 | 3233 | 83.1 |
| Black | 1712 | 7.3 | 354 | 9.1 |
| Asian | 605 | 2.6 | 106 | 2.7 |
| Other | 447 | 1.9 | 79 | 2.0 |
| Unknown | 1099 | 4.7 | 118 | 3.0 |
| Ethnicity | | | | |
| Non-hispanic | 19,732 | 84.0 | 3263 | 83.9 |
| Hispanic | 3747 | 16.0 | 627 | 16.1 |

**TABLE 2**

Period HTC-treated prevalence of von Willebrand disease (VWD) by sex and VWD type, 2012–2019

| | Sex | Total N | Crude prevalence | Age-adjusted prevalence |
|---|---|---|---|---|
| Type 1 | Males | 6717 | 4.2 | 2.4 |
| | Females | 13,128 | 8.0 | 4.8 |
| | Both | 19,845 | 6.2 | 7.2 |
| Type 2 | Males | 1287 | 0.8 | 0.5 |
| | Females | 1891 | 1.2 | 0.7 |
| | Both | 3178 | 1.0 | 1.2 |
| Type 3 | Males | 230 | 0.1 | 0.1 |
| | Females | 226 | 0.1 | 0.1 |
| | Both | 456 | 0.1 | 0.2 |
| All Types | Males | 8234 | 5.2 | 3.0 |
| | Females | 15,245 | 9.3 | 5.6 |
| | Both | 23,479 | 7.3 | 8.5 |

Prevalence is cases per 100,000 population.

**TABLE 3**

HTC-treated incidence rates by sex and year of birth based on 3890 subjects with von Willebrand disease born 1995–1999

| **Males** | **Number of male subjects** | | | **NCHS data** | **Male Incidence (per 100 K live male births)** | | | **Males average 1995–1999** | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Year of birth** | **Type 1** | **Type 2** | **Type 3** | **# US male births** | **Type 1** | **Type 2** | **Type 3** | **Type 1** | **Type 2** | **Type 3** |
| 1995 | 141 | 22 | 5 | 1,996,355 | 7.1 | 1.1 | 0.25 | 9.9 | 1.4 | 0.21 |
| 1996 | 169 | 21 | 4 | 1,990,480 | 8.5 | 1.1 | 0.20 | | | |
| 1997 | 179 | 34 | 4 | 1,985,596 | 9.0 | 1.7 | 0.20 | | | |
| 1998 | 244 | 38 | 7 | 2,016,205 | 12.1 | 1.9 | 0.35 | | | |
| 1999 | 261 | 24 | 1 | 2,026,854 | 12.9 | 1.2 | 0.05 | | | |
| **Females** | **Number of female subjects** | | | **NCHS data** | **Female incidence (per 100 K live female births)** | | | **Females average 1995–1999** | | |
| **Year of birth** | **Type 1** | **Type 2** | **Type 3** | **# US female births** | **Type 1** | **Type 2** | **Type 3** | **Type 1** | **Type 2** | **Type 3** |
| 1995 | 397 | 33 | 3 | 1,903,234 | 20.9 | 1.7 | 0.16 | 26.2 | 2.2 | 0.26 |
| 1996 | 455 | 36 | 5 | 1,901,014 | 23.9 | 1.9 | 0.26 | | | |
| 1997 | 512 | 49 | 6 | 1,895,298 | 27.0 | 2.6 | 0.32 | | | |
| 1998 | 531 | 49 | 3 | 1,925,348 | 27.6 | 2.5 | 0.16 | | | |
| 1999 | 608 | 41 | 8 | 1,932,563 | 31.5 | 2.1 | 0.41 | | | |
| **All** | **Total number of subjects** | | | **NCHS data** | **Incidence (per 100 K live births)** | | | **Average 1995–1999** | | |
| **Year of birth** | **Type 1** | **Type 2** | **Type 3** | **# US births** | **Type 1** | **Type 2** | **Type 3** | **Type 1** | **Type 2** | **Type 3** |
| 1995 | 538 | 55 | 8 | 3,899,589 | 13.8 | 1.4 | 0.21 | 17.8 | 1.8 | 0.24 |
| 1996 | 624 | 57 | 9 | 3,891,494 | 16.0 | 1.5 | 0.23 | | | |
| 1997 | 691 | 83 | 10 | 3,880,894 | 17.8 | 2.1 | 0.26 | | | |
| 1998 | 775 | 87 | 10 | 3,941,553 | 19.7 | 2.2 | 0.25 | | | |
| 1999 | 869 | 65 | 9 | 3,959,417 | 21.9 | 1.6 | 0.23 | | | |