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RESEARCH ARTICLE

# Serum 25-Hydroxyvitamin D Concentration Spectrum in Patients with Sickle Cell Disease: A Cross-Sectional Descriptive Study

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## ABSTRACT

**Background:** Low serum 25-hydroxyvitamin D [25(OH)D] concentrations are frequently reported in sickle cell disease (SCD), but prevalence estimates alone can obscure the depth and shape of the concentration distribution.

**Objective:** To characterize the serum 25(OH)D concentration spectrum in patients with SCD using vitamin D measurements alone.

**Materials and Methods:** A cross-sectional analysis was performed on 54 vitamin D measurements available in an institutional research database. Only the vitamin D variable was analyzed. Descriptive statistics and conventional concentration strata were used.

**Results:** Mean 25(OH)D was 15.98 ± 8.62 ng/mL and median concentration was 14.40 ng/mL (range, 4.5–32.0 ng/mL). Thirty-four measurements (63.0%) were below 20 ng/mL, including 17 (31.5%) below 10 ng/mL. Sixteen (29.6%) were 20–29.9 ng/mL and only four (7.4%) were ≥30 ng/mL.

**Conclusion:** The dataset demonstrates a pronounced lower-end shift in serum 25(OH)D concentrations. Reporting the complete concentration spectrum may be more informative than a binary deficient/non-deficient label. Longitudinal multicenter studies are needed to determine whether the depth and persistence of low 25(OH)D concentrations have clinical significance.

**Keywords:** Sickle cell disease; Vitamin D; 25-hydroxyvitamin D; Vitamin D deficiency; Hypovitaminosis D.

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## Introduction

Sickle cell disease (SCD) is an inherited hemoglobin disorder characterized by chronic hemolysis, recurrent vaso-occlusion and cumulative organ injury. Nutritional and skeletal abnormalities are also recognized components of the disease burden. Vitamin D has attracted particular interest because of its role in calcium homeostasis and bone mineralization and because low vitamin D concentrations are repeatedly reported in people living with SCD.

Serum 25-hydroxyvitamin D [25(OH)D] is the principal circulating marker used to assess vitamin D status. Published SCD studies have used different thresholds for deficiency and insufficiency, contributing to variation in reported prevalence. A systematic review by Nolan et al. identified 15 eligible studies and reported that, when deficiency was defined as <20 ng/mL, prevalence in SCD populations ranged from 56.4% to 96.4%.[1] Goodman et al. found that 98% of 142 adults with SCD had concentrations below 30 ng/mL and 60% were below 10 ng/mL.[2] In children with HbSS disease, Rovner et al. reported a median 25(OH)D concentration of 15 ng/mL.[3]

These observations suggest that vitamin D abnormality in SCD may be better viewed as a concentration-spectrum phenomenon than as a simple binary state. A prevalence percentage does not distinguish a value just below a threshold from a profoundly low concentration. The depth of deficiency may be relevant when designing supplementation studies and when describing the nutritional phenotype of SCD.

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Interpretation of vitamin D thresholds also requires caution. The 2024 Endocrine Society guideline updated the previous guideline and emphasized that universally applicable serum 25(OH)D thresholds linked to prevention of disease outcomes have not been established for the general population without established indications for testing or treatment.[4] Threshold-based categories remain common in observational SCD research and in Indian clinical guidance. A recent Indian expert consensus defines deficiency as <20 ng/mL, severe deficiency as <10 ng/mL and insufficiency as 20–30 ng/mL.[5] In the present study these categories are used descriptively rather than as universal treatment targets.

The present work was deliberately restricted to the vitamin D variable. No hemoglobin, reticulocyte count, hemolysis marker, inflammatory marker, coagulation parameter, symptom variable or other numerical variable from the master dataset was incorporated. This avoids multiple exploratory comparisons and allows the analysis to answer one focused question: how is the vitamin D concentration distributed within this SCD dataset?

The objective was to describe the distribution of serum 25(OH)D concentrations, quantify the proportion of observations within commonly used concentration strata and highlight the extent of lower-end concentration burden.

An additional reason to examine the concentration spectrum is that vitamin D status is inherently continuous. A threshold-based analysis is useful for communication, but it can conceal whether a cohort is concentrated just below a cutoff or substantially below it. In a chronic disorder such as SCD, this distinction may be relevant when interpreting nutritional burden and when deciding how future studies should define eligibility for intervention. The present analysis consequently reports both central tendency and categorical distribution.

The study also deliberately avoids a common limitation of retrospective observational analyses: the temptation to search a large clinical dataset for statistically significant relationships simply because many variables are available. Such analyses can generate apparently interesting findings that are difficult to reproduce and may reflect multiple-testing effects. By restricting the present investigation to a single prespecified biochemical variable, the study has a narrower question but a clearer evidentiary boundary. Every principal numerical result can be traced directly to the vitamin D measurements themselves.

This design also makes the manuscript suitable as a baseline descriptive study. The findings can be used to inform sample-size calculations, identify the expected proportion of markedly low concentrations and provide a reference distribution for future longitudinal work. A future study could then prospectively examine seasonal variation, supplementation response, bone health or patient-reported outcomes without conflating those questions with the present descriptive objective.

The study should therefore be viewed as a characterization of a biochemical phenotype rather than an etiological investigation. Its purpose is not to explain why the concentrations are low, but to document how the values are distributed and how much of the observed cohort occupies the lower end of the spectrum.

## Materials and Methods

Study design and data source. This was a cross-sectional observational analysis of serum vitamin D measurements contained in an institutional research master dataset of patients with SCD.

Eligibility. Records with a numerical vitamin D value were included. Fifty-four observations contained usable numerical measurements and were analyzed. No patient identifier or other master-chart variable was used for the present analysis.

Analyte and unit. The sole analytical variable was serum 25-hydroxyvitamin D [25(OH)D]. The standard unit for serum 25(OH)D is ng/mL; therefore, all values in this manuscript are expressed as ng/mL rather than reproducing the incorrect unit label present in the spreadsheet. The original laboratory report or assay documentation should be retained and checked before submission to ensure concordance.

Descriptive categories. For comparison with prior SCD literature, concentrations were grouped as severe deficiency (<10 ng/mL), deficiency (10–19.9 ng/mL), insufficiency (20–29.9 ng/mL) and sufficiency (≥30 ng/mL).[1,5] These categories are descriptive and are not presented as universal therapeutic targets.

Statistical analysis. Mean, standard deviation, median, minimum, maximum and quartiles were calculated. The number and percentage of observations in each concentration category were determined. No correlation, regression, subgroup comparison or multivariable analysis was performed because the study question was restricted to vitamin D values alone.

Ethics. The final manuscript must include the exact Institutional Ethics Committee name and approval/reference number from the parent thesis or institutional study. The ethics statement should be harmonized with the original approval and should specify whether secondary analysis of the dataset was covered. Patient confidentiality was maintained.

Data handling and quality control. Vitamin D values were transcribed from the source research dataset without incorporating any of the other numerical fields. The numerical values were reviewed for presence and plausibility before descriptive analysis. No value was imputed, rounded to a threshold, or otherwise altered for statistical purposes. The unit was standardized in the manuscript to ng/mL because this is the conventional unit for serum 25(OH)D. This editorial unit correction does not change any numerical observation.

Handling of repeated low values. Repeated observations of the same numerical concentration were retained because the analysis was intended to describe the available dataset rather than remove values on the basis of their frequency. In particular, repeated 4.5-ng/mL observations were not collapsed into a single value. However, because the source laboratory reporting characteristics were not available for this secondary analysis, the possibility of a lower reporting limit is acknowledged.

Choice of summary measures. Mean and standard deviation were reported because they describe the arithmetic center and dispersion of the observed measurements. Median and quartiles were also reported because vitamin D distributions may be skewed and because the lower tail is of particular interest in this dataset. Minimum and maximum values were retained to demonstrate the observed concentration range.

Reporting percentages. Percentages were calculated using the 54 available vitamin D measurements as the denominator. The manuscript reports both absolute counts and percentages so that the reader can assess the underlying sample size. No inferential P values were generated because there was no prespecified comparison group and the study question was descriptive.

Reproducibility. The final study archive should retain the original master dataset, the laboratory documentation for the vitamin D assay, the ethics approval and the analysis worksheet. These records will permit verification of the unit, assay method and individual observations if requested during peer review.

## Results

Overall vitamin D distribution. Fifty-four serum 25(OH)D measurements were available. Mean concentration was 15.98 ± 8.62 ng/mL and median concentration was 14.40 ng/mL. The range was 4.5–32.0 ng/mL. The first and third quartiles were 9.40 and 23.79 ng/mL, respectively.

Distribution across concentration strata. Seventeen measurements (31.5%) were below 10 ng/mL. Another 17 (31.5%) were between 10 and 19.9 ng/mL. Thus, 34 of 54 observations (63.0%) were below 20 ng/mL. Sixteen observations (29.6%) were 20–29.9 ng/mL, while only four (7.4%) were ≥30 ng/mL. Overall, 50 of 54 observations (92.6%) were below 30 ng/mL.

Lower-end pattern. Several observations occurred at the lower end of the recorded range, including repeated measurements of 4.5 ng/mL. Because original laboratory reporting records were not available for this secondary analysis, these repeated values should not be interpreted as proven biological clustering; they may reflect genuine values or a reporting floor.

Quartile distribution and concentration burden. The interquartile interval extended from 9.40 to 23.79 ng/mL. Consequently, half of the observations occupied a concentration interval that began below 10 ng/mL and ended below 24 ng/mL. This provides a useful complement to the category-based analysis because it demonstrates that the lower concentration pattern was not produced solely by a small number of extreme observations.

The difference between the mean and median was modest. The mean was 15.98 ng/mL, whereas the median was 14.40 ng/mL. This relationship is compatible with a distribution containing a smaller number of higher observations, including values above 30 ng/mL, while most observations remained at lower concentrations.

Threshold-based burden. The lower-end burden can also be expressed cumulatively. One-third of the observations were below 10 ng/mL. Nearly two-thirds were below 20 ng/mL, and more than nine-tenths were below 30 ng/mL. Only four observations crossed the 30-ng/mL threshold. These cumulative descriptions show the concentration gradient more clearly than a single deficiency percentage.

The individual observations were therefore important to retain in the analysis. The dataset did not consist predominantly of values clustered immediately around one

**Table 1:** Descriptive statistics of serum 25(OH)D concentrations

| *Parameter* | *25(OH)D (ng/mL)* |
|---|---|
| Number of measurements | 54 |
| Mean ± SD | 15.98 ± 8.62 |
| Median | 14.40 |
| Minimum–maximum | 4.5–32.0 |
| 25th percentile | 9.40 |
| 75th percentile | 23.79 |

**Table 2:** Distribution of serum 25(OH)D measurements by concentration category

| *Category* | *Concentration* | *n* | *%* |
|---|---|---|---|
| Severe deficiency | <10 ng/mL | 17 | 31.5 |
| Deficiency | 10–19.9 ng/mL | 17 | 31.5 |
| Insufficiency | 20–29.9 ng/mL | 16 | 29.6 |
| Sufficiency | ≥30 ng/mL | 4 | 7.4 |
| Total | — | 54 | 100.0 |

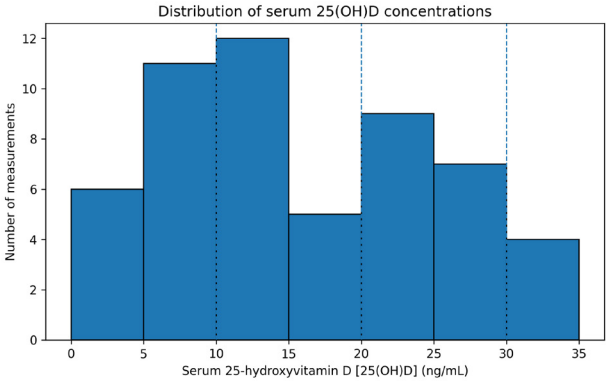


**Figure 1:** Distribution of serum 25(OH)D concentrations. Dashed lines indicate 10, 20 and 30 ng/mL

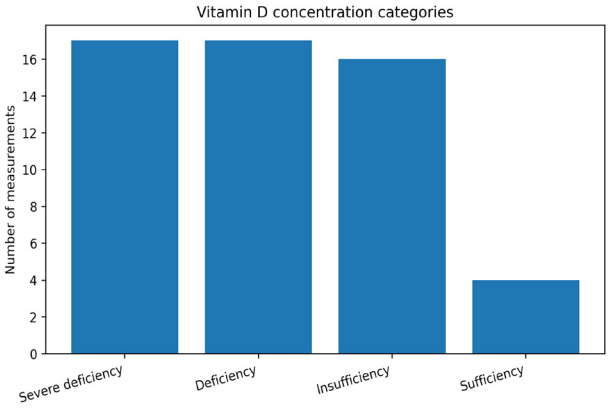


**Figure 2:** Distribution of vitamin D measurements across descriptive concentration categories

threshold. Instead, observations were distributed throughout the low range, with a substantial number below 10 ng/mL and another substantial group between 10 and 20 ng/mL.

## Discussion

The principal finding of this vitamin D-only analysis is a pronounced lower-end distribution of serum 25(OH)D concentrations. The median concentration was 14.4 ng/mL, nearly two-thirds of measurements were below 20 ng/mL, and fewer than one in ten reached 30 ng/mL. The pattern therefore extends beyond isolated borderline deficiency and indicates that low concentrations dominate the observed distribution.

The findings are broadly consistent with previous SCD literature. Nolan et al. reported a 56.4–96.4% prevalence of deficiency when <20 ng/mL was used across published SCD cohorts, while emphasizing heterogeneity in definitions.[1] The 63.0% proportion below 20 ng/mL in the present dataset lies within that published range. Goodman et al. reported 98% below 30 ng/mL and 60% below 10 ng/mL in 142 adults with SCD.[2] A cohort of 56 adults reported a median concentration of 6 ng/mL and 75% below 10 ng/mL.[6] Thus, although the magnitude differs between cohorts, a predominance of low 25(OH)D concentrations is a recurrent observation.

The present study adds emphasis on the concentration distribution itself. Categorical prevalence compresses continuous information: two patients classified as deficient may have very different concentrations. Reporting the median, quartiles, range and lower-tail burden alongside categories therefore provides a fuller description of vitamin D status.

This distributional approach may also be useful for future intervention research. A Cochrane review found that vitamin D supplementation in SCD increases serum 25(OH)D but concluded that available randomized evidence was insufficient to guide clinical practice.[7] More recent pediatric research has continued to evaluate high-dose supplementation and has reported improvement in vitamin D levels alongside selected skeletal or functional outcomes, while emphasizing the need for further trials.[8] Baseline characterization of the depth of deficiency can help future studies define inclusion criteria and stratify participants.

The biological basis of low vitamin D in SCD is likely multifactorial. Published work has considered nutritional intake, sunlight exposure, bone metabolism and other disease-related determinants. Han et al. evaluated risk factors for vitamin D deficiency in SCD and confirmed that low 25(OH)D is common.[9] The present dataset does not contain the variables required to test these mechanisms, so no causal explanation is proposed.

Vitamin D is relevant to skeletal health in SCD. Adewoye et al. reported correction of vitamin D levels and improvement in bone mineral density after vitamin D2 and calcium treatment in a small adult study.[10] Pediatric studies have also described low bone mineral density and

vitamin D abnormalities.[3,11] These observations provide context but do not establish bone disease in the present dataset because bone measurements were not analyzed.

Vitamin D has also been investigated in relation to pain and vaso-occlusive complications. A pediatric cross-sectional study found an association between deficiency and pain, although observational data cannot establish causality. [12] The current manuscript deliberately does not reproduce such associations because only vitamin D values were included in the requested analysis. This restraint reduces the risk of multiple-testing bias and unsupported conclusions.

Threshold terminology requires care. Traditional research definitions commonly use <20 ng/mL for deficiency, <10 ng/mL for severe deficiency and 20–30 ng/mL for insufficiency.[1,5] The 2024 Endocrine Society guideline cautions against treating universal serum 25(OH) D thresholds as disease-prevention targets in the general population.[4] Accordingly, the thresholds in this paper are used only for descriptive comparison with earlier literature and Indian practice guidance.

The source spreadsheet labels the vitamin D variable as "VIT-D (ng/dl)." Serum 25(OH)D is conventionally reported in ng/mL, and the values are compatible with the ranges used in published 25(OH)D studies. The manuscript therefore uses ng/mL. This should be verified against the original laboratory report before submission so that the laboratory record, thesis and manuscript are completely concordant.

The study has several limitations. The sample size is modest and the dataset is not a population-based sample. The analysis is cross-sectional and cannot describe longitudinal change. There is no non-SCD control group, so the study cannot quantify excess vitamin D deficiency attributable specifically to SCD. Information on season, diet, supplementation, sunlight exposure, age, sex, genotype, renal function and other potential determinants was intentionally excluded. If more than one observation came from the same individual, this should be clarified before any future inferential analysis. Finally, repeated values at 4.5 ng/mL may indicate a lower reporting limit; confirmation from laboratory records would improve interpretation.

Despite these limitations, the study provides a clear descriptive finding: low vitamin D concentrations are the dominant feature of the observed SCD vitamin D spectrum. Future multicenter studies should examine longitudinal vitamin D trajectories, standardized assay methods and whether the depth and persistence of deficiency have independent clinical significance.

The magnitude of the lower-end distribution deserves emphasis because it changes the way the finding should be communicated. If the result were presented only as "63% deficient," the reader would know the proportion below 20 ng/mL but would not appreciate that 31.5% of all observations were below 10 ng/mL. Conversely, describing only the proportion below 30 ng/mL would obscure the difference between moderate and marked depletion. Reporting the three concentration zones together therefore preserves clinically relevant information without claiming that one threshold has universal biological validity.

The present results can also be compared cautiously with the broader SCD literature. The systematic review by Nolan and colleagues demonstrated that prevalence estimates vary substantially according to the threshold and population studied.[1] This variability is important when comparing individual studies. A lower prevalence in one cohort does not necessarily indicate a genuinely higher vitamin D status unless the assay, season, population characteristics and threshold definitions are comparable. By reporting the raw distribution and multiple conventional strata, the current manuscript provides more information for future comparisons.

The lower concentration range observed in this dataset is also compatible with reports from both adult and pediatric SCD populations. Goodman et al. described a very high proportion of adults below 30 ng/mL and a large severely deficient subgroup.[2] Rovner et al. observed a median concentration of 15 ng/mL in children with HbSS disease. [3] A separate adult cohort reported an even lower median concentration of 6 ng/mL.[6] These studies differ in age, geography, clinical setting and methodology, so they should not be treated as directly interchangeable. Nevertheless, the repeated observation of low central vitamin D concentrations across different cohorts strengthens the rationale for continued investigation.

There is an important distinction between demonstrating low concentrations and demonstrating clinical benefit from correcting them. The former is supported by the present dataset; the latter is not. Supplementation studies have produced heterogeneous findings, and the Cochrane evidence review judged the available randomized evidence insufficient to guide clinical practice.[7] A later pediatric trial reported favorable changes in vitamin D levels and selected outcomes after supplementation, but such intervention data do not establish that every patient with a low baseline concentration will experience a meaningful clinical benefit.[8] A related trial similarly linked vitamin D supplementation to improved

health-related quality of life and physical performance in children with SCD.[13] The present study therefore avoids recommending a dose, duration or target concentration.

The same principle applies to skeletal outcomes. SCD is associated with altered bone health, and vitamin D is biologically relevant to calcium and bone metabolism. Adewoye et al. reported improved 25(OH)D levels and bone mineral density after vitamin D2 and calcium treatment in a small adult study.[10] Other pediatric studies have documented osteopenia and vitamin D abnormalities.[11] These observations support the clinical interest in vitamin D but do not allow the current dataset to infer skeletal consequences. The absence of bone measurements in the present analysis is intentional rather than an omission.

Another consideration is that vitamin D concentration can vary with season and other environmental or behavioral factors. Published SCD cohorts have differed in latitude, ethnicity, age distribution, nutritional practices and healthcare access. Even within one geographic region, sunlight exposure and supplementation can change across the year. Because the present analysis does not contain these variables, it cannot determine whether the observed distribution is stable over time. A longitudinal study would be particularly valuable because repeated measurements in the same participants could separate persistent deficiency from transient low values.

Assay methodology is another source of variation. Different laboratory platforms and calibration procedures can produce systematic differences in 25(OH)D measurements. For that reason, future multicenter research should record the assay platform and, where possible, use standardized laboratory methods. The current manuscript cannot reconstruct the assay characteristics from the spreadsheet alone. Verification from the original laboratory records is therefore an important pre-submission quality-control step.

The unit correction in this manuscript is also methodological rather than cosmetic. The source spreadsheet contains the label "ng/dl," whereas serum 25(OH)D is conventionally reported in ng/mL. A unit error in a manuscript can create major interpretive problems even when the underlying numerical values are correct. The present paper therefore uses ng/mL throughout. Before submission, the author should verify the laboratory report and assay documentation. If the laboratory report uses nmol/L, the numerical values would require conversion rather than simple relabeling. The conversion factor is 1 ng/mL = 2.5 nmol/L.[5]

The Indian setting gives the findings additional practical relevance. Vitamin D deficiency is common in the general Indian population, and contemporary Indian guidance continues to use concentration-based categories for clinical assessment.[5,14] The present study does not attempt to determine whether the observed burden is greater than that in the local non-SCD population because no control group is available. A future matched study could address that question directly and would be more informative than making assumptions based on published populations from other regions.

The absence of a control group should therefore be considered a limitation rather than a reason to overstate the findings. The appropriate conclusion is that low vitamin D concentrations were common within this SCD dataset. It is not appropriate to conclude from these data alone that SCD caused the low concentrations, that the prevalence is higher than in the surrounding population, or that vitamin D deficiency explains any particular clinical manifestation.

The use of only vitamin D values also limits the possibility of confounding analysis, but it protects the study from a different problem: selective reporting of statistically significant relationships. Because the source master chart contains multiple clinical and laboratory variables, a broad exploratory analysis could generate numerous associations by chance. The present analysis intentionally does not perform those tests. This is particularly important for a small dataset of 54 observations, where extensive multivariable modeling would be unstable and potentially misleading.

From a publication perspective, the novelty should therefore be stated carefully. The novelty is not the discovery that vitamin D deficiency occurs in SCD; that is already well documented. The contribution is the focused characterization of the concentration spectrum in this specific SCD dataset, including central tendency, quartile spread, severe-deficiency burden and cumulative threshold distribution. This distinction makes the manuscript more scientifically honest and reduces the risk of an editor rejecting it because the central research question has already been extensively answered.

The study also provides a practical framework for future work. Researchers could use the present distribution to design a prospective study in which baseline 25(OH)D is measured using a standardized assay, repeated at predefined intervals and related prospectively to prespecified outcomes. Such a study could distinguish persistent deficiency from seasonal variation and could determine whether the magnitude of baseline deficiency predicts response to supplementation. A multicenter design would additionally allow assessment of geographic and laboratory variation.
Finally, the study illustrates why raw concentration data

should be preserved whenever possible. Once continuous measurements are converted only into "deficient" and "non-deficient" labels, information about the lower tail, median and spread is lost. Maintaining the original numerical values allows investigators to examine alternative thresholds as clinical guidance evolves. This is particularly relevant because contemporary vitamin D recommendations have moved away from assuming that a single universal serum threshold can define benefit for every population.[4]

Taken together, the findings support a restrained but useful conclusion. In this dataset, low vitamin D was not a peripheral feature; it characterized most observations, with a substantial subgroup at very low concentrations. The appropriate next step is not to infer causality from the cross-sectional data but to determine whether the observed concentration pattern is reproducible and whether its depth or persistence carries clinically meaningful information.

## Conclusion

This vitamin D-only analysis of 54 serum 25(OH)D measurements from patients with SCD demonstrated a marked shift toward low concentrations. The median was 14.4 ng/mL, 63.0% of measurements were below 20 ng/mL, 31.5% were below 10 ng/mL and only 7.4% reached ≥30 ng/mL. The findings indicate a broad lower-end concentration burden rather than a small group with borderline deficiency.

Reporting the full concentration spectrum alongside conventional categories provides a more informative description of vitamin D status. Larger longitudinal and multicenter studies with verified assay methodology are needed to determine whether the depth and persistence of low 25(OH)D concentrations have independent clinical significance.

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## Data Availability

The underlying dataset is not publicly available because it contains research information subject to institutional confidentiality requirements. De-identified data may be considered subject to appropriate institutional approval.

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