

Disparities in the diagnostic process of Duchenne and Becker muscular dystrophy

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Purpose: To determine whether sociodemographic factors are associated with delays at specific steps in the diagnostic process of Duchenne and Becker muscular dystrophy. **Methods:** We examined abstracted medical records for 540 males from population-based surveillance sites in Arizona, Colorado, Georgia, Iowa, and western New York. We used linear regressions to model the association of three sociodemographic characteristics with age at initial medical evaluation, first creatine kinase measurement, and earliest DNA analysis while controlling for changes in the diagnostic process over time. The analytical dataset included 375 males with information on family history of Duchenne and Becker muscular dystrophy, neighborhood poverty levels, and race/ethnicity. **Results:** Black and Hispanic race/ethnicity predicted older ages at initial evaluation, creatine kinase measurement, and DNA testing ($P < 0.05$). A positive family history of Duchenne and Becker muscular dystrophy predicted younger ages at initial evaluation, creatine kinase measurement and DNA testing ($P < 0.001$). Higher neighborhood poverty was associated with earlier ages of evaluation ($P < 0.05$). **Conclusions:** Racial and ethnic disparities in the diagnostic process for Duchenne and Becker muscular dystrophy are evident even after adjustment for family history of Duchenne and Becker muscular dystrophy and changes in the diagnostic process over time. Black and Hispanic children are initially evaluated at older ages than white children, and the gap widens at later steps in the diagnostic process. *Genet Med* 2011;13(11):942–947.

Key Words: Duchenne and Becker muscular dystrophy, disparities, diagnosis

Inequalities in healthcare access, treatment, and clinical outcomes are common between various segments of the US population.¹ Health disparities originate in the variation among populations for race and ethnicity, social and economic status, insurance provider, geographic location, age, and language usage.² Studies indicate that these factors act both alone and in concert to influence the severity of health disparities.³ Although some health

disparities have been documented in the pediatric population,^{4,5} the literature is sparse,⁶ and no studies have examined disparities in the diagnostic process of relatively rare conditions such as Duchenne and Becker muscular dystrophy (DBMD).

DBMD are allelic, X-linked recessive, neuromuscular disorders with prevalence in males aged 5–24 years between 1.3 and 1.8 per 10,000.⁷ Spontaneous mutations in the dystrophin gene are reported in approximately 30% of cases.⁸ The clinical course of Duchenne muscular dystrophy is characterized by delayed development, progressive disability, and loss of ambulation by age 13 years.⁹ Becker muscular dystrophy is associated with a milder clinical course, but in practice, there is an overlap of clinical manifestations.¹⁰

In the absence of a family history of the disease, symptoms of DBMD are usually recognized approximately 2.5 years before diagnosis and 1 year before the child is brought to a healthcare professional for evaluation of those symptoms.¹¹ Clinic and population-based studies indicate a mean age of diagnosis of DBMD approximately 5 years, and this age has remained unchanged for 2 decades.^{11–13} Diagnostic testing for DBMD should be initiated, irrespective of family history, when characteristic physical signs and symptoms or abnormal laboratory findings are identified. Clinical symptoms include muscle weakness, delayed walking, and difficulty with running and climbing stairs, which should prompt creatine kinase (CK) measurement and subsequent referral to a neuromuscular specialist and genetic testing for definitive diagnosis.¹⁴

Clinical management of DBMD has improved over the past 20 years.¹⁴ Treatments that prolong ambulation, increase quality of life, and extend life expectancy are now available, but their efficacy may depend on a timely diagnosis.¹⁴ Studies of the diagnosis of other diseases in children and adults show disparities exist in at least two related diagnostic steps that are relevant to the experience of children as they undergo the DBMD diagnostic process, including (1) disparities at the family and physician level in noting symptoms and taking action as a result of symptoms^{15–18} and (2) disparities in access to and uptake of diagnostic and genetic testing.^{19–21} Disparities at each of these diagnostic steps result in a longer diagnostic timeline, which may have negative psychological effects on patients and their families^{21–23} and may unjustly exclude certain groups of patients from mutation-specific clinical trials and the highest standard of care.¹¹ The purpose of this study, therefore, is to determine whether sociodemographic factors are associated with disparities at steps in the diagnostic process of DBMD. A better understanding of the factors that place children at risk for diagnostic delay can inform efforts to improve early diagnosis for all patients.

MATERIALS AND METHODS

Data collection

The Muscular Dystrophy Surveillance, Tracking, and Research Network (MD STARnet) is a group of five US sites

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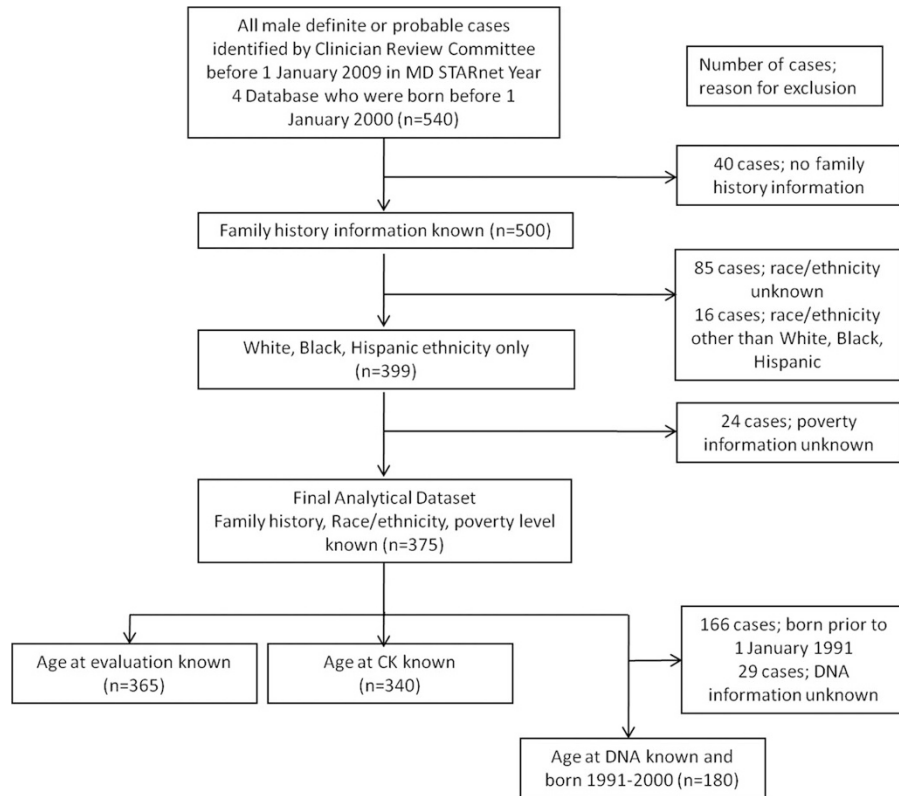


Fig. 1. Analytic dataset selection criteria.

(Arizona, Colorado, Georgia, Iowa, and western New York), which identifies and collects information on all cases of DBMD born or residing in those areas between 1982 and the present. The MD STARnet gathers information on cases from medical records, birth certificates, and interviews with caregivers and patients with DBMD. Using a computerized abstraction instrument, trained abstractors review and collect data from medical records for each case annually from multiple locations. Record sources include neuromuscular clinics, hospitals and hospital discharge databases, private physicians, service units for children with special health-care needs, and birth defects surveillance systems. Following record abstraction, the MD STARnet Clinical Review Committee conducts a blind review of selected diagnostic variables and classifies each patient to one of the following case definitions: definite, probable, possible, asymptomatic, female, or not DBMD. A full characterization of the population-based MD STARnet surveillance methodology is published elsewhere.²⁴

Data abstracted from medical records that we analyzed include family history of DBMD, date of first medical evaluation for symptoms associated with DBMD, initial CK measurement, and DNA analysis for dystrophin mutations. Information gathered from birth certificates that we analyzed included date of birth, education level of the mother, race/ethnicity of the child, and home address at birth.

Mother’s education information was missing from birth certificates of 29 cases of the final dataset. For these cases, we used mother’s education information collected as part of a telephone survey that targeted families of all cases known to the MD STARnet.

Sample

We compiled the analytic dataset by examining data from all 540 males included in the MD STARnet year 4 database who were

born before January 1, 2001, and identified by the Clinical Review Committee as definite or probable cases before January 1, 2009. Both definite and probable cases have a clinical diagnosis of DBMD from a qualified professional, characteristic signs and symptoms of DBMD, and an elevated CK level. The only difference between the classifications is that probable cases do not have an identified dystrophin gene mutation or diagnostic muscle biopsy. Figure 1 presents our selection criteria for the final dataset containing 375 (69.4%) of those cases. We excluded 40 cases missing family history information, 85 cases missing race/ethnicity information, and 16 cases that were identified with race/ethnicity other than white, black, or Hispanic. Twenty-four cases missing poverty information were also excluded.

Study variables

We designated three time points in the diagnostic pathway as continuous outcome variables: (1) age at initial evaluation for symptoms associated with DBMD, (2) age at first CK measurement, and (3) age at first DNA test. We created three dichotomous individual level sociodemographic variables: maternal education, classified as “high school graduate or less” versus “some college or above”; race and ethnicity, limited to either non-Hispanic white or non-Hispanic Black and Hispanic; and family history, designated as negative if records indicated that a patient’s caregivers had no knowledge of family members affected with DBMD before the diagnosis of the index child.

We used one area-based socioeconomic measure, census tract poverty level, to investigate the effect of neighborhood characteristics on disparities in diagnostic delay and testing. Methods to create area-based socioeconomic measures and their

sensitivity to detect theoretically expected gradients in healthcare access and outcomes are documented by the Public Health Disparities Geocoding Project.²⁵ Census tract poverty level is the percent of residents within a census tract living below the federally stated poverty line at the time of the census. We derived this variable for each case by first geocoding patient birth addresses to the census tract level using University of Southern California WebGIS Services.²⁶ We then matched census tract identification numbers with poverty levels for that tract calculated by the Public Health Disparities Geocoding Project for the 1980, 1990, and 2000 census. We used poverty data from the census year proximal to the patient's birth year in the analysis, and this measure was modeled as a continuous variable. Among cases for which race and family history were known, 53 cases were missing poverty information. We found that increased poverty levels were inversely correlated with mother's education (Point Biserial Correlation, -0.032 , $P < 0.001$). Twenty-nine of the 53 cases missing poverty data had data for mother's education level. For these cases, we imputed the average percent poverty level corresponding to the mother's education level. The remaining 24 cases missing poverty information were excluded from the analysis.

We theorized that society wide improvements in disease awareness, increases in perceived utility of early diagnosis, or better access to diagnostic laboratory technology likely reduced the time to diagnosis over the past 25 years. We controlled for these societal changes over time by creating a continuous variable that we defined as months between January 1, 1982, and the patient's birth date. We also restricted our analysis of age at DNA testing to individuals within the analytic dataset born after 1991 because expert opinion and a literature review indicated that mutation analysis was neither commonly recommended nor widely available before 1991.^{27,28}

Statistical analysis

We used SPSS 17.0 software (SPSS Inc., Chicago, IL) to analyze the data. We evaluated the distribution of outcome variables (age at evaluation, CK measurement, and DNA testing) by inspecting histograms, normal probability plots, and by conducting Kolmogorov-Smirnov and Shapiro-Wilks tests. Each variable deviated from normality, but we judged the distribution to be sufficiently close to normal to proceed using common parametric statistical techniques and linear regression. We compared mean differences between dichotomous groups by conducting Student's *t* tests and searched for potential differences across MD STARnet regional sites using analysis of variance.

We used linear regressions to determine which sociodemographic variables affected outcome variables. Because we had no preconception as to which independent variables might best predict outcome variables, we entered all variables into the model simultaneously. Interaction terms were generated and entered into the model, but none showed association with the outcome variable and thus were removed from the final model. We reviewed regression residual plots to confirm that model assumptions were justified and evaluated the collinearity of independent variables by examining variance inflation factors and tolerances. The analytic dataset contains 27 sibling pairs, one triplet, and one quadruplet. We found no evidence of correlations between brothers on outcome variables and treated these cases as independent observations.

RESULTS

Participants

Table 1 illustrates the demographic composition of the analytical dataset and compares it with all males in the year 4

Table 1 Characteristics of participants

	Final analytical data set (%)	MD STARnet database ^a number (%)
Family history		
With family history	128 (34.1)	151 (28.0)
Without family history	247 (65.9)	349 (64.6)
Missing		40 (7.4)
Total	375	540
Race/ethnicity		
White non-Hispanic	259 (69.1)	288 (53.4)
Black non-Hispanic or Hispanic	116 (30.9)	143 (26.4)
Other		18 (3.3)
Unknown		91 (16.9)
Total	375	540
Birth cohort		
1982–1990	166 (44.3)	239 (44.3)
1991–2000	209 (55.7)	301 (55.7)
Total	375	540
Percent poverty		
Mean (SD)	13.6 (11.1)	13.2 (11.4) ^b

^aMales born before January 1, 2001, in year 4 pooled database ($n = 540$).

^bPoverty information known for 499 cases.

database born 1982–2000. The distribution of sociodemographic variables in the analytical dataset did not differ significantly from the year 4 database. Among included cases, approximately two thirds (65.9%) had no family history of DBMD. Approximately 70% of cases were white and 30% were black (21 patients) or Hispanic (95 patients). On average, 13.6% of people in each census tract lived below the poverty line.

Bivariate analysis

Figure 2 presents box plots of the relationship between age and diagnostic steps stratified by family history. Table 2 complements the box plots, reporting on mean case age at diagnostic steps stratified by family history. On average, cases with a family history were first evaluated at 30.8 months, 26 months earlier than cases with no family history. Cases with a family history had CK testing at 35.1 months, 28.9 months before cases without a family history. Age at DNA testing was not significantly different by family history.

Multivariate analysis

Table 3 provides results of linear regressions predicting age at evaluation, CK measurement, and DNA testing. Race/ethnicity and neighborhood poverty independently predicted age at first evaluation after adjusting for family history and birth date. Black and Hispanic children were evaluated 11.7 months after white children and children who lived in poorer neighborhoods were evaluated at younger ages; a 10% rise in neighborhood poverty reduced age at first evaluation by 3.8 months. Mother's education was not a significant predictor in our preliminary analysis and was removed from the final models.

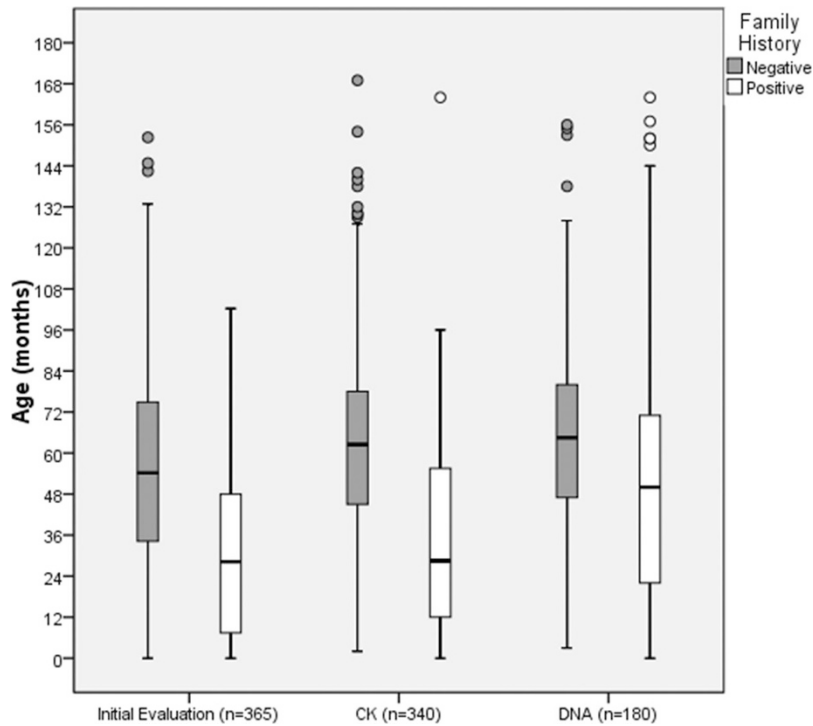


Fig. 2. Median age at 3 steps in the diagnostic process by family history.

Table 2 Mean age at steps to diagnosis

	With family history		Without family history		Total n (% of analytical dataset)
	n	Mean age (mo) (SD)	n	Mean age (mo) (SD)	
Evaluation	123	30.8 (26.2)	242	56.8 ^a (31.2)	365
CK measurement	112	35.1 (28.6)	228	64.0 ^a (30.1)	340
DNA testing ^b	46	56.2 (46.5)	134	64.3 (29.7)	180

^aP < 0.001.

^bBirth years 1991–2000.

In the regression analysis modeling age to CK measurement, adjusting for family history and birth date, we found white children had CK measurement 12.9 months earlier than black and Hispanic children. Similarly, our model of age to DNA testing indicates that white children were tested 24.5 months earlier than black and Hispanic children. Neighborhood poverty level was not a significant predictor of age at CK measurement or DNA testing.

DISCUSSION

This population-based study found that children without a family history of DBMD born between 1982 and 2000 were evaluated for symptoms later and received CK measurement at older ages than was found by Ciafaloni et al.¹¹ using a prior dataset derived from the MD STARnet. This study also found a trend toward younger ages at all steps in the diagnostic process over time that was not found previously.¹² There are important

differences in the case composition of the prior and current analytical datasets. The prior study drew cases from 384 definite or probable cases identified by the MD STARnet at that time. That study excluded cases with a family history, those with no recorded symptoms before age 7 years, those who were recognized by newborn screening or because of concomitant conditions, and those with incomplete data on diagnostic steps. In contrast, this study excludes cases missing information on sociodemographic variables of interest. Also, more cases are now identified by the MD STARnet, including 92 cases from the Georgia MD STARnet site, which were not identified at the time of the previous study. However, we found that mean ages did not differ by site; it is, therefore, unlikely that additional cases from Georgia are responsible for observed differences.

As expected, boys with a family history of DBMD were evaluated and had CK levels measured at younger ages than boys without a family history. Although still a significant predictor in regression modeling, the effect of a positive family history on age at DNA testing was much reduced. Parents may perceive less utility in quickly obtaining DNA testing of a boy if a disease-causing mutation in another family member has been identified. Insurance providers may also be reluctant to authorize confirmational testing of boys born into families with known mutations.

Boys born in neighborhoods with higher poverty levels were evaluated at younger ages. Although the effect size was small, this trend is contrary to the influence that poverty gradients generally have on health outcomes.²⁹ However, in a study examining family level factors associated with age at diagnosis for autism spectrum disorders, Mandell et al.³⁰ found that poverty effects were not linear: children of near-poor families were diagnosed later than either poor or wealthy families. We did not,

Table 3 Linear regression models predicting age at steps to diagnosis

	Model A: age at evaluation (<i>n</i> = 365), B coefficients	Model B: age at CK measurement (<i>n</i> = 340), B coefficients	Model C: age at DNA testing ^a (<i>n</i> = 180), B coefficients
Constant	2548.205	2702.377	7615.630
Family history	-26.556 ^b	-30.249 ^b	-15.769 ^b
Date of birth	-1.250 ^b	-1.325 ^b	-3.786 ^b
Race/ethnicity	11.734 ^c	12.91 ^c	24.508 ^c
Poverty	-37.739 ^c	-27.152	-33.055
R ²	0.20	0.24	0.20

^aBirth years 1991–2000.^b*P* < 0.001.^c*P* < 0.05.

however, find evidence of nonlinear trends in bivariate plots of poverty data and any outcome variable.

Measures of neighborhood poverty levels are not merely a proxy for family level income information but rather capture a mix of individual and area-based socioeconomic effects.³¹ It may be that poorer neighborhoods are more likely to have a range of social services available that monitor child development closely and promote earlier recognition of symptoms, which in turn may lead to earlier evaluation. It is also possible that residents from poorer neighborhoods are more likely to be publicly insured. Families in this group may have better access to health system resources than near-poor families who do not qualify for public assistance.^{32–34}

Racial and ethnic disparities in the diagnostic process might be linked to the actions parents and providers take after the initial evaluation. Although race/ethnicity differences were apparent in the ages of boys at initial evaluation, delays to CK measurement and DNA testing were progressively longer, even after adjustment for other sociodemographic characteristics. At the family level, blacks and Hispanics may be less likely to pursue CK and DNA testing due to a variety of access and knowledge barriers, as well as cultural factors among these groups. For example, Peters et al.³⁵ found that blacks have a lower awareness of diagnostic genetic testing. Suther and Kiroso³⁶ found that blacks and Hispanics have less knowledge of genetic testing, less trust in a medical doctor, and greater concerns about the misuse of genetic testing than do whites; and Zimmerman et al.³⁷ found that blacks were more likely to believe that genetic testing would lead to discrimination.

Barriers contributing to increased diagnostic delays for black and Hispanic children likely also reside at the physician and system levels. Previous investigations found that minority patients are more likely to seek care from lower performing or underresourced hospitals³⁸; and Bach et al.³⁹ found that doctors who treat blacks were less likely to be board certified and to report more access barriers when referring their patients to subspecialists or diagnostic imaging services. Parental survey data suggest that providers refer black and Hispanic children to specialists less often than white children.⁶ Access to medical insurance may also represent a barrier for minority populations. Hispanics and blacks are more likely to be uninsured than whites⁴⁰ and, therefore, may be unable to pay for expensive DNA testing. For Spanish speaking Hispanics, language difficulties may also play a role. Non-English-primary-language households experience multiple disparities in medical health, access to care, and use of services.⁴¹

This study has limitations. First, the distribution of black and Hispanic cases in the MD STARnet varies significantly by site ($\chi^2 = 23.13$, *P* = 0.000). Arizona, Colorado, and Georgia contribute 79% of black and Hispanic cases. Racial and ethnic disparities evident in our regression models may, therefore, be confounded by regional variation in clinical standards of practice or insurance provider policies. However, the inclusion of site in our exploratory regression modeling did not increase the explanatory power of the models, and no individual site was a significant predictor of age at any step in the diagnostic process.

Second, our regression models account for a range of 20–24% of the variance in ages at specific steps in diagnosis, indicating that factors other than those considered herein also play a role in diagnostic delay. Socioeconomic characteristics, such as insurance status, urban rural contrasts, and family level income likely affect the diagnostic process. Because the MD STARnet database is compiled from retrospective chart reviews, patient information on these and other potentially important variables was unavailable or of poor quality and not suitable for inclusion.

CONCLUSION

A delay in the diagnosis of DBMD may create negative consequences for patients and caregivers, including prolonged anxiety about the cause of patients' disabilities; unnecessary, costly, and sometimes painful diagnostic testing, and lost opportunities for time-sensitive treatments such as corticosteroid therapy. These negative outcomes are likely to be particularly acute among black and Hispanic families because their children face relatively longer diagnostic delays than do whites. This population-based study suggests that racial and ethnic disparities arise before the first clinical encounter for symptoms related to DBMD and often continue throughout the diagnostic process. Improving diagnostic disparities for DBMD will require health-care providers and healthcare systems to identify all children who exhibit DBMD symptoms, so they can receive timely CK measurement, early referral to neuromuscular specialists, and definitive diagnostic testing.

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