Low Socioeconomic Status (Measured by Education) and Outcomes in Systemic Sclerosis: Data from the Canadian Scleroderma Research Group

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ABSTRACT. **Objective.** In systemic lupus erythematosus, socioeconomic status (SES) affects outcomes. SES can modify outcomes by altering timing of access to care and adherence. It is unknown whether SES affects systemic sclerosis (SSc) outcomes. Disease can affect income and cause work disability, thus education (completed long before SSc onset) may be a proxy for SES.

Methods. The Canadian Scleroderma Research Group collects annual data on patients with SSc. Baseline data were used from a prevalent cohort. Education was stratified by whether participants completed high school. Regression models assessed effects of education on organ complications and survival.

Results. In our study, 1145 patients with SSc had 11.0 ± 9.5 years' disease duration; 86% were women, with a mean age of 55.4 ± 12.1 years. About one-quarter did not complete high school; this was more common in older patients ($p < 0.0001$), men ($p = 0.017$), those with lower income ($p < 0.0001$), the unemployed ($p < 0.054$), smokers ($p < 0.001$), where DLCO was $< 70\%$ predicted ($p = 0.009$), in those with arthritis ($p = 0.047$), higher Health Assessment Questionnaire-Disability Index ($p = 0.017$), elevated erythrocyte sedimentation rate ($p = 0.019$), median C-reactive protein ($p = 0.002$), proteinuria ($p = 0.016$), steroid use ever ($p = 0.039$), and those more likely to have died in followup (12.7% vs 8.0%; $p = 0.024$). However, adjusting for confounders, there was no effect of education on mortality; whereas mortality was related to age, diffuse cutaneous SSc (dcSSc) subset, elevated pulmonary arterial (PA) pressure on echocardiography, low forced vital capacity expressed as percentage of predicted, and proteinuria (similar in the dcSSc subset and in limited cutaneous SSc), mortality was increased in older patients, those with elevated PA pressure, and those with low DLCO.

Conclusion. Completing less education than high school was not associated with a worse prognosis in SSc after adjustment for confounding characteristics. (First Release Feb 15 2013; J Rheumatol 2013;40:447–54; doi:10.3899/jrheum.120570)

Key Indexing Terms:
EDUCATION SYSTEMIC SCLEROSIS SOCIOECONOMIC STATUS
DISEASE ACTIVITY MORTALITY DISEASE SEVERITY

People with lower socioeconomic status (SES) have worse health outcomes. There is abundant evidence that this association is strong in a variety of outcomes such as mortality and morbidity[1], and for several conditions including infection[2], coronary heart disease[3], diabetes[4], cancer[5], frailty[6], obesity[7], and chronic renal failure[8]. In addition, socioeconomic–demographic variables are important mediators of high levels of disease activity in systemic lupus erythematosus (SLE; an autoimmune connective tissue disease), and poor social support is

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consistently associated with SLE disease activity[9]. Lower SES is also associated with higher rates of death from SLE[10].

Systemic sclerosis (SSc) is a multisystem connective tissue disease characterized by tissue thickening and fibrosis of the skin, often with involvement of internal organs including the gut, lungs, pulmonary arteries, heart, and kidneys[11]. SSc can be divided into limited cutaneous SSc (lcSSc) and diffuse cutaneous SSc (dcSSc)[12]. The epidemiology of SSc is not totally known[13].

There are several measures of SES, such as education[14], income[15], employment status, and occupation[16]. The onset of SSc is often in middle age, so it usually occurs years after education is completed. One could speculate that low education level (a surrogate of SES that has occurred long before SSc diagnosis) may be associated with worse outcomes in SSc (more damage, disease activity, more severe organ involvement, and even death). Other measures of SES such as income and employment status are likely to be affected by SSc and thus lower SES can be the result of worse SSc if measuring employment and income. The goal of our study was to analyze the effect of education (as a proxy of SES) on SSc for disease activity, severity, disability, organ involvement, and mortality in a large database to determine whether low SES predicts worse outcomes in SSc.

MATERIALS AND METHODS

Study population and protocol. We used data obtained from a cohort study of patients with both prevalent and incident SSc who are followed annually at 20 centers within the Canadian Scleroderma Research Group (CSRG). Baseline data were mostly from longstanding patients who enrolled in the cohort. Data included a unique identification number, demographics (age, sex, marital status, SES measures such as education and household income, employment status, and ethnicity), disease duration since first non-Raynaud phenomenon (RP) symptoms, SSc type, disease manifestations [including the presence and severity of organ involvement such as chronic renal failure (serum creatinine, scleroderma renal crisis), functional class, presence of pulmonary arterial hypertension (PAH) and interstitial lung disease (ILD)], modified Rodnan skin score (mRSS) and tests [hemoglobin, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), autoantibodies, pulmonary function tests (PFT), chest radiographs, echocardiograms, and high-resolution computed tomography (HRCT) chest scanning (where indicated)]. Standardized scales used in the CSRG include the SSc disease activity[17,18,19,20], SSc disease severity[21], and function as measured by the HAQ-DI.

Ethics approval. All sites obtained ethics approval (centrally and/or locally) and all patients enrolled in the CSRG signed informed consent after receiving a letter of information in either English or French.

Statistical analyses. Statistical analyses were performed using IBM SPSS statistics software, version 20. Frequencies were expressed as the percentage and compared between groups by Fisher's exact test. Continuous variables were reported as the mean ± SD and their differences were analyzed using independent t-tests. Outcomes of interest were specific organ involvement, skin fibrosis as measured by mRSS, pulmonary hypertension (PH; defined below), ILD, scleroderma renal crisis (SRC) ever, polymyositis by Bohan and Peter criteria[22,23], 28-joint swollen joint count (SJC) ≥ 4, tender joint count (TJC) ≥ 8, arthritis (SJC28 ≥ 4 or TJC28 ≥ 8), current digital ulcers, hemoglobin (g/l), creatinine (μmol/l), creatine kinase (U/l), ESR (mm/h), CRP (mg/dl), proteinuria (present or absent), autoantibodies (antinuclear antibody, anti-topoisomerase I, anticentromere, anti-RNA polymerase III), HAQ-DI (0–3), and mortality. PAH was present if right heart catheterization showed pulmonary arterial pressure (PAP) > 25 mm Hg and a wedge pressure < 15 mm Hg[24]. PH was present if no right heart catheterization was done but the estimated systolic PAP (sPAP) on echocardiogram was > 40 mm Hg[25]. ILD was measured by chest radiograph, HRCT of the chest, and PFT.

Socioeconomic status was measured by education level ("did not complete high school" and "completed high school or more"). Other SES factors studied were annual household income before tax (< $30,000 CAN per year) and employment status (yes/no), but these were measured at first visit into the cohort and thus after disease onset, so income and work disability could not be predictive of SSc outcome. Education is only a part of total SES but was used as a surrogate because the exposure of "highest education level completed" would have occurred long before SSc onset and we wanted to determine whether education level was predictive of outcomes in SSc.

The associations between SES and SSc outcomes were studied initially with Spearman's correlation coefficients (rho) and then logistic regression analyses between education level and other factors that were statistically significantly associated with education. The effect of education level on SSc mortality was studied overall and in the dcSSc and lcSSc groups, adjusting for length of followup and other confounders. Survival curves for patients with SSc divided by education level (did not complete vs completed high school) were constructed using Kaplan-Meier survival estimates (Figure 1) and log-rank (Mantel-Cox) tests. The analyses defined disease duration starting at the date of first non-RP symptom attributed to SSc to the last annual followup visit or the date of death. All variables in which p value was < 0.05 between education groups were adjusted in the analyses if they were potential confounders. Survival analyses were redone using forced vital capacity (FVC) expressed as percentage of predicted and DLCO as percentage of predicted as continuous outcomes and removing inflammatory markers. The effect of education on the presence or absence of significant organ involvement (such as SRC ever, elevated creatinine, right heart catheterization-proven PAH, ILD requiring treatment) was explored using logistic regression models that adjusted for important variables that were related to the outcome such as dcSSc, autoantibodies, etc.

RESULTS

The study included 1145 patients with SSc; 86% (986) were women. Most of the patients were white (92%). The mean age was 55.4 ± 12.1 years (range 18–88 yrs) and the mean disease duration from onset of first non-RP symptom was 11.0 ± 9.5 years (range < 1 yr to 54 yrs). Table 1 shows the baseline characteristics of the entire sample, and a comparison between patients who did not and those who did complete high school, and Spearman's correlations and p values. Having less education was significantly more common among older patients (p < 0.0001), men (p = 0.017), patients with lower income (p < 0.0001), less employment (p < 0.0001), current smoker (p = 0.001), lower DLCO percentage predicted (p = 0.019), DLCO < 70% predicted (p = 0.009), SJC ≥ 4 (p = 0.013), higher HAQ-DI (p = 0.017), higher ESR (p = 0.019), elevated ESR > 20 mm/h (p = 0.0001), higher median CRP (p = 0.002), proteinuria (p = 0.016), corticosteroid use (p = 0.039), and death (12.7% vs 8.0%; p = 0.018).

For the outcomes of interest, by univariate analyses in patients with SSc, having less education was a predictor of DLCO < 70% predicted (OR 1.512, 95% CI 1.117-2.047),
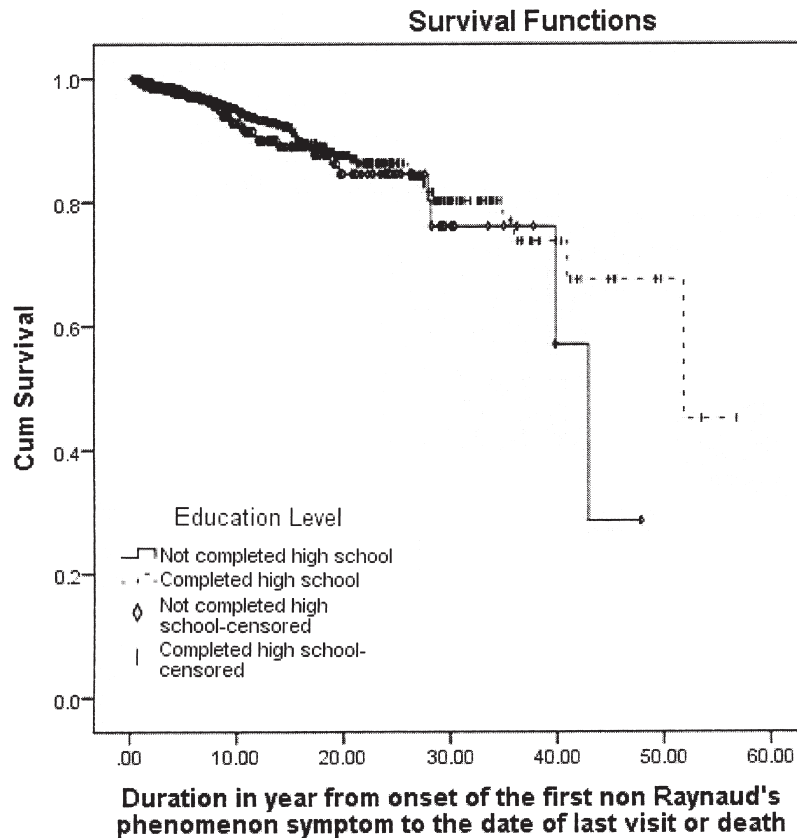
Survival Functions

Figure 1. Kaplan-Meier curves for survival between patients with different education levels (did not complete high school vs completed high school or higher). Survival was not different in those with low education at baseline entry into the Canadian Scleroderma Research Group database (p = 0.297).

SJC28 ≥ 4 (OR 2.086, 95% CI 1.206-3.609), elevated ESR > 20 mm/h (OR 1.811, 95% CI 1.341-2.447), proteinuria (OR 1.896, 95% CI 1.144-3.143), corticosteroid use (OR 1.489, 95% CI 1.034-2.144), and death (OR 1.677, 95% CI 1.088-2.585). There was no effect of education on various organ outcomes such as ILD, PH, or SRC when adjusting for variables known to be associated with each organ involvement (such as topoisomerase I and dcSSc subset with ILD, RNA polymerase III and dcSSc with SRC, etc.; data not shown).

Habits that could be associated with education. Smoking was more common in lower education (p = 0.001) and lower education predicted smoking in patients with SSc (OR 1.872, 95% CI 1.319-2.658) by univariate analysis, whereas the mean weekly alcohol consumption was not different between the 2 education groups.

Effect of education on SSc mortality in all patients (overall), and dcSSc and lcSSc subsets. Table 2 shows the multivariate analyses in SSc survival for the 2 education groups (overall, and in subsets dcSSc and lcSSc). After adjustment for age and other significant variables, low education level lost its significance for SSc, and in the dcSSc and lcSSc groups.

PFT measures were studied as dichotomous and continuous for FVC and DLCO. In addition, analyses were performed with and without an elevated ESR because ESR could be related to active organ involvement. Survival curves for patients entering the CSRG separated by education level did not show significant differences in survival between the education levels (p = 0.297). The survival curves associated with death and adjusted for length of followup are not shown. Similarly, education was not significantly associated with survival in adjusted analyses in the lcSSc and dcSSc subsets.

DISCUSSION
Our study demonstrated that low education defined by "did not complete high school" as a proxy of SES did not affect outcomes after adjustment for other factors associated with less education such as worse pulmonary function (FVC in overall and dcSSc subsets and DLCO in lcSSc subset), arthritis, proteinuria, higher systemic inflammation measured by elevated ESR and CRP, and more corticosteroid use. In lcSSc, survival was less in those with a low DLCO, which is indicative of pulmonary fibrosis[26],

Table 1. Comparison of demographic and disease characteristics between education levels (less than high school vs high school or beyond) for patients with systemic sclerosis from the Canadian Scleroderma Research Group (CSRG) database.

Characteristic	Total	Education < High School	Education ≥ High School	p	Spearman ρ	p
N (% of total group)	1145 (100)	292/1058 (27.6)	766/1058 (72.4)	—	—	—
Age at baseline visit, yrs, mean ± SD	55.39 ± 12.13	60.3 ± 10.84	53.61 ± 12	< 0.0001	–0.263	0.0001
Women (%)	986/1145 (86.1)	239/292 (81.8)	672/766 (87.7)	0.017*	0.076	0.013*
Disease duration from onset of RP to baseline visit, yrs, mean ± SD	14.70 ± 12.33	14.50 ± 12.36	14.74 ± 12.11	0.785	—	—
Disease duration from onset of first non-RP manifestation to baseline visit, yrs, mean ± SD	11.00 ± 9.50	11.56 ± 9.43	10.78 ± 9.34	0.235	—	—
Diffuse cutaneous subset (%)	396/1043 (38)	88/264 (33.3)	285/711 (40.1)	0.054	0.062	0.054
Disease duration ≤ 3 yrs (%)	244/1102 (22.1)	61/282 (21.6)	164/746 (22.0)	0.933	—	—
Early diffuse cutaneous subset (%)	117/1100 (10.6)	28/281 (10.0)	81/747 (10.8)	0.734	—	—
Early limited cutaneous subset (%)	109/1094 (10)	29/280 (10.4)	74/743 (10.0)	0.907	—	—
White (%)	953/1036 (92.0)	262/280 (93.6)	687/749 (91.7)	0.362	—	—
Asian (%)	39/825 (4.7)	4/223 (1.8)	34/595 (5.7)	0.015*	0.083	0.018*
Black (%)	14/818 (1.7)	3/223 (1.3)	11/588 (1.9)	0.768	—	—
Aboriginal (%)	55/1063 (5.2)	21/289 (7.3)	33/766 (4.3)	0.060	0.060	0.052
Married (%)	732/1061 (69.0)	119/292 (68.2)	531/765 (69.4)	0.710	—	—
Working (%)	432/918 (47.1)	75/238 (31.5)	356/672 (53.0)	0.0001*	0.189	0.0001*
Disabled/sick leave (%)	230/871 (26.4)	66/242 (27.3)	163/622 (26.2)	0.797	—	—
Unemployed (%)	52/831 (6.3)	21/231 (9.1)	31/593 (5.2)	0.054	–0.071	0.041*
Yearly household income < $30,000 CAN	262/951 (27.5)	116/250 (46.4)	141/694 (20.3)	0.0001*	–0.259	0.0001*
Current smoker (%)	161/1061 (15.2)	63/292 (21.6)	98/765 (12.8)	0.001*	–0.109	0.0001*
Mean no. drinks per wk (%)	2.5 (4.9)	2.4 (4.2) (258)	2.6 (5.2) (674)	0.679		
mRSS	10.04 ± 9.54	9.52 ± 9.14	10.38 ± 9.79	0.202	0.027	0.379
mRSS > 20 (%)	155/1102 (14.1)	32/282 (11.3)	116/748 (15.5)	0.091	—	—
sPAP, mm Hg	36.91 ± 16.23	37.60 ± 15.54	36.75 ± 16.73	0.551	—	—
Elevated sPAP > 40 mm Hg (%)	172/668 (25.7)	50/183 (27.3)	115/457 (25.2)	0.617	—	—
Moderate to large pericardial effusion (%)	16/949 (1.7)	4/243 (1.6)	10/653 (1.5)	1.000	—	—
Lung fibrosis (%)	228/1062 (21.5)	65/274 (23.7)	150/717 (20.9)	0.344	—	—
Lung fibrosis in those with HRCT (%)	279/889 (31.4)	83/230 (36.1)	182/599 (30.4)	0.134	—	—
HRCT chest; honeycomb appearance (%)	57/262 (21.8)	13/73 (17.8)	40/178 (22.5)	0.497	—	—
TLC, % predicted	93.12 ± 19.11	92.98 ± 20.11	93.12 ± 18.61	0.925	—	—
FVC, % predicted	90.84 ± 19.75	89.03 ± 19.5	91.52 ± 19.87	0.094	0.060	0.074
DLCO, % predicted	70.65 ± 21.96	67.74 ± 22.32	71.72 ± 21.81	0.019*	0.096	0.005*
% with TLC < 80% predicted	207/899 (23.0)	64/236 (27.1)	131/613 (21.4)	0.084	0.061	0.075
% with FVC < 70% predicted	125/946 (13.2)	40/241 (16.6)	76/654 (11.6)	0.056	–0.066	0.049*
% with DLCO < 70% predicted	439/917 (47.9)	128/231 (55.4)	286/634 (45.1)	0.009*	–0.091	0.007*
SRC (%)	48/1121 (4.3)	10/286 (3.5)	38/760 (5.0)	0.407	—	—
Polymyositis by Bohan & Peter criteria (%)	15/1105 (1.4)	3/282 (1.1)	12/744 (1.6)	0.771	—	—
Tendon friction rub (%)	163/1110 (14.7)	42/282 (14.9)	111/754 (14.7)	0.922	—	—
28 SJC ≥ 4 (%)	59/1096 (5.4)	24/280 (8.6)	32/744 (4.3)	0.013*	–0.084	0.007*
Arthritis (SJC ≥ 4 or TJC ≥ 8; %)	94/1096 (8.6)	33/280 (11.8)	57/744 (7.7)	0.047*	–0.065	0.038*
Current digital ulcer (%)	93/1121 (8.3)	22/287 (7.7)	64/757 (8.5)	0.801	—	—
Digital gangrene/necrosis (%)	12/1123 (1.1)	1/287 (0.3)	11/759 (1.4)	0.197	—	—
Digital loss/amputation (%)	66/1124 (5.9)	14/288 (4.9)	47/759 (6.2)	0.463	—	—
HAQ-DI (0–3)	0.81 ± 0.71	0.9 ± 0.67	0.78 ± 0.72	0.017*	–0.090	0.004*
Hemoglobin (g/l)	13.59 ± 1.24	14.13 ± 1.54	13.22 ± 0.91	0.171	—	—
Serum creatinine (μmol/l)	81.70 ± 51.31	85.05 ± 40.25	81.07 ± 56.56	0.291	—	—
Creatine kinase (μ/l)	95.57 ± 86.31	95.08 ± 83.21	95.58 ± 86.29	0.938	—	—
ESR, mm/h	21.90 ± 21.03	24.53 ± 19.73	20.81 ± 21.52	0.019*	–0.135	0.0001*
Elevated ESR > 20 mm/h (%)	353/923 (38.2)	118/244 (48.4)	212/622 (34.1)	0.0001*	–0.132	0.0001*
CRP, median (mg/l)	3.60	4.30	3.40	0.002*	–0.106	0.002*
CRP, mean (mg/l)	9.54 ± 19.98	12.20 ± 25.05	8.78 ± 18.35	0.055		
Elevated CRP > 8 mg/l (%)	235/913 (25.7)	69/239 (28.9)	150/613 (24.5)	0.191	—	—
ANA (%)	959/1020 (95.0)	253/267 (94.8)	655/689 (95.1)	0.869	—	—
ACA (%)	353/1020 (34.6)	93/267 (34.8)	236/689 (34.3)	0.880	—	—
Antitopoisomerase I (Scl70; %)	153/1021 (15.0)	37/268 (13.8)	107/689 (15.5)	0.547	—	—
Anti-RNA polymerase III (%)	144/1021 (14.1)	39/268 (14.6)	102/689 (14.8)	1.000	—	—

Table 1. Continued.

Characteristic	Total	Education < High School	Education ≥ High School	p	Spearman ρ	p
Proteinuria (%)	72/939 (7.7)	28/243 (11.5)	41/638 (6.4)	0.016*	−0.085	0.012*
Corticosteroids ever-use (%)	161/1124 (14.3)	53/288 (18.4)	100/760 (13.2)	0.039*	−0.066	0.032*
Immunomodulator ever-use (%)	275/1124 (24.5)	69/288 (24.0)	195/760 (25.7)	0.633	—	—
Dead (%)	107/1145 (9.3)	37/292 (12.7)	61/766 (8.0)	0.024*	−0.073	0.018*
Duration from baseline visit to death, yrs	1.75 ± 1.26	1.77 ± 1.04	1.77 ± 1.35	0.974	—	—
Duration from onset of RP to death, yrs	16.34 ± 12.65	16.95 ± 11.6	14.99 ± 12.29	0.464	—	—
Duration from onset of first non-RP symptom to death, yrs	13.24 ± 11.25	12.87 ± 8.94	12.95 ± 12.01	0.973	—	—

* $p < 0.05$. RP: Raynaud phenomenon; mRSS: modified Rodnan skin score; sPAP: systolic pulmonary arterial pressure; HRCT: high-resolution computed tomography; TLC: total lung capacity; FVC: forced vital capacity; SRC: scleroderma renal crisis; SJC: swollen joint count; TJC: tender joint count; HAQ-DI: Health Assessment Questionnaire-Disability Index; ESR: erythrocyte sedimentation rate; CRP: C-reactive protein; ANA: antinuclear antibody; ACA: anticentromere antibodies.

Table 2. Multivariate analysis of dead versus alive in SSc (overall) and in dcSSc and lcSSc subsets. Data are from the univariate models comparing dead versus alive, adjusting for followup time for overall and subsets (data not shown). Education did not appear in the model as statistically significant.

Significant Predictors	Data with All Significant Variables in the Regression Analysis OR (95% CI); p	Data Excluding ESR and Using % FVC and DLCO % Predicted as Continuous Variables OR (95% CI); p
Overall		
Age	1.066 (1.030–1.103); 0.0001	1.067 (1.032–1.102); 0.0001
dcSSc	3.817 (1.805–8.069); 0.0001	3.076 (1.499–6.310); 0.002
sPAP > 40 mm Hg	6.330 (3.042–13.172); 0.0001	5.025 (2.450–10.305); 0.0001
FVC, % predicted < 70	NS	—
FVC, % predicted (continuous)	—	0.983 (0.066–1.000); 0.048
Proteinuria	3.295 (1.288–8.426); 0.013	2.700 (1.115–6.542); 0.028
dcSSc subset		
Age	1.056 (1.010–1.103); 0.016	1.059 (1.016–1.104); 0.007
sPAP > 40 mm Hg	3.735 (1.289–10.826); 0.015	4.016 (1.504–10.720); 0.006
FVC, % predicted < 70	3.039 (1.008–9.163); 0.048	—
FVC, % predicted (continuous)	—	0.972 (0.949–0.997); 0.027
ESR > 20 mm/h	3.001 (1.011–8.905); 0.048	—
Proteinuria	3.978 (1.110–14.255); 0.034	4.583 (1.449–14.500); 0.010
lcSSc subset		
Age	1.076 (1.014–1.140); 0.015	1.080 (1.019–1.146); 0.010
sPAP > 40 mm Hg	4.101 (1.301–12.924); 0.016	3.515 (1.064–11.611); 0.039
DLCO, % predicted < 70	6.162 (1.306–29.083); 0.022	—
DLCO, % predicted (continuous)	—	0.961 (0.933–0.991); 0.010

SSc: systemic sclerosis; dcSSc: diffuse cutaneous SSc; lcSSc: limited cutaneous SSc; ESR: erythrocyte sedimentation rate; FVC: forced vital capacity; sPAP: systolic pulmonary arterial pressure. NS: nonsignificant.

pulmonary vasculopathy[27,28], or both, or smoking. Impaired diffusing capacity has also been linked to altered nailfold capillary microscopy findings in SSc[29]. Proteinuria that has been reported in 25% of patients with SSc is a predictor of glomerular permeability[30] and has been previously shown to be related to worse 5-year survival in SSc[31]. Elevated ESR[31] and CRP[32] occur in those with SSc disease activity and poor survival. Increased CRP is also correlated with arthritis in SSc[33,34].

It may be that behaviors and attitudes related more in low education compared to higher education may be related to different outcomes, but with adjustment for confounders (factors associated with education), the effect of education on survival and other outcomes is not seen.

In SLE, lower SES is associated with more disease activity, organ damage, worse physical function, and work disability[35,36,37]. Abnormal illness-related behavior[9,37,38,39], low self-efficacy for disease management, less knowledge about SLE, and less personal control over one's health are associated with higher disease activity, damage, and worse physical and mental function in SLE[36,40].

However, low education in SSc lost its significance for

poor outcomes in SSc. Overall, the dcSSc subset has a worse prognosis[41,42,43,44,45]. In the lcSSc subset, lower education was correlated with death in univariate correlations but did not retain significance after adjustment for important significant variables (data not shown). This finding could imply that behaviors and/or attitudes related to poor education might not show a significant effect on disease manifestations and organ damage in dcSSc, but they could modify outcomes in the less-severe subset (lcSSc). However, this was not the case in lcSSc after multivariate analyses.

Our study included mostly whites, so an interaction between certain ethnicities and education could not be ascertained. For instance, Aboriginal people in our study had lower education. In US studies, African Americans have more dcSSc subset[46], PAH[46], digital ulcers[46], early pulmonary involvement[47], and later hospitalization and death[48].

Epidemiologic data have established cigarette smoking as an important environmental factor interacting with the shared epitope and increasing the risk of rheumatoid arthritis[49], and smoking is more common in connective tissue diseases. Smoking in SSc may worsen disease severity[50,51]. Smoking was more common in the low education group.

After adjustment for variable length of followup, SSc mortality was related to older age, dcSSc, sPAP > 40 mm Hg, low FVC, and proteinuria, and these findings are similar to those of other studies[52,53,54,55,56,57]. In patients with dcSSc, the results were similar. In the lcSSc subset, low DLCO (not FVC) was associated with mortality. These results are in agreement with other studies[52,53,54,56,57,58,59,60].

Although the sample size was large, there are some limitations of our study. The CSRG is a prevalent and incident cohort that initially enrolled patients with long disease duration, so the disease could have been less severe (a survivor cohort). In addition, the database could be biased because only outpatients were enrolled, and severely ill patients may be less apt to be approached (they may be too sick to complete all the forms), which could underestimate those with the worst outcomes. We also did not compare the education levels of the patients with SSc to the regional population matched for age and sex, so we cannot say whether there is more lower education in SSc than expected in the general population (and this was not the purpose of our study). We can conclude that, with adjustment for other important factors, education is not associated with SSc outcomes. There is a lack of information on temporal relationships because of the way the questions were asked. Data were collected by many centers throughout Canada, which allows the results to be more generalizable, but minorities may be underrepresented. The definition of SES as solely related to education is a limitation, but was chosen because other SES measures would be affected by SSc and

we were looking for a clear temporal relationship between SES (as measured by education) occurring long before SSc onset. The patients in the CSRG database have universal healthcare, but it does not apply to medications in many Canadian provinces.

Completion of education that occurred prior to SSc onset was used as a proxy for SES in our study. Low education was associated with worse outcomes but after adjustment for known risk factors/confounders, low education was not associated with poor outcomes in SSc.

APPENDIX

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