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The COVID-19 syndemic: a perfect storm for the life expectancy of the most disadvantaged Americans

Charlotte Probst[1,2,3,4,*], Sophie Bright[5], Yachen Zhu[6], Carolin Kilian[1,7,8], Charlotte Buckley[5,9], Yu Ye[6], Nina Mulia[6], William C. Kerr[6], Jürgen Rehm[1,2,3,4,8,10,11]

[1]Institute for Mental Health Policy Research, Centre for Addiction and Mental Health (CAMH), Toronto, ON, Canada
[2]Campbell Family Mental Health Research Institute, Centre for Addiction and Mental Health (CAMH), Toronto, ON, Canada
[3]Department of Psychiatry, University of Toronto, Toronto, ON, Canada
[4]Faculty of Medicine, Institute of Medical Science, University of Toronto, Toronto, ON, Canada
[5]School of Electrical and Electronic Engineering, University of Sheffield, Sheffield, United Kingdom
[6]Alcohol Research Group, Public Health Institute, Emeryville, CA, United States
[7]National Institute of Public Health, University of Southern Denmark, Copenhagen, Denmark
[8]Department of Psychiatry and Psychotherapy, Center for Interdisciplinary Addiction Research, University Medical Center Hamburg-Eppendorf, Hamburg, Germany
[9]Department of Psychology, University of Liverpool, Liverpool, United Kingdom
[10]Dalla Lana School of Public Health, University of Toronto, Toronto, ON, Canada
[11]Program on Substance Abuse & WHO Collaborating Centre on Substance Use, Noncommunicable Diseases, and Policy Impact, Public Health Agency of Catalonia, Barcelona, Spain

*Corresponding author. Institute for Mental Health Policy Research, Centre for Addiction and Mental Health (CAMH), 250 College Street, Toronto, ON M5T 1R8, Canada. E-mail: charlotte.probst@camh.ca

Abstract

Background: To explore the syndemic nature of the COVID-19 pandemic by identifying which subpopulations in the United States (US) suffered the greatest losses in life expectancy (LE) in 2020 and 2021, and to which extent these losses can be attributed to COVID-19 and 'other' causes of death.

Methods: We analysed individual death records for 2018–2021 from the National Vital Statistics System and population counts from the American Community Survey. Life table and continuous change decomposition analyses were used to quantify cause-/.specific contributions to changes in LE over time in population subgroups defined by sex, educational attainment, and race/ethnicity.

Results: From 2019 to 2020, educational differences in LE (high minus low education) increased substantially by 5.0 and 2.6 years in Hispanic men and women, respectively, with increases of one to two years among Black and White adults. Nearly all losses in LE among high-education Hispanic and White groups were due to COVID-19, while among low-education White and Black groups, COVID-19 accounted for 40%–47% of the total losses in LE. Changes in LE were much smaller during 2020–2021.

Conclusions: COVID-19 widened preexisting inequalities in LE in the US, both via direct mortality and through syndemic interactions with other diseases and health conditions. The underlying social, political, economic, and environmental factors driving the clustering and interaction of diseases among the most disadvantaged Americans need to be addressed.

Keywords: syndemic; life expectancy; COVID-19 mortality; health inequalities; educational disparities; race and ethnicity.

Key Messages

- The syndemic nature of COVID-19 is demonstrated by decomposing the unequal increases in both COVID-19 mortality 'and' changes in mortality from other causes that drove losses in life expectancy across the intersections of sex, race/ethnicity, and education.
- During the COVID-19 pandemic, preexisting socioeconomic and racial and ethnic inequalities in life expectancy in the United States (US) widened, with Hispanic and Black men with low education being disproportionally affected.
- Addressing the underlying social, political, economic, and environmental factors driving the clustering and interaction of diseases for these populations is essential to counter increasing inequalities in life expectancy in the US.

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Background

In the United States (US), racial and ethnic minority groups and individuals with low socioeconomic status (SES) faced higher risks of COVID-19 infections, hospitalizations, and mortality [1–3]. Mortality from 'other' causes also increased during the pandemic, particularly in certain subgroups [4–6]. These trends unfolded against a backdrop of preexisting inequalities in life expectancy (LE) across the US, with LE stagnating or deteriorating for certain demographic groups over the last decade [7, 8].

Previous studies have examined COVID-19 deaths rates by race/ethnicity, sex, or SES, demonstrating substantially higher mortality in racial and ethnic minority groups (except Asian Americans), especially in those with a high school degree or less compared to White individuals with college degree [2, 9–11]. However, no known study has decomposed overall mortality by the intersections of sex, SES, and race/ethnicity while considering deaths from other causes during the pandemic.

COVID-19 has been conceptualized as a syndemic [12–16], characterized by: (1) clustering of multiple diseases or health conditions within a population or context, (2) adverse interactions between these conditions resulting in worse health, 'and' (3) social, political, or environmental factors that further exacerbate the disease/disease interactions [12, 17]. This framework was evident in the context of COVID-19 in the US, exemplified by: (1) Health condition clustering: racial and ethnic minorities (except Hispanics) have higher odds of hypertension [18] while behavioral risk factors like smoking and obesity are more common among individuals with a low SES [19–21]. Type 2 diabetes prevalence and mortality follow a socioeconomic gradient [20, 22]. (2) Adverse interactions: smoking and obesity exacerbate the course of a COVID-19 infection [23] and preexisting conditions like diabetes and hypertension were linked to increased risks of severe COVID-19 disease and mortality [23]. (3) Social, political, and environmental factors: unequal health insurance coverage left 28 million US citizens uninsured or underinsured in 2020 [24], disproportionately affecting socioeconomically deprived populations and racial and ethnic minorities [25]. Additionally, exposure risks and the ability to cope with pandemic-related hardships strongly reflected social determinants of health, including job security, savings, occupational hazards, and transportation access [26].

Not all diseases will exhibit an equal amount of syndemic interaction with COVID-19, nor will the effects be equivalent across population groups. To better understand COVID-19 as a syndemic, it is necessary to identify which diseases, besides COVID-19, contributed most to inequalities in LE, and for which groups. Given that previous studies observed heightened COVID-19 death rates for certain groups at the intersection of race/ethnicity, SES, and sex [2, 9–11], we hypothesized that:

1) The largest declines in overall LE in 2020 were experienced by racial and ethnic minority groups with low education. Moreover, we sought to explore the syndemic nature of COVID-19 by quantifying the extent to which losses in LE were due to COVID-19 and 'non-COVID-19' causes of mortality, hypothesizing that:
2) The unequal LE losses were composed of both COVID-19 mortality 'and' changes in non-COVID-19 mortality with compositions varying with race/ethnicity, education, and sex.

Methods

Data sources

We obtained individual death records published in the National Vital Statistics System (NVSS) 'Multiple Cause of Death Files' (2018–2021) [27], containing information on underlying causes of death (COD; ICD-10), education, race/ethnicity, age, and sex [28]. Mortality rates were calculated using corresponding population estimates from the nationally representative American Community Survey (ACS) [29, 30]. To address data collection disruption in 2020, we applied experimental weights provided by the US Census Bureau to adjust population estimates.

Variable definitions

Five-year age groups were used (25–29 to 80+). Education, the only available SES indicator, was classified into high (college degree or more), middle (some college, no Bachelor's degree), and low (high school or less). Race/ethnicity included Hispanic, non-Hispanic Black, non-Hispanic White, and non-Hispanic 'other' (e.g. Indigenous, Asian, multiracial).

NVSS data were missing information on education (1.8%) and race/ethnicity (0.3%). Missing data were imputed by randomly assigning deaths to categories based on observed proportions by year, age, sex, and race/ethnicity or education [7].

Nineteen COD categories were selected to include (i) leading COD in the US in 2019 and 2020 [31, 32]; and (ii) COD that contributed to the growing socioeconomic inequalities over the past two decades [33, 34]. These included COVID-19, cardiovascular diseases, cancers, other non-communicable diseases (NCD), unintentional injuries, deaths of despair (including alcohol use disorder, alcohol and opioid poisoning, suicide, liver disease, and cirrhosis). Remaining COD were grouped into 'rest' (see Supplementary Table S1). Hierarchical exhaustive and mutually exclusive classifications of ICD-10 codes were based on those defined by the global burden of disease study [35].

Statistical analyses

Cause-specific death counts were converted to mortality rates using survey-weighted ACS data. Due to inconsistencies in population counts for the non-Hispanic 'other' group in 2020 and the difficulties in interpreting this heterogenous group, we excluded this group from the LE analyses (Supplementary Fig. S1).

LE at age 25 was calculated by year (2018–2021), education, race/ethnicity, and sex [36], assuming that most of the individuals had obtained their highest educational attainment by age 25 [6, 37, 38]. A period-based, cause-specific lifetable approach was used, assuming a synthetic cohort, where individuals experience age-specific mortality rates observed in that period. While this method approximates what would happen if a cohort experienced current mortality rates throughout life, it does not reflect 'actual' mortality experiences, especially for periods of significant change such as the COVID-19 pandemic. Twenty-five years were added to all results so that the resulting LE can be interpreted as the age an adult can expect to live until, assuming constant mortality rates over time and given that they have already survived until the age of 25. Next, we used the continuous change decomposition method proposed by Horiuchi *et al.* [39], based on a line integral model, to quantify annual cause-specific contributions to changes in LE from one year to the next (2019–2020 and 2020–2021), stratified by race/ethnicity, education, and sex. In brief, the method estimates the

contribution of each COD to changes in LE by updating mortality rates for one COD at a time.

Statistical analyses were performed in R version 4.2.1 [40].

Sensitivity analyses

We performed two sensitivity analyses to account for differences in population counts by data source (ACS, Current Population Surveys [CPS]) and disruptions in the assessment in 2020, see Supplementary Data.

Results

In 2020, over 3.3 million adults died in the US. COVID-19 was the fourth leading COD (350 223 deaths) following diseases of the heart (694 893 deaths), cancers (599 888 deaths), and other NCDs (361 646 deaths). By 2021, COVID-19 became the third leading COD (415 202 deaths), after heart diseases (693 325 deaths) and cancers (602 753 deaths). Complete mortality counts are shown in Supplementary Table S2.

Overall changes in life expectancy

LE at age 25 was relatively stable from 2018 to 2019 but dropped for all subgroups between 2019 and 2020 (Fig. 1). In 2020, the largest losses were among low-education Hispanic men (−6.0 years; from 81.0 to 75.0 years) and women (−5.4 years; from 87.9 to 82.5 years), followed by Black men with middle or low education and Black women with low education (−4.4 to −4.7 years). High-education White and Hispanic men, and White women with middle and high education, lost about one year. By 2021, LE remained similar to or lower than 2020 levels for most subgroups except Black men with high education.

Changes in socioeconomic and racial and ethnic inequalities in life expectancy

In 2019, 25-year-old low-education Black men had the lowest LE at 71.0 years, compared to 87.2 years for high-education Hispanic men and 83.9 years for high-education White men—a gap of up to 16 years. For women, low-education Black women had a LE of 78.5, while high-education Hispanic and White women had LEs of 91.7 and 87.9 years, respectively—a gap of up to 13 years. These gaps widened substantially during COVID-19 to up to 20 years for men (Black, low education: 66.6 vs. Hispanic, high education: 86.2) and 15 years for women (Black, low education: 73.9 vs. Hispanic, high education: 88.9) in 2020.

To examine how these socioeconomic and racial-ethnic LE disparities evolved during COVID-19, we analysed educational (Fig. 2, Supplementary Table S3) and racial-ethnic LE disparities (Fig. 3, Supplementary Table S4). 'Educational disparities' in LE (high minus low education) were amplified across all racial-ethnic subgroups from 2019 to 2020, with the largest increases among Hispanic men and women, with LE gaps between high and low education increasing by 5.1 years (from 6.1 to 11.2 years) and 2.5 years (from 3.8 to 6.3 years), respectively. Among Black men and women educational LE gaps increased by 1.0 and 1.9 years, and for White men and women by 1.6 and 1.3 years, respectively. Between 2020 and 2021, educational gaps further widened for Black and White men and women, especially for Black men (an additional 2.1 years). Among Hispanic men, educational gaps in

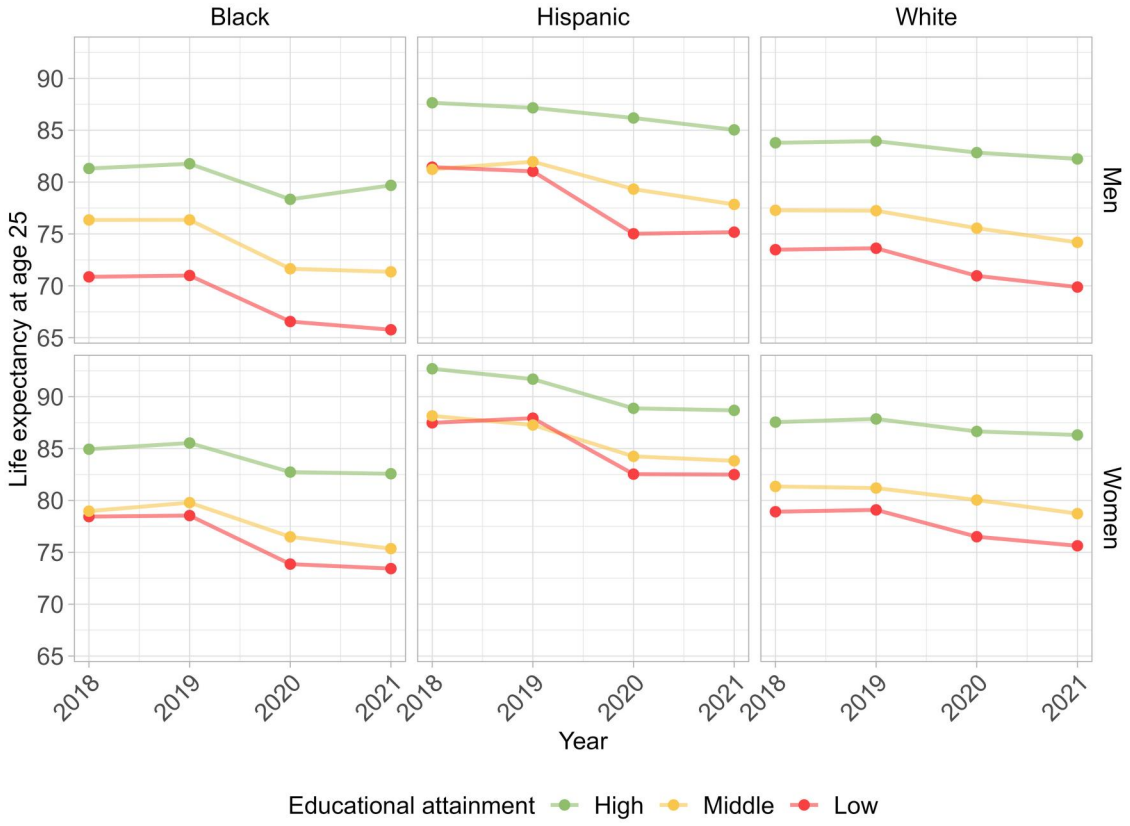

Figure 1. Life expectancy at age 25 by education, race/ethnicity, and sex in the United States between 2018 and 2021. Low education = high school degree or less; middle education = some college but no Bachelor's degree; and high education = college degree or more. The race/ethnicity category 'other' was omitted.

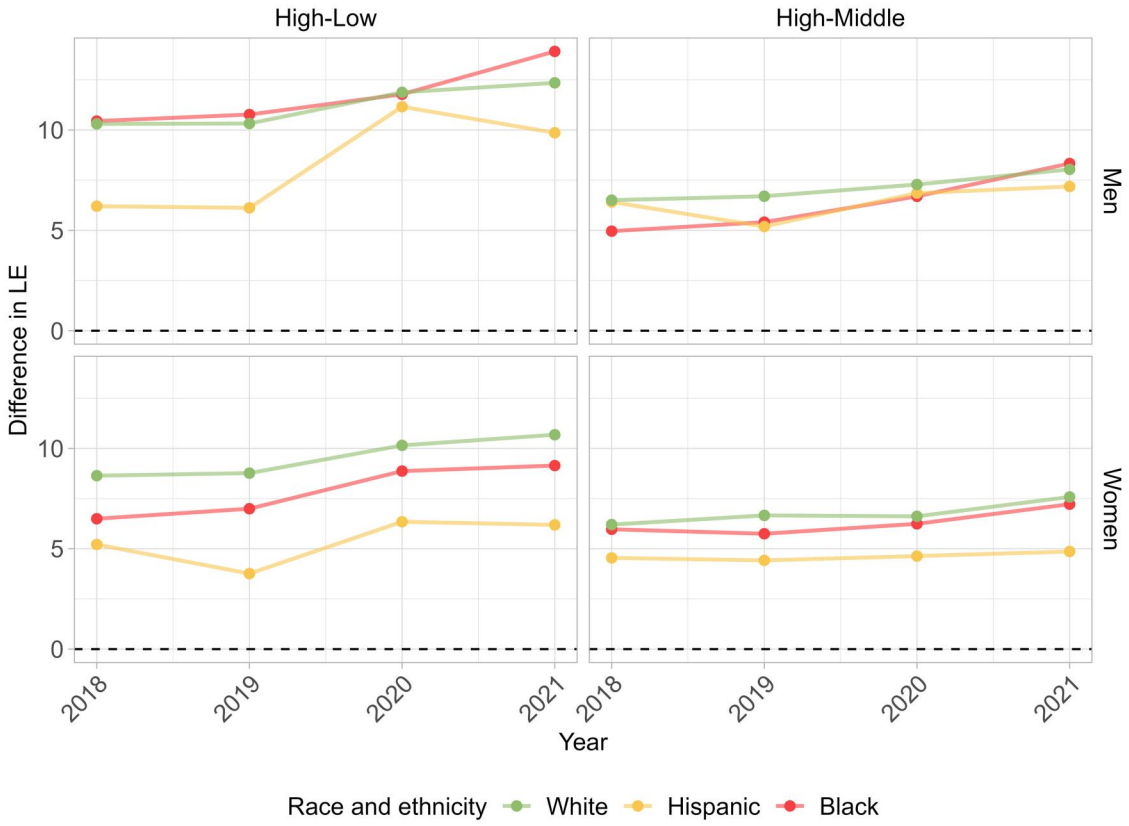

Figure 2. Trends in educational differences in years of life expectancy (LE) at age 25 by race/ethnicity and sex in the United States between 2018 and 2021. High-Low: LE in high education minus LE in low education; High-Middle: LE in high education minus LE in middle education. Low education = high school degree or less; middle education = some college but no Bachelor's degree; and high education = college degree or more. Dashed line indicates no educational differences in LE. The race/ethnicity category 'other' was omitted.

LE began to narrow again but remained above pre-pandemic levels.

'Racial and ethnic' LE gaps widened from 2019 to 2020 (Fig. 3). White-Black disparities 'grew' by 1.6–3.0 years, varying by sex and education, before narrowing slightly in 2021. Hispanic-White gaps 'shrank' by up to 3.3 years for men and 2.8 years for women, due to a larger LE declines among Hispanic than White individuals (see Fig. 1). In 2021, Hispanic-White gaps slightly increased again for men with low education and women with low and middle education.

Decomposition of cause-specific contributions to changes in life expectancy

LE losses due to COVID-19 varied considerably at the intersections of education, race/ethnicity, and sex (Fig. 4; Supplementary Fig. S2 for results on detailed causes of death). From 2019 to 2020, irrespective of the level of education, White men and women lost about a year of LE at age 25 due to COVID-19 (40%–100% of their total losses), while Black men and women lost around two years due to COVID-19 (44%–66%). Hispanic men and women saw the largest COVID-19-related losses, ranging from −2.4 (Hispanic women, high education, 86%) to −4.7 years (Hispanic men, low education, 78%). Those with low education faced greater LE losses overall than those with higher education (Supplementary Table S5).

Importantly, education played a key role in non-COVID-19 mortality. Nearly all LE losses among high-education Hispanic and White individuals were due to COVID-19, whereas for low-education White and Black individuals, COVID-19 only accounted for 40%–47% of losses. In each subgroup of race/ethnicity and sex, individuals with low education lost −1.3 to −2.5 years (22%–60%) from combined non-COVID-19 causes. Black men with low and medium education and Black women with low education lost the most LE from non-COVID-19 causes (−2.5 to −2.6 years, 53%–56%). For White men and women with low education, the loss from non-COVID-19 causes was −1.6 (60%) and −1.5 (58%) years, respectively.

In 2020, some of the largest LE losses from non-COVID-19 causes were due to NCDs (i.e. cardiovascular diseases, cancers, other NCDs), with a clear socioeconomic gradient observable for all racial-ethnic groups. Cardiovascular diseases in particular disproportionately affected Black individuals, where men and women with low education and men with middle education each lost over −0.5 years (12%–14%). To provide an example from the detailed causes of death (Supplementary Fig. S2), diseases of the heart alone were related to losses of −0.5 to −0.6 years (12%–14%) among Black men with low and middle education and Black women with low education. An example from the category of other NCDs is diabetes, which affected Black men with middle education (−0.2 years, 5%) and Black women with low education the most (−0.2 years, 4%). By contrast, diseases of the heart and diabetes accounted for smaller LE losses or gains for White men and women, especially of higher education. Unintentional injuries also contributed large LE losses, with −0.6 years (14%) among Black men with low and −0.4 years

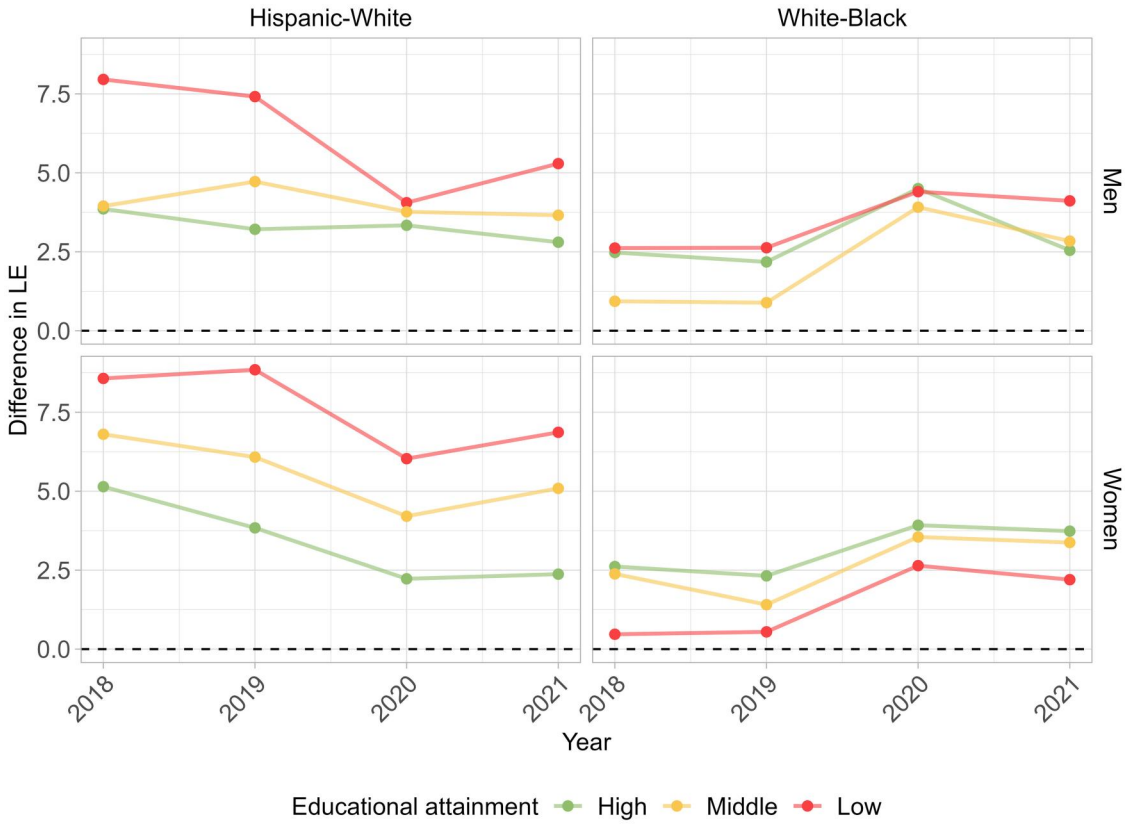

Figure 3. Trends in racial and ethnic differences in years of life expectancy at age 25 by education and sex in the United States between 2018 and 2021. Hispanic-White: LE in Hispanic minus LE in White; White-Black: LE in White minus LE in Black. Low education = high school degree or less; middle education = some college but no Bachelor's degree; and high education = college degree or more. Dashed line indicates no racial and ethnic differences in LE. The race/ethnicity category 'other' was omitted.

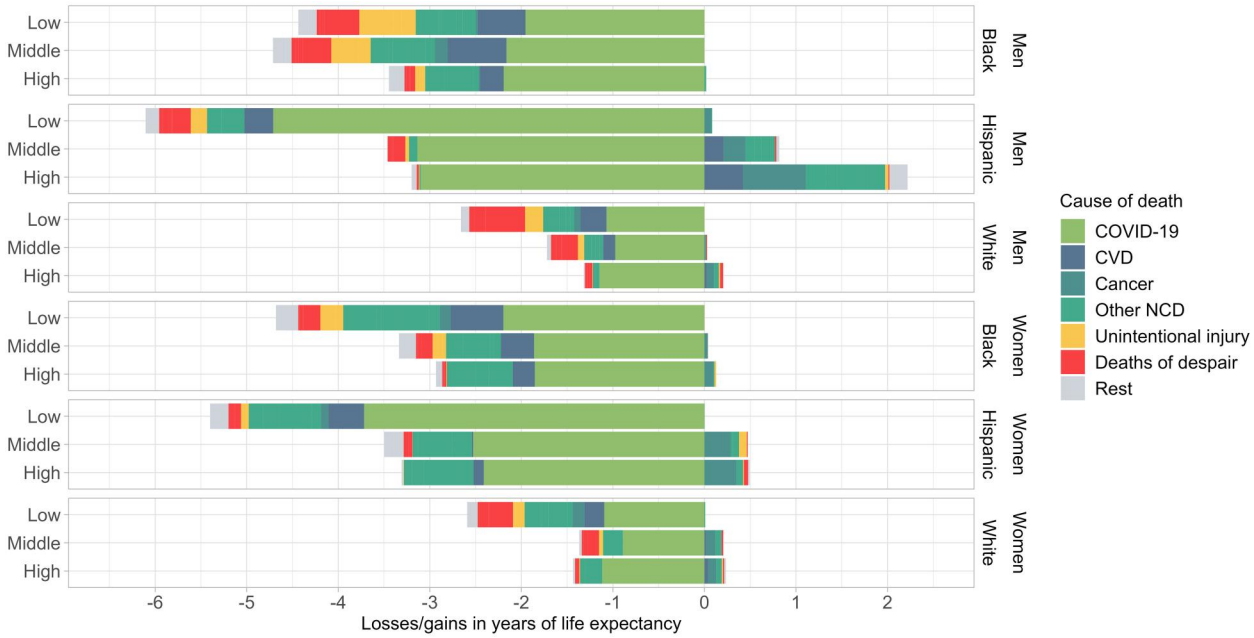

Figure 4. Cause-specific contributions (i.e. gains/losses, in years) to annual changes in years of life expectancy (LE) at age 25 from 2019 to 2020 among United States adult men and women by race and ethnicity and education. Underlying population estimates are based on the American Community Survey. Low education = high school degree or less; middle education = some college but no Bachelor's degree; and high education = college degree or more. The race and ethnicity category 'other' was omitted. CVD = cardiovascular diseases; NCD = non-communicable diseases.

(9%) among Black men with middle education. Several other subgroups, all of which had low education, lost between −0.1 and −0.3 years to unintentional injuries (2%–5%). Deaths of despair heavily impacted White and Black men with low education and Black men with middle education, with LE losses of −0.4 to −0.6 years (9%–23%). Low-education White women lost −0.4 years (15%) due to deaths of despair. By contrast, Hispanic men and women with high and middle education experienced some LE 'gains' in 2020, mainly from NCDs. For example, Hispanic men with high education experienced LE gains of about two years from NCDs.

From 2020 to 2021, LE changes were much smaller compared to 2019 to 2020 (Supplementary Fig. S3 and Table S6; Supplementary Fig. S4 for results on detailed causes of death). COVID-19 deaths contributed the largest losses in White men with middle (−0.9 years, 63%) and low education (−0.8 years, 72%), and Hispanic men with middle education (−0.8 years, 56%). Among women, COVID-19-related losses were highest in White women with low and middle education (−0.6 years, 74% and 45%, respectively), Black and Hispanic women with middle education (−0.5 years, 44% and 114%, respectively), and Black with low education (−0.4 years, 88%). LE gains were observed in Black men with high education and Hispanic men with low education due to fewer COVID-19 deaths relative to 2020. For non-COVID-19 causes, deaths of despair were the leading contributors to LE losses across population subgroups, followed by unintentional injuries. Deaths of despair were the largest contributor to LE losses in 2021 among Black men with low (−0.4 years, 46%) and middle education (−0.2 years, 54%), Black women with low and middle education (−0.2 years each, 51% and 13%, respectively), and White and Hispanic men with low education (−0.2 years each, 20% and −120%, respectively). NCDs contributed to both LE losses and gains in population subgroups.

Sensitivity analyses

The results of the sensitivity analyses are shown in Supplementary Data. LE at age 25 was slightly lower when using CPS than when using ACS among individuals with low education, while it was slightly higher when using CPS than using ACS among individuals with middle and high education. The largest difference was found among Hispanic women with high education, for whom the LE was 88.9 based on the ACS source and it was 95.8 based on the CPS source. However, the socioeconomic gradient in LE losses and the pattern of losses from COVID-19 and other causes of deaths did not fundamentally differ across data sources.

Discussion

COVID-19 is a syndemic which has intersected with and worsened preexisting social, economic, and health disparities, creating a 'perfect storm' of negative factors leading to disastrous outcomes [41]. This study demonstrates its particularly devastating impact on those with low SES and for racial and ethnic minoritized populations. It highlights how COVID-19 has exacerbated inequalities in LE not only directly through COVID-19 deaths but also indirectly through 'other' COD. For some groups, LE losses in 2020 were driven primarily by increases in mortality from 'other' causes, reflecting the syndemic interplay of COVID-19, endemic diseases, and social determinants of health.

By quantifying LE 'differences' between specific groups over time, we demonstrate how racial and ethnic, and socioeconomic inequalities have worsened during the COVID-19 period. The particularly large increases in educational inequalities in LE in 2020 amongst Hispanic men and women appear to be driven by a combination of LE 'losses' amongst those with low education, particularly from direct COVID-19 deaths, and LE 'gains' in LE amongst those with high education for causes such as cardiovascular diseases, cancer, and other NCDs.

The high rate of COVID-19 mortality amongst Hispanics with low education reflects how socio-environmental factors exacerbated disease vulnerability, clustering, and interactions for specific groups. For example, Black and Hispanic communities, and those with low SES, faced barriers to social distancing such as reliance on public transit, higher housing density [42–44], and low-wage, 'essential worker' occupations [44–46], necessitating decisions between financial risk and viral exposure. Additionally, these populations typically have fewer resources, such as savings and health insurance [24, 25], hindering their ability to seek treatment for COVID-19 and preexisting health conditions.

LE 'gains' from some COD among Hispanic individuals with high and middle education are surprising and may be explained by: a high contribution from COVID-19 mortality among this population, meaning individuals who would have otherwise died from other causes dying from COVID-19 instead; differential misclassification of the underlying COD by race/ethnicity leading to over-estimation of COVID-19 deaths and under-reporting of deaths from other causes; or protective factors amongst this group that mitigated against the negative impacts of the syndemic on NCDs. However, we found insufficient evidence to support any one hypothesis and further research is needed to contextualize these findings.

Our decomposition of changes in LE by COD has identified important intersectional differences in 'how' mortality outcomes were shaped differently for different groups. For example, in contrast to Hispanic individuals with low education discussed above, LE reductions among Black men with middle or low education were largely attributable to other causes, particularly NCDs and unintentional injuries. Increased losses of LE from deaths of despair were also notably higher amongst groups with low education. Even before the COVID-19 pandemic, an epidemic of deaths of despair was taking hold in the US [47], with deaths largely concentrated in individuals with a low SES [33, 48]. The rise in LE losses from these causes in 2020 and 2021 was likely exacerbated by heightened psychological distress, particularly among those with lower educational attainment. COVID-19 introduced additional stressors, such as social isolation, fear of illness, economic strain, and uncertainty, worsening mental health for the general population, especially among low-SES individuals [49, 50].

Revisiting the components of a syndemic helps explain these findings. Firstly, COVID-19, NCDs and their risk factors, and deaths of despair *cluster* within the same populations, particularly Black and Hispanic communities and those with low SES [18–21, 51, 52]. Secondly, 'adverse biological interactions' exist between COVID-19 and NCDs [18], e.g. individuals with diabetes mellitus are more susceptible to COVID-19, while COVID-19 and medications used to treat

it can adversely affect glucose control, exacerbating diabetes [53]. Finally, 'socio-environmental factors' exacerbated disease vulnerability, clustering, and interactions for specific groups, as aforementioned.

Limitations

Our study has several limitations. First, since LE is calculated under a hypothetical scenario where mortality conditions in a given year persisted indefinitely, using it during shocks as the COVID-19 pandemic overstates the long-term impacts of the COVID-19 pandemic because those conditions are temporary and not reflective of future trends. Second, COVID-19 disrupted routine data collection, resulting in lower data quality, particularly in 2020. Prior research noted potential underreporting or misclassification of COVID-19 deaths to other COD in the NVSS, particularly among young adults and racial and ethnic minority groups [54]. In the Census 2020, certain population groups were undercounted (Black: 3.3%, Hispanic: 5.0%), while non-Hispanic White were overcounted (1.6%) [55]. These irregularities were mirrored in the ACS trends (Supplementary Fig. S1), which were accounted for in several sensitivity analyses. Third, the differences in LE seen for Hispanic men and women may partly reflect changes in death recording locations, rather than true changes. Pre-pandemic, it was shown that Hispanic migrants returning to Mexico generally have worse health than those remaining in the US [56]. Travel restrictions during the pandemic may have forced less healthy Hispanic individuals to remain (and in some cases die) in the US, changing where their deaths were recorded. Given return migrants to Mexico appear to have generally lower levels of education [57], and the impact of this may have been more noticeable in groups with low education.

Conclusion

COVID-19 widened preexisting socioeconomic and racial and ethnic inequalities in LE in the US, disproportionally affecting Hispanic and Black individuals with low education. Changes in LE stemmed from both direct COVID-19 deaths and increased mortality from other causes, reflecting COVID-19's syndemic nature as well as potential misclassifications. Addressing the underlying social, political, economic, and environmental factors driving the clustering and interaction of diseases for these populations is essential to counter these trends.

Ethics approval

This study was exempt from ethics review as it involved the analysis of publicly available secondary data and did not include any identifiable personal information.

Author contributions

Conceptualization: C.P.; Data curation: C.P., Y.Y., C.B., C. K.; Formal analysis: C.P., Y.Z.; Funding acquisition: C.P.; Methodology: C.P.; Visualization: C.P., Y.Z.; Writing—original draft: C.P., S.B.; Writing—reviewing & editing: all authors. Guarantor: C.P.

Supplementary data

Supplementary data is available at *IJE* online.

Conflict of interest: Dr. Kerr has received funding and travel support from the National Alcoholic Beverage Control Association (NABCA). Dr. Kerr has been paid as an expert witness regarding cases on alcohol policy issues retained by the Attorney General's Offices of the US states of Indiana and Illinois under arrangements where half of the cost was paid by organizations representing wine and spirits distributors in those states. All other authors have no conflict to declare.

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Data availability

All data used in the analyses were retrieved from public use datafiles. Our R scripts are publicly available under the following DOI: 10.6084/m9.figshare.28920935.

Use of artificial intelligence (AI) tools

No AI tools were used in conducting this study or writing the manuscript.

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