Impact of Socioeconomic Status on Outcomes of Patients with Kawasaki Disease

Audrey Dionne, MD[1,2], Emily M. Bucholz, MD, PhD, MPH[1,2], Kimberlee Gauvreau, ScD[1,2], Patrick Gould, BS[1], Mary Beth F. Son, MD[2,3], Annette L. Baker, MSN, PNP[1], Sarah D. de Ferranti, MD, MPH[1,2], David R. Fulton, MD[1,2], Kevin G. Friedman, MD[1,2], and Jane W. Newburger, MD, MPH[1,2]

Objective To evaluate the association of neighborhood socioeconomic status (SES) with time to intravenous immunoglobulin treatment, length of stay (LOS), and coronary artery aneurysms (CAAs) in patients with Kawasaki disease.

Study design We examined the relationship of SES in 915 patients treated at a large academic center between 2000 and 2017. Neighborhood SES was measured using a US census-based score derived from 6 measures related to income, education, and occupation. Linear and logistic regression were used to examine the association of SES with number of days of fever at time of treatment, LOS, and CAA.

Results Patients in the lowest SES quartile were treated later than patients with greater SES (7 [IQR 5, 9] vs 6 [IQR 5, 8] days, P = .01). Patients in the lowest SES quartile were more likely to be treated after 10 days of illness, with an OR 1.9 (95% CI 1.3-2.8). In multivariable analysis, SES remained an independent predictor of the number of days of fever at time of treatment (P = .01). Patients in the lowest SES quartile had longer LOS than patients with greater SES (3 [IQR 2, 5] vs 3 [IQR 2, 4], P = .007). In subgroup analysis of white children, those in the lowest SES quartile vs quartiles 2-4 were more likely to develop large/giant CAA 17 (12%) vs 30 (6%), P = .03.

Conclusions Lower SES is associated with delayed treatment, prolonged LOS, and increased risk of large/giant CAA. Novel approaches to diagnosis and education are needed for children living in low-SES neighborhoods. *(J Pediatr 2019;212:87-92)*.

Kawasaki disease is an inflammatory febrile illness of childhood and the leading cause of acquired heart disease in children of developed countries.[1] Coronary artery aneurysms (CAAs) are a serious complication of Kawasaki disease.[1-3] Late diagnosis and treatment of Kawasaki disease are the most important risk factors for the development of aneurysms.[4] Several additional risk factors, such as younger and older age at diagnosis, male sex, longer duration of fever, and failure to respond to initial intravenous immunoglobulin (IVIG) therapy, have been extensively studied[5,6]; however, relatively few studies have examined socioeconomic predictors of outcomes in Kawasaki disease.

Socioeconomic status (SES) is an important predictor of outcomes in pediatric diseases. High SES has been associated with a greater incidence of Kawasaki disease.[7,8] Low SES may limit access to pediatric care or larger referral centers with pediatric cardiology services. In addition, low SES may be associated with lower health literacy or greater caregiver burden, which may in turn delay time to presentation. Only a few studies have examined the association of SES with outcomes in Kawasaki disease,[4,9] each with limitations, including use of zip-code level data rather than smaller Census blocks.[4] Census block data are much smaller units of analysis than zip codes and thus may represent a more homogenous economic status. Recent studies have used Census block data to demonstrate an association between SES and health outcomes in diseases in which proxy variables using zip codes had failed previously.[10]

We sought to study the association of neighborhood SES measured at the census-block level with time to IVIG treatment, length of stay (LOS), and the development and severity of CAAs in pediatric patients with Kawasaki disease. Such information may help identify potential targets and interventions to improve outcomes in patients with Kawasaki disease.

BCH	Boston Children's Hospital
CAA	Coronary artery aneurysm
IVIG	Intravenous immunoglobulin
LOS	Length of stay
SES	Socioeconomic status

From the [1]Department of Cardiology, Boston Children's Hospital; [2]Department of Pediatrics, Harvard Medical School; and [3]Division of Immunology, Boston Children's Hospital, Boston, MA

Funded by the McCance Family Foundation (to J.N.) and the Vella Fund (J.N.). J.N. serves on the Editorial Board for *The Journal of Pediatrics*. The other authors declare no conflicts of interest.

Portions of this study were presented as a poster at the 12th International Kawasaki Disease Symposium, June 12-15, 2018, Yokohama, Japan.

https://doi.org/10.1016/j.jpeds.2019.05.024

Methods

This retrospective study included children aged 0-18 years with a diagnosis of Kawasaki disease between 2000 and 2017 and who were evaluated at Boston Children's Hospital (BCH). We included patients who were treated in the acute phase or referred for a second opinion. We excluded patients without a US mailing address (n = 48); patients with coexisting congenital heart disease except for bicommissural aortic valve, mitral valve prolapse, and hemodynamically insignificant ventricular septal defects (n = 19); and recurrent episodes of Kawasaki disease (n = 12). This study was approved by our institutional review board.

Medical charts were reviewed for demographic characteristics, clinical course, coronary artery dimensions, and SES. Clinical criteria for Kawasaki disease were reviewed and children classified as complete or incomplete Kawasaki disease. Incomplete Kawasaki disease was defined as the presence of fever and 3 or fewer clinical criteria. Patient race was classified as white, black, Asian, or other. Patients with mixed race or missing race (n = 22) were included in the other category. Neighborhood SES was measured using a US Census-based score previously developed by Diez Roux et al using data from the 2010 US Census.[11] The score was calculated from 6 variables related to wealth and income (log of the median household income, log of the median value of housing units, percentage of households receiving interest, dividend, or net rental income), education (percentage of adults 25 years of age or older living in the household who had completed high school, percentage of adults 25 years of age or older living in the household who had completed college), and occupation (percentage of employed persons 16 years of age or older living in the household in executive, managerial, or professional specialty occupations). The neighborhood summary score was constructed by summing the z scores for each of the 6 variables, with greater scores indicating greater neighborhood SES. For analyses, SES was divided into quartiles.

The primary outcome was the number of days of fever at time of IVIG treatment. Secondary outcomes included delay in Kawasaki disease diagnosis, IVIG resistance, hospital LOS, and presence and size of baseline and worst CAA during the first year of follow-up. Delay in IVIG administration was defined as >10 days between onset of fever and IVIG administration. IVIG resistance was defined as persistent or recrudescent fever (>38°C) at least 36 hours after the end of IVIG infusion. Coronary artery z scores were calculated for the right coronary artery and the left anterior descending artery.[12] Coronary dilation and CAA were defined as coronary artery z score between 2-2.5 and ≥2.5, respectively. CAA severity was classified as small if z scores were ≥2.5 to <5, medium if ≥5 to <10, and large/giant if ≥10 or greater than 8 mm.[1]

Quantitative variables were summarized as median [IQR], and categorical variables as frequencies and percentages. Baseline characteristics were compared across SES quartiles using a nonparametric test for trend, the χ^2 test for trend, or Spearman rank correlation depending on the data type. Additional analysis compared patient characteristics for subjects in SES quartile 1 vs quartiles 2-4 using the Wilcoxon rank sum test and Fisher exact test. Linear regression was used to examine the relationship of number of days of fever at the time of IVIG treatment with SES (specifically, SES quartile 1 vs quartiles 2-4), adjusting for age at time of Kawasaki disease diagnosis, complete vs incomplete clinical criteria, and race. Cumulative incidence of hospital discharge by time since admission was estimated using the Kaplan−Meier method and compared between SES quartiles using the log-rank test. We performed subgroup analyses of (1) patients whose initial treatment with IVIG occurred at our center, and (2) children of white race. All analyses were performed with Stata version 15 (StataCorp, College Station, Texas). A 2-tailed P value <.05 was deemed statistically significant.

Results

During the study period, 915 patients met the inclusion criteria, of whom 342 (37%) were female. Median age at time of diagnosis was 3 [IQR 1.5-5.3] years. Patients had a median of 4 [IQR 3-4] clinical criteria, with 650 (71%) having complete clinical criteria. All patients were treated with IVIG, at a median of 7 [IQR 5-9] days after onset of fever. IVIG administration was delayed beyond the 10th day of illness in 118 (13%) of patients. IVIG resistance, defined as fever without other explanation occurring ≥36 hours after completion of IVIG, was found in 212 (24%) of patients. Patients were admitted for a median of 3 [IQR 2-4] days.

At time of diagnosis, coronary dilation was found in 63 (8%) of patients and CAA in 187 (23%) of patients. Of patients with CAA at diagnosis, 145 (78%) had small aneurysms, 24 (13%) medium aneurysms, and 18 (10%) large/giant aneurysms. On at least 1 follow-up evaluation, 281 (31%) had CAA, including 66 (7%) with large/giant aneurysms.

The SES neighborhood summary score ranged from −14.6 to 14.0, with a median of −0.2 [IQR −3.3, 3.8]. **Table I** presents the sociodemographic and clinical characteristics of patients according to quartile of neighborhood SES summary score. Patients in the lowest quartile were younger at time of Kawasaki disease treatment (2.6 [IQR 1.3, 5.2] vs 3.2 [IQR 1.6, 5.4] years, P = .04). The proportion of black and Hispanic children was greater in the lowest SES quartile compared with quartiles 2-4 (28% vs 8% and 28% vs 7%, respectively), and the proportion of Asian children was greater in quartiles 2-4 compared with quartile 1 (20% vs 5%, P < .001). Patients initially treated at BCH compared with those referred from an outside hospital for management and/or second opinion did not differ significantly with respect to SES (median SES −0.04 [IQR −3.3, 3.9] vs −0.5 [IQR −3.9, 3.1], P = .19).

Table I. Baseline characteristics according to SES quartile

Baseline characteristics	Quartile 1 (n = 229)	Quartile 2 (n = 230)	Quartile 3 (n = 227)	Quartile 4 (n = 229)	*P* value*
Age at diagnosis, y	2.6 [1.3, 5.2]	2.9 [1.3, 5.1]	3.5 [1.6, 5.9]	3.2 [1.7, 5.0]	.02
Female	84 (37)	86 (37)	87 (38)	85 (37)	.93
Race†					
White	137 (60)	163 (71)	172 (76)	133 (58)	<.001
Black	63 (28)	37 (16)	10 (4)	11 (5)	
Asian	12 (5)	21 (9)	39 (17)	72 (31)	
Other	7 (3)	3 (1)	4 (2)	6 (3)	
Not reported	10 (4)	5 (2)	2 (1)	5 (2)	
Hispanic ethnicity‡	64 (28)	24 (10)	17 (7)	9 (4)	<.001
Complete Kawasaki disease	160 (70)	167 (73)	165 (73)	158 (69)	.87
Days of fever at time of IVIG treatment	7 [5, 9]	6 [5, 8]	7 [5, 9]	6 [5, 8]	.13
Delay in IVIG treatment	43 (19)	25 (11)	28 (12)	22 (10)	.009
IVIG resistance	60 (27)	50 (23)	56 (25)	46 (20)	.18
Hospital LOS, d	3 [2, 5]	3 [2, 5]	2 [2, 4]	3 [2, 4]	.002

Values are median [IQR] or n (%).
**P* values are based on a nonparametric test of trend for continuous variables and the χ^2 test for trend for nominal variables.
†Race not reported in 22 (2%) patients.
‡Ethnicity not reported in 42 (5%) patients.

Compared with patients in greater SES quartiles, patients in the lowest SES quartile were treated later (7 [IQR 5, 9] vs 6 [IQR 5, 8] days, $P = .01$) and were more likely to have delayed IVIG administration beyond the 10th day of illness (19% vs 11%, $P = .02$). Patients in the lowest SES quartile were more likely to have delayed treatment (unadjusted OR 1.9 [95% CI 1.3-2.8], $P = .002$). After adjustment for age, race, and incomplete clinical presentation, SES remained an independent predictor of the number of days of fever at time of treatment ($P = .01$). Individual components of the SES summary score were not associated with the number of days of fever at time of treatment. Patients in the lowest SES quartile had longer hospital LOS than those in greater SES quartiles despite having the same median value (3 [IQR 2, 5] vs 3 [IQR 2, 4] days, $P = .007$) (Figure); 41% of patients in the lowest SES quartile were admitted for >4 days, compared with only 29% in the other quartiles ($P = .003$).

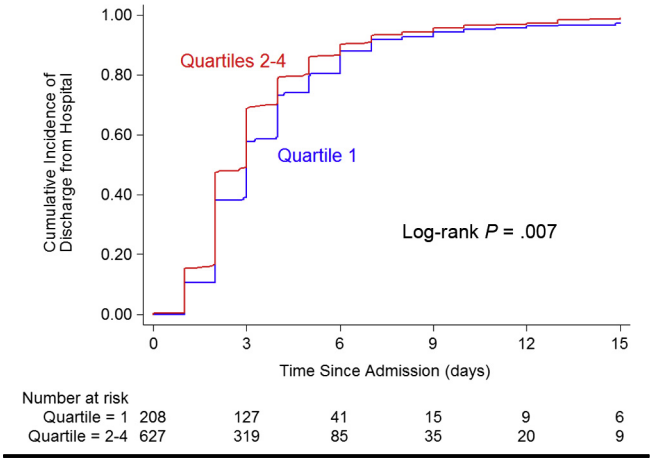

Figure. Comparison of time to hospital discharge for patients in socioeconomic quartile 1 vs quartiles 2-4.

At the time of diagnosis, coronary artery z scores (Table II) were similar in the lowest SES quartile compared with quartiles 2-4 combined (1.5 [IQR 0.6, 2.2] vs 1.5 [IQR 0.7, 2.3], $P = .40$). Moreover, a similar proportion of patients had coronary artery dilation or aneurysm in the lowest vs other quartiles (60 [29%] vs 190 [31%], $P = .79$). The presence and severity of CAA did not differ significantly at baseline between the lowest vs other 3 SES quartiles in the subgroup of white children.

During the first year of follow-up, the largest coronary artery z score (Table II) was also similar across SES quartiles (1.8 [IQR 1.2, 2.9] in the lowest quartile vs 1.8 [IQR 1.3, 2.8] in the combined higher quartiles, $P = .72$). The maximum coronary z scores and categories of aneurysm size did not differ significantly across the 4 SES quartiles when we used a nonparametric test for trend and Spearman rank correlation, respectively (Table II). Large/giant aneurysms were found in 21 (9%) patients in the lowest SES quartile, vs 45 (7%) in the remainder ($P = .19$).

Because racial group was a potential confounder of the relationship between SES quartile and coronary outcome, we further explored the question of association of coronary outcomes with SES quartile within racial groups. In the subgroup of white race, children in the lowest SES quartile were more likely to develop large/giant aneurysms on follow-up than those in combined quartiles 2-4 (17 [12%] vs 30 [6%], $P = .03$). The small number of black and Asian children precluded further subgroup analysis due to insufficient statistical power.

Discussion

Our findings contrast with earlier work on the relationship of SES and Kawasaki disease outcomes. A previous large multicenter retrospective study found no association between neighborhood SES variables and delayed diagnosis in Kawasaki disease.[4] The differences in findings between studies may be related to the measures used to assess SES level. Whereas

Table II. Coronary artery status according to quartile of SES

Coronary artery statuses	Quartile 1 (n = 229)	Quartile 2 (n = 230)	Quartile 3 (n = 227)	Quartile 4 (n = 229)	*P* value*
Baseline echocardiogram					
Coronary artery z score	1.5 [0.6, 2.2]	1.7 [0.8, 2.7]	1.4 [0.7, 2.1]	1.4 [0.7, 2.1]	.61
Coronary artery classification					.29
Normal	144 (71)	131 (63)	150 (74)	150 (72)	
Dilation	17 (8)	18 (9)	13 (6)	15 (7)	
Small aneurysm	35 (17)	45 (22)	25 (12)	40 (19)	
Medium aneurysm	4 (2)	6 (3)	11 (5)	3 (1)	
Large/giant aneurysm	4 (2)	9 (4)	4 (2)	1 (1)	
Follow-up echocardiogram during first year after diagnosis					
Worst coronary artery z score	1.8 [1.2, 2.9]	1.9 [1.3, 3.1]	1.8 [1.3, 2.7]	1.8 [1.3, 2.7]	.60
Coronary artery classification					.14
Normal	137 (60)	128 (56)	146 (65)	146 (64)	
Dilation	24 (10)	17 (7)	18 (8)	17 (7)	
Small aneurysm	43 (19)	56 (24)	33 (15)	49 (21)	
Medium aneurysm	4 (2)	8 (3)	15 (7)	7 (3)	
Large/giant aneurysm	21 (9)	21 (9)	14 (6)	10 (4)	

Values are median [IQR] or n (%).
*P values are based on based on a nonparametric test of trend for z scores and Spearman rank correlation for ordinal coronary artery classification.

zip codes include an average of 30 000 people, US Census blocks are smaller, including only 1000 individuals, and thus represent a more homogenous group. The use of Census block groups and tracts has been shown to perform better than zip codes in capturing economic deprivation.[13] Other publications both in pediatric and adult medicine reported a significant association between SES and outcomes using US Census block data, where zip codes had failed previously.[10,11,13,14] In addition, in this study we used a composite SES summary score including 6 variables related to wealth and income, education, and occupation, following the approach used by Diez Roux et al.[11] Indeed, we found that when individual SES variables such as education, income, or occupation were examined, none were significantly related to time to IVIG treatment. This suggests that these individual variables may not adequately capture all dimensions of SES and highlights the potential incremental value of using a summary score to describe neighborhood SES more accurately.

The identification of patients at risk of delayed Kawasaki disease diagnosis may be helpful for targeting novel approaches to diagnosis and education; however, this requires an understanding of the obstacles interfering with access to care. Health insurance has not proven to be a risk factor for adverse Kawasaki disease outcomes.[9,15] However, greater distance to an academic medical center was significantly associated with delayed diagnosis and coronary complications in a secondary analysis of the Pediatric Heart Network's Kawasaki disease steroid trial.[4,9] Interestingly, studies also have shown that delayed diagnosis of Kawasaki disease often is related to delays in recognizing Kawasaki disease by physicians, rather than delays in seeking medical care by the parents.[15] Consistent with this observation, several studies have demonstrated that centers vary in their percentages of patients with Kawasaki disease with delayed diagnosis.[4,9,15] These observations suggest that, in addition to patient factors, hospital and physician factors may increase the likelihood of delayed diagnosis in patients with Kawasaki disease and be modifiable by targeted clinician education and clinical care pathways.

Other barriers to healthcare access in families with low SES include unstable employment with decreased access to health insurance or sick days, lower health literacy leading to inability to recognize symptoms of illness, language barriers, and lack of transportation.[16-19] A study on delayed diagnosis of developmental dysplasia of the hip showed that patients with late diagnosis were more likely to be non-white, to not speak English, to be of lower income areas, and to have public insurance.[20] The decreased use of healthcare services by non–English-speaking patients, even when adjusted for other social factors, is well described in the literature.[21,22] In a study looking at age at surgical consultation for children with craniosynostosis,[18] African American children were seen later than white children, and this remained true even when controlling for potential socioeconomic confounding variables (family unit structure, parental education, insurance, income). Thus, social and cultural factors other than SES have a significant role in determining access to care and outcome of children. Efforts to improve care and decrease disparities in children with Kawasaki disease should engage minority groups to identify approaches that are culturally acceptable.

In addition to delayed diagnosis, we found prolonged hospital LOS in the lowest SES quartile of patients. Longer hospital LOS and greater hospitalization costs have been reported previously in children of lower SES with non-cardiac disorders.[23,24] Whereas the severity of the underlying disease affects hospital LOS,[25,26] the association between lower SES and longer LOS remained statistically significant, even after adjusting for potential confounders.[24] Similarly, in our cohort of patients with Kawasaki disease, children in the lowest SES quartile had longer hospital LOS. Other factors, including concerns for parental monitoring and access to healthcare in case of recurrent fever or coronary aneurysms, may drive the decision of physicians to prolong hospitalization in children of lower SES families. Identification and mitigation of

barriers to hospital discharge in vulnerable populations are needed to identify strategies to support those families after discharge, and thereby to reduce hospitalization costs.

We did not find a significant relationship between SES quartile and CAA in analyses of the entire cohort, including black and Asian children. However, our cohort had a greater proportion of black children and a lower proportion of Asian children in the lowest vs combined 3 higher SES quartiles. Small retrospective studies have reported a lower risk of CAA in black children[27] and a greater risk in Hispanic and Asian children.[28-31] In contrast, in the largest series, black children were at greater risk of nonresponse to IVIG and had slower normalization of coronary artery z scores, but they did not differ from non-black children in rates of coronary artery abnormalities on baseline echocardiography or of subsequent coronary ectasia or aneurysms.[32] Moreover, genetic variants have been shown to be associated with susceptibility to CAA.[33,34] Because race may be associated with coronary outcome and also was associated with SES, we performed a subgroup analysis of the association of SES with development of CAA in white children; those in the lowest SES quartile had a greater risk of developing large/giant CAA on follow-up. This finding is consistent with the known association of longer duration of fever before IVIG treatment with development of CAA.[1,5,12,35]

Our study should be interpreted in light of its limitations. We used the 2010 US census data for patients with an initial Kawasaki disease diagnosis between 2000 and 2017. Moreover, patients' street address was extracted from the medical chart at the time of analysis and not at the time of initial presentation. Because there may be variation over time in the SES of a neighborhood, and patients may have moved in the interim, the SES variables may not be representative of the SES situation of the family at the time of initial presentation. This study included patients evaluated at a single center, either initially or for a second opinion. Thus, caution should be exercised before extrapolating the findings to other centers in the US. Because not all patients received their initial IVIG therapy at our center, it was not feasible for us to evaluate the impact of distance from center on the time of IVIG treatment. Moreover, the inclusion of patients transferred to BCH may induce a significant bias in the population. Patients initially treated elsewhere were sicker, with a greater rate of coronary complication. However, we did not find any difference in the SES between patients initially treated at our center vs those referred for a second opinion. When analyses were repeated in the subgroup of patients initially treated at BCH, the same results were found. To overcome confounding of SES effects by race, we performed subgroup analyses within children of white race, but we had insufficient power to perform subgroup analyses within the small number of black and Asian children.

Our findings that lower SES is associated with delayed treatment, prolonged LOS, and increased risk of large/giant CAA suggest that social and economic factors are important determinants of outcome in Kawasaki disease. Novel approaches to diagnosis and education may be needed for timely diagnosis and treatment of children who live in low-SES neighborhoods. ■

Submitted for publication Dec 7, 2018; last revision received May 8, 2019; accepted May 10, 2019.

Reprint requests: Jane W. Newburger, MD, MPH, Commonwealth Professor of Pediatrics, Harvard Medical School, Associate Chief for Academic Affairs, Department of Cardiology, Children's Hospital, 300 Longwood Ave, Boston, MA 02115. E-mail: jane.newburger@cardio.chboston.org

References

1. McCrindle BW, Rowley AH, Newburger JW, Burns JC, Bolger AF, Gewitz M, et al. Diagnosis, treatment and long-term management of Kawasaki disease: a scientific statement for health professionals from the American Heart Association. Circulation 2017;135:e927-9.
2. Kato H, Sugimura T, Akagi T, Sato N, Hashino K, Maeno Y, et al. Long-term consequences of Kawasaki disease. A 10- to 21-year follow-up study of 594 patients. Circulation 1996;94:1379-85.
3. Daniels L, Gordon JB, Burns J. Kawasaki disease: late cardiovascular sequelae. Curr Opin Cardiol 2012;27:572-7.
4. Minich LL, Sleeper LA, Atz AM, McCrindle BW, Lu M, Colan SD, et al. Delayed diagnosis of Kawasaki disease: what are the risk factors? Pediatrics 2007;120:e1434.
5. Kim T, Choi W, Woo CW, Choi B, Lee J, Lee K, et al. Predictive risk factors for coronary artery abnormalities in Kawasaki disease. Eur J Pediatr 2007;166:421.
6. Beiser AS, Takahashi M, Baker AL, Sundel RP, Newburger JW. A predictive instrument for coronary artery aneurysms in Kawasaki disease. US Multicenter Kawasaki Disease Study Group. Am J Cardiol 1998;81:1116-20.
7. Harnden A, Mayon-White R, Perera R, Yeates D, Goldacre M, Burgner D. Kawasaki disease in England: ethnicity, deprivation, and respiratory pathogens. Pediatr Infect Dis J 2009;28:21-4.
8. Bronstein DE, Dille AN, Austin JP, Williams CM, Palinkas LA, Burns JC. Relationship of climate, ethnicity and socioeconomic status to Kawasaki disease in San Diego County, 1994 through 1998. Pediatr Infect Dis J 2000;19:1087-91.
9. Okubo Y, Nochioka K, Sakakibara H, Testa M, Sundel RP. National survey of pediatric hospitalizations due to Kawasaki disease and coronary artery aneurysms in the USA. Clin Rheumatol 2017;36:413-9.
10. Singh TP, Naftel DC, Addonizio L, Mahle W, Foushee MT, Zangwill S, et al. Association of race and socioeconomic position with outcomes in pediatric heart transplant recipients. Am J Transplant 2010;10:2116-23.
11. Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, et al. Neighborhood of residence and incidence of coronary heart disease. N Engl J Med 2001;345:99-106.
12. McCrindle BW, Li JS, Minich LL, Colan SD, Atz AM, Takahashi M, et al. Coronary artery involvement in children with Kawasaki disease: risk factors from analysis of serial normalized measurements. Circulation 2007;116:174-9.
13. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramanian SV, Carson R. Geocoding and monitoring of US socioeconomic inequalities in mortality and cancer incidence: does the choice of area based measure and geographic level matter? The Public Health Disparities Geocoding Project. Am J Epidemiol 2002;156:471-82.
14. Epstein D, Reibel M, Unger JB, Cockburn M, Escobedo LA, Kale DC, et al. The effect of neighborhood and individual characteristics on pediatric critical illness. J Community Health 2014;39:753-9.
15. Wilder MS, Palinkas LA, Kao AS, Bastian JF, Turner CL, Burns JC. Delayed diagnosis by physicians contributes to the development of coronary artery aneurysms in children with Kawasaki syndrome. Pediatr Infect Dis J 2007;26:256-60.
16. Weinick RM, Krauss NA. Racial/ethnic differences in children's access to care. Am J Public Health 2000;90:1771-4.
17. Heck KE, Parker JD. Family structure, socioeconomic status, and access to health care for children. Health Serv Res 2002;37:173-86.

18. Brown ZD, Bey AK, Bonfield CM, Westrick AC, Kelly K, Kelly K, et al. Racial disparities in health care access among pediatric patients with craniosynostosis. J Neurosurg Pediatr 2016;18:269-74.
19. Cassell CH, Mendez DD, Strauss RP. Maternal perspectives: qualitative response about perceived barriers to care among children with orofacial clefts in North Carolina. Cleft Palate Craniofac J 2012;49:262-9.
20. Lindberg AW, Bompadre V, Satchell EK, Larson ACR, White KK. Patient factors associated with delayed diagnosis of developmental dysplasia of the hip. J Child Orthop 2017;11:223-8.
21. Fiscella K, Franks P, Doescher MP, Saver BG. Disparities in health care by race, ethnicity, and language among the insured: findings from a national sample. Med Care 2002;40:52-9.
22. Applebaum B, Robbins S. Language access and health equity: changes under the Affordable Care Act. J Health Care Poor Underserved 2016;27:416-26.
23. Wang G, Haberland C, Thurm C, Bhattacharya J, Park KT. Health outcomes in US children with abdominal pain at major emergency departments associated with race and socioeconomic status. PLoS One 2015;10: e0132758.
24. Gipson DS, Messer KL, Tran CL, Herreshoff EG, Samuel JP, Massengill SF, et al. Inpatient health care utilization in the United States among children, adolescents, and young adults with nephrotic syndrome. Am J Kidney Dis 2013;61:910-7.
25. Heys M, Rajan M, Blair M. Length of paediatric inpatient stay, socioeconomic status and hospital configuration: a retrospective cohort study. BMC Health Serv Res 2017;17:274.
26. McConnochie KM, Russo MJ, McBride JT, Szilagyi PG, Brooks AM, Roghmann KJ. Socioeconomic variation in asthma hospitalization: excess utilization or greater need? Pediatrics 1999;103:e75.
27. Porcalla AR, Sable CA, Patel KM, Martin GR, Singh N. The epidemiology of Kawasaki disease in an urban hospital: does African American race protect against coronary artery aneurysms? Pediatr Cardiol 2005;26:775-81.
28. Belay ED, Maddox RA, Holman RC, Cums AT, Ballah K, Schonberger LB. Kawasaki disease syndrome and risk factors for coronary artery abnormalities: United States, 1994-2003. Pediatr Infect Dis J 2006;25:245.
29. Son MB, Gauvreau K, Ma L, Baker AL, Sundel RP, Fulton DR, Newburger JW. Treatment of Kawasaki disease: analysis of 27 US pediatric hospitals from 2001 to 2006. Pediatrics 2009;124:1.
30. Ogata S, Tremoulet AH, Sato Y, Ueda K, Shimizu C, Sun X, et al. Coronary artery outcomes among children with Kawasaki disease in the United States and Japan. Int J Cardiol 2013;9168:3825-8.
31. Son MBF, Gauvreau K, Kim S, Tang A, Dedeoglu F, Fulton DR, et al. Predicting coronary artery aneurysms in Kawasaki disease at a North American center: an assessment of baseline z scores. J Am Heart Assoc 2017;6: e005378.
32. Clark DE, Denby KJ, Kaufman LM, Fill MA, Piya B, Krishnaswami S, et al. Predictors of intravenous immunoglobulin nonresponse and racial disparities in Kawasaki disease. Pediatr Infect Dis J 2018;37: 1227-34.
33. Shimizu C, Eleftherohorinou H, Wright VJ, Kim J, Alphonse MP, Perry JC, et al. Genetic variation in the SLC8A1 calcium signaling pathway is associated with susceptibility to Kawasaki disease and coronary artery abnormalities. Circ Cardiovasc Genet 2016;9: 559-68.
34. Kuo HC, Li SC, Guo MM, Huang YH, Yu HR, Huang FC, et al. Genome-wide association study identifies novel susceptibility genes associated with coronary artery aneurysm formation in Kawasaki disease. PLoS One 2016;11:e0154943.
35. Anderson MD, Todd JK, Glode MP. Delayed diagnosis of Kawasaki syndrome: an analysis of the problem. Pediatrics 2005;115:e428-33.