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The impact of childhood obesity on human capital in high-income countries: A systematic review

Alexa Blair Segal | **M. Carmen Huerta** | **Elisabetta Aurino** | **Franco Sassi**

Centre for Health Economics & Policy Innovation, Imperial College Business School, London, UK

Correspondence
Alexa Blair Segal, Centre for Health Economics & Policy Innovation, Imperial College Business School, Imperial College London, South Kensington Campus, SW7 2AZ London, UK.
Email: a.segal17@imperial.ac.uk

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Summary

Current evidence of the impact of childhood obesity on human capital development does not point in a consistent direction, and its interpretation is challenging. We carried out a systematic review of studies from high-income countries that used robust causal inference approaches to assess the impact of childhood overweight and obesity on outcomes typically linked to human capital development in economics. Global Health, Medline and EconLit were used to search for peer-reviewed papers. Three reviewers independently assessed study quality using the Newcastle-Ottawa Scale. Nineteen papers representing 22 studies met the inclusion criteria. Included studies were categorized based on three components of human capital: cognitive performance (n = 18), measured through test scores; educational attainment (n = 3), through grade progression and college completion; and labour market outcomes (n = 1), through wages. We find that childhood overweight and obesity hinder education outcomes, with effects mostly observed at older ages of exposure measurement (12+ years). Girls with overweight and obesity experienced larger negative effects and more often than boys. Future research should elucidate the pathways through which childhood obesity impacts human capital development, to support policies that may mitigate those impacts, thus averting social costs that are currently widespread, increasing and unaccounted for.

KEYWORDS

children and adolescents, human capital, obesity and overweight, systematic review

Abbreviations: AddHealth, US National Longitudinal Study of Adolescent Health; ALSPAC, UK Avon Longitudinal Study of Parents and Children; BMI, body mass index; CCA, comprehensive cognitive ability; CDC, Centre for Disease Control; CP, cognitive performance; DFE, dynamic fixed effects; DID, differences-in-differences; DXA, dual-energy X-ray absorptiometry; EA, educational achievement; ECLS-K, US Early Childhood Longitudinal Study, Kindergarten Class; FE, fixed effects; GATOR, Georgetown Adolescent Tobacco Research; GCM, growth curve models; GPA, grade point average; IOTF, International Obesity Task Force; IV, instrumental variables; KABC, Kaufman's Assessment Battery for Children; KS2, Key Stage 2 exam; KS3, Key Stage 3 exam; LMO, labour market outcome; LSAC, Longitudinal Survey of Australian Children; MLR, multivariate linear regression; MR, Mendelian randomization; NAPLAN, National Assessment Program Literacy and Numeracy exam; NLSY79, US National Longitudinal Survey of Youth 1979; NLSY97, US National Longitudinal Survey of Youth 1997; NOS, Newcastle-Ottawa Quality Assessment Form for Cohort Studies; OLS, ordinary least squares; PIAT, Peabody Individual Achievement Test; PPVT-R, Peabody Picture Vocabulary Test-Revised; PSM, propensity score matching; QLSCD, Quebec Longitudinal Study of Child Development; TEPS, Taiwan Education Panel Survey data; WISC-R, Wechsler Intelligence Scale for Children-Revised.

1 | INTRODUCTION

The global number of children with overweight or obesity has increased more than tenfold, from 11 million in 1975 to 124 million in 2016.[1] Evidence suggests that the longer a person has obesity, the larger the excess morbidity and costs are in adulthood, with costs ranging from increased healthcare costs to reduced productivity in the workforce as a result of increased sick days.[2] The global economic cost of obesity was estimated at $2.0 trillion US dollars in 2012, including the loss of productive life, direct healthcare expenditures and the investments to lessen these costs.[3] For example, employees with obesity are likely to be less productive at work due to increased health issues (i.e. arthritis, fatigue, or depression) and related work absenteeism.[2] Investment costs include expenditures on public health programmes as well as commercial weight management and fitness products and plans.[4] Further, obesity is an intergenerational phenomenon, meaning that obesity is likely transmitted from parent to child in a cyclic manner.[5] Given the life-course and intergenerational effects of childhood overweight and obesity, governments around the world have been devising policies and programmes to curb the childhood obesity epidemic.[6,7]

The case for government action to address childhood obesity would be further strengthened by robust evidence of its detrimental impacts on key social and economic outcomes such as education, employment or social capital, helping also to identify the best periods in which to intervene.[8] For ease of reading, we use the term 'childhood obesity' to refer to childhood and adolescent overweight and obesity (unless otherwise specified). Therefore, childhood obesity refers to anyone who is under 18 years of age and has an age- and sex-adjusted body mass index (BMI) *z*-score greater than or equal to the 85th percentile. While there is reasonably established evidence of the effects of childhood obesity on later health, evidence of the effects on social and economic outcomes is mixed and largely relies on studies that use less robust inference designs.[2,9–12]

Human capital is defined as 'the agency of human beings - through skill and knowledge as well as effort—in augmenting production possibilities.'[13 (p1959)] Human capital development is represented by a cumulative production function framework that combines cognitive and noncognitive inputs.[14–17] We have conceived this systematic review in line with a human capital theoretical approach in which an individuals' social and economic outcomes are the result of a dynamic and cumulative process of human capital development over his or her life-course.[18] As such, human capital includes skills and knowledge that give an individual returns—be it economically or socially—that allow them to be valuable in a workforce. A recent review concludes that early childhood circumstances can have relatively substantial negative impacts in adulthood, though impacts are heterogeneous due to differences in the child's inputs and family environments.[19] In addition, research finds strong evidence of a negative association between childhood health and later socioeconomic outcomes (mainly educational and employment-related outcomes), but evidence on the long-term effects, especially those of childhood obesity, remains undeveloped.[19–21] This is because estimating the effects of childhood obesity on human capital outcomes is complicated by a range of potential observed and unobserved confounders and mediators (i.e., socioeconomic status, parents' education, obesogenic environments, and genetic makeup), issues of reverse causation and uncertainty over the time lag between exposure and outcome.[20,21]

There is no clear consensus on the effect of childhood obesity on human capital. While previous literature suggests a negative relationship between adult obesity and labour market outcomes, the magnitude and statistical significance of this relationship depends on the gender and race or ethnicity of participants.[22–26] Literature on the effects of childhood obesity and educational outcomes suggests a negative relationship—though again, results vary according to gender, race/ethnicity, age and location of participants.[27–30] Some studies report no significant effects as well—demonstrating that the results are sensitive to the specific context of the data and model specifications used.[31–33] A study by Palermo and Dowd[34] used fixed effect models to investigate the effect of childhood obesity on cognitive and noncognitive outcomes and concluded that obesity in children and adolescents negatively affects noncognitive but not cognitive outcomes.[34] In 2011, a review by Suhrcke and de Paz Nieves[20] concluded that obesity and overweight are negatively related with negative educational outcomes though the 'evidence is contradictory concerning the gender-differentiated effect of these risk factors, and endogeneity issues also persist as obstacles in the estimation of causality.'[20 (p13)] In the latter review, only a limited number of studies used longitudinal data and implemented econometric methods to control for the biases produced both by confounding and reverse causality. However, in most reviews, the majority of included studies were cross-sectional, meaning that they are prone to confounding, including from reverse causation. For example, the review by Caird et al[35] concluded that increased weight in childhood and adolescence was weakly related with decreased educational attainment. However, 3 years later, a different review concluded that the relationship between obesity and academic achievement was not clear—except for adolescent females, who experienced a negative relationship.[36] This, in addition to the inclusion of cross-sectional studies, means that the evidence on the effect of obesity on educational outcomes remains contradictory.

Another reason for the uncertainty of the effect of childhood obesity on human capital is that no previous reviews have investigated the impact on human capital as a multifaceted concept, including education and labour market outcomes, as the majority of the literature reviews on the topic included only educational outcomes. The review by Gondek et al[37] is the most similar to this review, though they focused on the social and economic impacts associated with any type of ill health (not just obesity) at any life stage (not just childhood). The authors also included studies that used longitudinal data, regardless of whether they used a rigorous framework or not.[37] Altogether, past reviews have shown inconsistent findings on the association between childhood obesity and subsequent outcomes depending on the age of exposure and methods used. We thus contribute to the literature by focusing on childhood obesity only and its effect on multiple human capital outcomes, and by only reporting results from studies with a sound inferential design, as defined in Section 2.2.

The aim of this review is to assess whether existing studies provide evidence that childhood obesity has an impact on human capital on middle and late childhood (including adolescence) or adulthood. The review is designed to summarize the evidence generated by studies based on causal inference approaches and assess the strength of this evidence. By focusing on the effect of obesity in childhood on future outcomes, our review sheds light into the effect of obesity on later human capital.

2 | METHODS

2.1 | Search strategy

Global Health via Ovid, Medline via Ovid and EconLit were used to search for studies. The search strategy was adapted for each database. Google Scholar and the National Bureau of Economic Research were used to ensure no relevant studies had been omitted. The search was last updated on 14 February 2019.

A comprehensive search strategy was utilized to ensure all relevant literature were screened (Figure S1). A combination of medical subject headings terms and 'Explode' terms, as well as free-text, were employed to increase search sensitivity. The main search strategy had two main components—one for childhood obesity and the other for human capital outcomes. First, to search for literature on childhood obesity, the medical subject headings terms, subheadings and main terms for 'child' or 'infant' or 'paediatric' or 'adolescent' and 'obesity' were searched, as well as the free text. Other keywords for weight status like 'overweight' are included in the medical subject headings term obesity (Figure S1). To capture human capital and the variety of associated social and economic outcomes of interest, we included keywords for cognitive performance (test scores, literacy, mathematics), educational achievement (highest academic qualification, educational attainment), labour market outcomes (employment, unemployment, wages, employment disability), social capital (social relationships, interpersonal relationships, partnership status, social support, trust) and, finally, social participation (social engagement, voluntary work, membership to organization, voting). We note that while our original research question included social participation and social capital outcomes, no relevant papers were found. Thus, our review is solely on human capital outcomes.

2.2 | Selection criteria

The searches were restricted to peer-reviewed studies written in English published after 1980. Conference papers, dissertations, meta-analyses and working papers were excluded. Included studies had to assess the impact of childhood obesity on a wide set of outcomes including educational and labour market outcomes. Examples of educational outcomes can include standardized cognitive ability scores, grade point averages (GPAs) and subject scores. Cognitive performance is most often evaluated with ability tests[38]; it is a measure of function in various cognitive domains (i.e., memory or executive function) and a proxy for educational achievement. We acknowledge that outcomes such as GPA may reflect more than an objective measurement of a student's cognitive performance, most likely capturing a child's classroom homework and participation scores, which could be subjected to teacher's bias. However, because scores like GPA are mostly an average of exam scores, and hence reported as a continuous scale, we group GPA as a cognitive performance outcome.

Educational attainment measures a students' academic progression or attainment. It includes domains such as high-school completion, higher-education completion, formal qualifications and degrees. Labour market outcomes considered here include aspects associated with participation and productivity, such as employment, unemployment, work-related absences and wages or salaries. Included studies had to use longitudinal data and use methods that accounted for both reverse causation and unobserved confounding. Since our review draws on literature from different fields, such as economics, epidemiology and medical, we did not want to limit our search strategy by including specific methods. The aforementioned fields have different definitions of 'causal' inference methods, which is why we did not prespecify the types of inferential methods we were going to include in our search strategy (see Table S1). Instead, our goal with our search criteria was to be able to capture any quantitative study that met the rest of our criteria of having longitudinal data and took steps to deal with issues of reverse causation and unobserved confounding. Observational studies are excluded from the main analyses of this review as they lack robust and rigorous inference methods to control for the endogeneity of childhood obesity.

Only studies assessing childhood overweight and/or obesity, in children up to age 18 years, were eligible for inclusion. We excluded secondary analyses of datasets from low- and middle-income countries because of the rapidly transforming relationship between prevalence of childhood and adolescent overweight and obesity and the socioeconomic environment that the child is exposed. For example, a 2013 review concluded that the relationship between educational attainment and obesity is impacted by country's economic development level; high-income countries exhibit an inverse relationship, whereas lower-income countries exhibit a positive association between obesity and educational achievement.[39] We used the World Bank list of economies, last updated in June 2018, to identify the income level of the countries under study.[40] Further, studies of children with other comorbid conditions, such as type 2 diabetes, were excluded to avoid the possibility of confounding the relationships of interest. The complete inclusion and exclusion criteria are provided in Table S1.

2.3 | Data extraction

Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses, Figure 1 presents the flow diagram of the screening process.[41] The complete search strategy produced 4,168 articles after removing duplicates. After screening by title, this was narrowed down to 600 articles. Two reviewers screened title and abstracts for relevance. Concurrence was 90%. Disagreements were resolved through discussion and, when needed, by a third reviewer. After screening by abstract and title, two reviewers completed a full-text review on the 268 remaining articles. The interrater agreement for full-text review was assessed using Cohen's kappa (κ = 0.76). After full-text screening, 19 papers were selected for this review, representing 22 studies.

2.4 | Data synthesis

The 22 studies included in this review were categorized by the three distinct outcomes of interest: cognitive performance, educational attainment and labour market outcomes. Therefore, this manuscript is focusing on education and employment outcomes only. It was not possible to undertake a formal meta-analysis because of a high degree of heterogeneity in study populations, study designs and outcomes.

2.5 | Risk of bias

Three reviewers (ABS, MCH and EA) worked independently to appraise the methodological quality and risk of bias information for all papers using a modified Newcastle-Ottawa Quality Assessment Form for Cohort Studies (NOS); disagreements were resolved through discussion between the three reviewers.[42]

The NOS has been recommended to assess the methodological quality of nonrandomized studies of the effect of interventions in systematic reviews using three domains—selection, comparability and outcome.[42] We followed the authors of various systematic reviews on obesity who modified the NOS.[37,43–45] The original NOS has four criteria in the 'Selection' domain and a comparability domain, but because our exposed and unexposed participants come from the same cohort, we did not include these domains. Instead, we follow Gondek et al[37] and create an 'Adjustment' domain to evaluate the study's control of confounders, which consists of two questions. We did not assess papers according to the 'Design' domain because all our studies were longitudinal and therefore this domain was inapplicable for our review.[37]

Studies were awarded a maximum of three points in the selection domain, based on the representativeness of the sample (internal validity), ascertainment of exposure (child weight status measured) and evidence that the outcome of interest was controlled for at baseline (i.e., academic achievement balanced between participants with and without overweight or obesity at baseline). Papers were awarded a maximum of two points for adjustment. One of these points could be

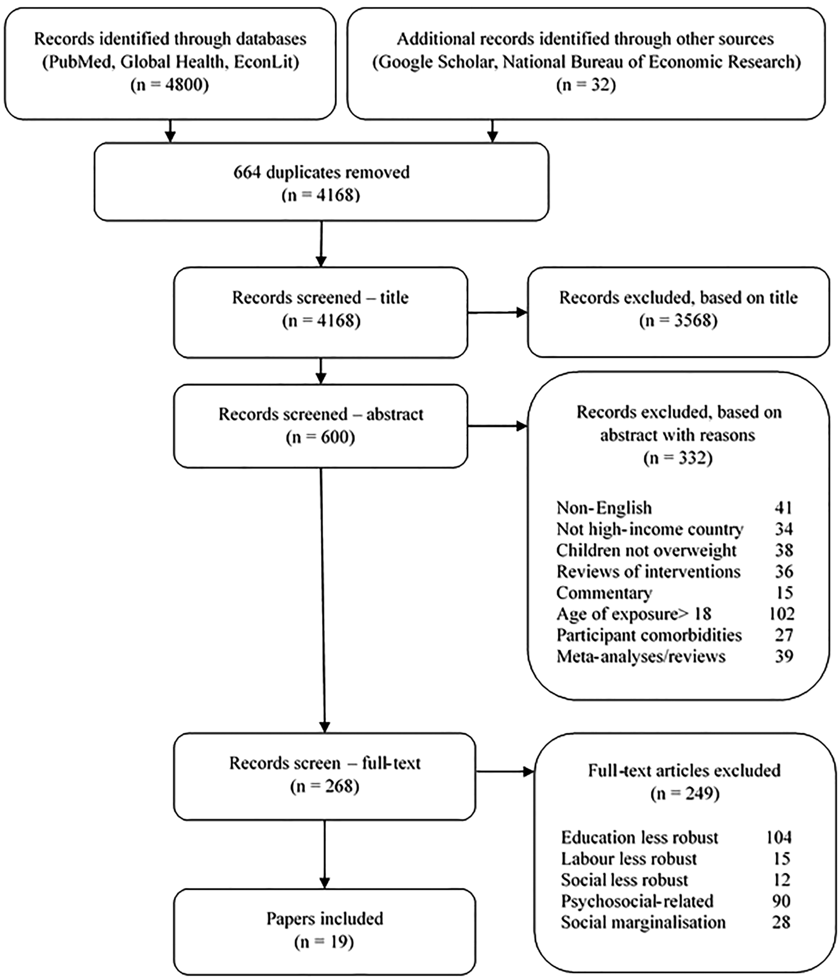

FIGURE 1 Preferred reporting items for systematic reviews and meta-analyses flow chart of study selection

earned if the study controlled for the main confounders associated with childhood overweight and respective outcomes (sex, gender, socioeconomic status, parent *or* home inputs and intelligence). The other point was given if the paper included covariates that could mediate the relationship of interest, such as mental health and self-efficacy (in addition to including basic covariates such as age, gender, socioeconomic status).[46–48] Finally, a paper could earn two points in the outcome domain: one for ascertainment of outcome (objective measurements of cognitive performance or educational attainment) and one for reporting the sample attrition rates and/or discussing the implications of follow up rates.

3 | RESULTS

This review is based on 19 eligible papers that equate to 22 relevant studies as three papers cover more than one cohort or outcome. Of these, there are 18 studies on cognitive performance, three on educational attainment and one on labour market outcomes. Table 1 provides an overview of the results. Key summary information for each paper with the respective reference, including methods, data, location, outcome and exposure details, growth reference chart used, overall results and risk of bias, is presented in Table 2. The sample characteristics of each study including sample size, sex distribution, age at baseline and follow up, obesity prevalence at baseline, period considered, number of data waves used in analyses and results categorized by outcome are summarized in Table 3. We present point estimates from the studies that report significant findings in Table S2. We include point estimates only where studies report significant findings for their most robust models—even when this model is not the author's preferred model.

Most papers are based on US data (*n* = 12) with the rest based in Taiwan (*n* = 2), the United Kingdom (*n* = 2), Australia (n = 2) and Canada (*n* = 1). Data were mostly from seven large cohorts: the US National Longitudinal Study of Adolescent Health (AddHealth) (*n* = 4), followed by the US Early Childhood Longitudinal Study, Kindergarten Class (ECLS-K) (*n* = 3), US National Longitudinal Survey of Youth 79 and 97 (NLSY79 and NLSY97) (*n* = 2 each), the Longitudinal Survey of Australian Children (LSAC) (*n* = 2) and the UK Avon Longitudinal Study of Parents and Children (ALSPAC) (*n* = 2). The other four papers conducted secondary analyses on data from the following: Quebec Longitudinal Study of Child Development (QLSCD); Georgetown Adolescent Tobacco Research (GATOR); Taichung City elementary school district, Taiwan; Taiwan Education Panel Survey data (TEPS) (*n* = 1 for each).

3.1 | Results by outcome

3.1.1 | Cognitive performance

Eighteen studies from 17 papers evaluated the effect of childhood obesity (measured at different ages ranging from four through

TABLE 1 Overview of included studies

	Studies *n* (number of papers)[a]	Significant negative effects *n* (%)[b]
Outcomes		
CP	18 (17)	8 (44%)
EA	3 (3)	2 (67%)
LMO	1 (1)	1 (100%)
Risk of bias		
High	5 (4)	5 (100%)
Medium	14 (12)	4 (29%)
Low	3 (3)	2 (67%)
Datasets		
NLSY79 (US)	3 (2)	1 (33%)
NLSY97 (US)	3 (2)	1 (33%)
AddHealth (US)	5 (4)	4 (80%)
ECLS-K (US)	3	1 (33%)
GATOR (US)	1	1 (100%)
ALSPAC (UK)	2	0 (0%)
LSAC (Australia)	2	2 (100%)
QLSCD (Canada)	1	0 (0%)
Taichung City elementary school district (Taiwan)	1	0 (0%)
TEPS (Taiwan)	1	1 (100%)
Analytic methods		
DID	3 (2)	0 (0%)
DFEs	2	2 (100%)
GCMs	3	1 (33%)
IVs	12 (10)	8 (67%)
MR	4	1 (25%)
PSM	2	2 (100%)
Exposure measurement		
Objectively measured	11	(45%)
Self-reported	7 (6)	(83%)
Both self-report and measured	4 (2)	(0%)

Abbreviations: ALSPAC, UK Avon Longitudinal Study of Parents and Children; CP, cognitive performance; DFE, dynamic fixed effects; DID, difference-in-difference; EA, educational achievement; ECLS-K, US Early Childhood Longitudinal Study, Kindergarten Class; GATOR, Georgetown Adolescent Tobacco Research; GCM, growth curve models; IV, instrumental variables; LMO, labour market outcome; LSAC, Longitudinal Survey of Australian Children; MR, Mendelian randomization; NLSY79, US National Longitudinal Survey of Youth 1979; PSM, propensity score matching; QLSCD, Quebec Longitudinal Study of Child Development; TEPS, Taiwan Education Panel Survey data.

[a]Number of studies per row item; number in parentheses represents the number of papers represented.

[b]Number of studies that reported significant negative results per the respective row item (percentage of significant negative studies of all studies per row).

TABLE 2 Summary of included papers

First author (year)	Outcome[c]	Stratification	Model overview[d]	Dataset[e]	Country	Outcome[f]	Exposure[g]	Growth reference used[h]	Overall result[i]	Risk of bias
Afzal (2015)[62,H]	CP	Sex	DID; MLR	NLSY79	US	PPVT-R, PIAT, WISC-R[i] (PIAT maths and reading)	BMI[j] always OB and never OB, became OB and non-OB	CDC 2000	1) N.S. 2) N.S.	Medium
Averett (2010)[28]	CP	Sex and race (White, Black)	IV; FE, OLS	NLSY79	US	PIAT[i] (maths and reading)	OV[j]	CDC 2000	Neg.	Medium
Bisset (2013)[49]	CP	N/A	GCM; MLR	QLSCD	Canada	KABC[i] (avg. of reading, writing, maths)	BMI[i] OV	CDC 2000 IOTF 2000	N.S.	Medium
Black (2015)[50]	CP	Sex and school grade	DFE and IV; OLS, VA, FE	LSAC	Australia	NAPLAN[i] (maths and reading)	BMIZ[i] OB	IOTF 2000	Neg.	Low
Capogrossi (2013)[51]	CP	Sex, race and school grade (White, 'minority')	IV, IVQR; OLS	ECLS-K	US	ECLS-K[i] (maths and reading)	BMIZ[i] OV, OB	CDC 2009	N.S.	Medium
Chen (2012)[52]	CP	N/A	GCM; MLR	Taichung City primary district	Taiwan	Mean scores[i] (language, maths, science, history)	BMIZ[i] always OV/OB, became OV/OB	IOTF 2000	N.S.	Medium
Ding (2009)[17]	CP	Sex	MR; OLS	GATOR	US	High school GPA[k]	OB[k]	BMI > 30	Neg.	High
Fletcher (2011)[53]	CP	Twin/sibling	MR, MR with FE; OLS, FE	AddHealth	US	High school GPA[i] PIAT[i] (verbal)	OB[k]	CDC 2000	N.S.	Medium
Kaestner (2009)[31,h]	1) CP 2) EA	Sex and age	IV; MLR	NLSY97	US	1) PIAT[i] (maths and reading) 2) Grade attainment[i]	OB[j]	CDC 2000	1) N.S. 2) N.S.	Medium
Kenney (2015)[54]	CP	Sex	DID	ECLS-K	US	ECLS-K[i] (maths and reading)	BMI[i] BMIZ, OV, OB	CDC 2000	N.S.	Low
Kranjac[55] (2015)	CP	N/A	GCM	ECLS-K	US	ECLS-K maths[i]	BMI[i] OV and became OV, OB and became OB	CDC 2000	Neg.	Low
Lu (2014)[56]	CP	Sex	PSM; OLS	TEPS	Taiwan	CCA[i]	BMI[i] OV	Taiwan Dep. of Health	Neg.	Medium
Okunade (2009)[57]	`EA	Sex and race (White, Black, Hispanic, Asian)	PSM; OLS	AddHealth	US	On-time graduation[j] (of high school)	OV[k] OB	Canadian Centre for Health Info.	Neg.	High
Pinkston (2017)[58]	LMO	Sex and race (White only)	DFE; OLS	NLSY97	US	Hourly wage[k] (following full-time labour market entry)	OV[k] OB, severely OB	CDC 2000	Neg.	Medium

TABLE 2 (Continued)

First author (year)	Outcome[c]	Stratification	Model overview[d]	Dataset[e]	Country	Outcome[f]	Exposure[g]	Growth reference used[h]	Overall result[i]	Risk of bias
Sabia (2007)[59]	CP	Sex and race (White, 'non-White')	IV; OLS, FE	AddHealth	US	High school GPA[k]	BMI[k] body weight (lbs), per lbs OV, OV, OB	CDC 2000	Neg.	High
Sabia (2015)[60,h]	1) CP 2) EA	Sex	IV; OLS, FE	AddHealth	US	1) High school GPA[i] (maths, English, language) 2) High school diploma[i] completed 4-year college[k]	Body weight (lbs)[k] BMI, OV and OB (both)	CDC 2000	1) Neg. 2) Neg.	High
Shi (2018)[61]	CP	School grade	IV; OLS	LSAC	Australia	NAPLAN[i] (reading, writing, maths, grammar, spelling)	BMI[i] OV, OB	Australian Department of Health	Neg.	Medium
von Hinke (2012)[33]	CP	Race (White only)	IV and MR; OLS, FE	ALSPAC	UK	KS3[i]	Fat mass[i] (using DXA)	Sample-standard. Percentiles	N.S.	Medium
von Hinke (2016)[32]	CP	Race (White only) and sex (robust)	MR; OLS	ALSPAC	UK	KS3[i]	Fat mass[i] (using DXA)	Sample-standard. Percentiles	N.S.	Medium

[a]CP, cognitive performance; EA, educational attainment; LMO, labour market outcomes.
[b]Methods before the semicolon are the causal methods: DID, difference-in-difference; DFE, dynamic fixed effects; GCM, growth curve models; IV, instrumental variables; MR, Mendelian randomization; PSM, propensity score matching. Less rigorous models: FE, fixed effects; MLR, multivariate linear regression; OLS, ordinary least squares.
[c]AddHealth, US National Longitudinal Study of Adolescent Health; ALSPAC, UK Avon Longitudinal Study of Parents and Children; ECLS-K, US Early Childhood Longitudinal Study, Kindergarten Class; GATOR, Georgetown Adolescent Tobacco Research; LSAC, Longitudinal Survey of Australian Children; NLSY79 US National Longitudinal Survey of Youth 1979; NLSY97, US National Longitudinal Survey of Youth 1997; QLSCD, Quebec Longitudinal Study of Child Development; Taichung City elementary district, Taiwan; TEPS, Taiwan Education Panel Survey data.
[d]CCA, comprehensive cognitive ability; ECLS-K, Early Childhood Longitudinal Study-Kindergarten Class Assessment; GPA, grade point average; KABC, Kaufman's Assessment Battery for Children; KS3, Key stage 3 exam; NAPLAN, National Assessment Program-Literacy and Numeracy; PIAT, Peabody Individual Achievement Test; PPVT-R, Peabody Picture Vocabulary Test-Revised; WISC-R, Wechsler Intelligence Scale for Children-Revised.
[e]BMI, body mass index; OB, obese; OV, overweight; DXA, dual-energy X-ray absorptiometry.
[f]CDC 2000/2009, Centre for Disease Control growth charts; IOTF 2000, International Obesity Task Force growth charts.
[g]Neg., significant negative findings; N.S., nonsignificant.
[h]These papers have two studies within them; two sample populations[62]; two different outcomes.[31,60]
[i]Objective measure.
[j]Mix of objective and subjective measures.
[k]Subjective measures.

14 years) on cognitive performance outcomes (measured at different ages ranging from eight through 18 years). Among such studies, 12 disaggregated analyses by sex, with the majority of the studies (*n* = 7) reporting a significant negative effect. Studies that reported significant negative effects were more likely either to detect these negative effects for female participants only, or to find stronger effects for females. Eleven studies presented analyses with and without sex-stratification; of these studies, only four detected significant negative results—again with half of these negative results only, or stronger, for females.

Studies used a range of outcome variables to measure cognitive performance, including standardized cognition exams or scores in school academic subjects (*n* = 16) or high school GPAs (*n* = 4). The majority of cognitive performance studies evaluated the effect on students' standardized cognition or national exams, including the Peabody Individual Achievement Test (PIAT), Key Stage exam (KS2 and KS3) and the National Assessment Program Literacy and Numeracy exam (NAPLAN). Studies that used standardized national exam scores most commonly report the effects for maths (83%; *n* = 15) and reading subjects (78%; *n* = 14). Of the 16 cognitive performance studies that used various standardized exam scores, five reported significant negative effects; and of these, two reported stronger negative effects for females versus males. Interestingly, of the 11 studies that reported no significant effects, we see that for all but one of these studies, the exposure age used was under 12 years of age. Again, the study by Fletcher and Lehrer[53] was the exception. Most of the studies (three of four) that used GPA as the outcome of interest reported significant negative results, with larger negative effects for White females and non-White males. Again, we see that age at exposure may be the driving force in this relationship, as all of these studies used older ages for exposure (ranging from ages 12 to 17 years).

Studies used a variety of exposure weight variables, including continuous BMI or weight in pounds to categorical overweight or obese categories, to identify the effect of childhood obesity on cognitive performance. Two studies only used continuous weight and reported no significant effects. Eight studies ran their analyses with both continuous and categorical exposures. In general, these studies report significant negative findings. The evidence suggests that a dose-response relationship exists between BMI and cognitive performance, especially for females (see Table S2).

Finally, age of exposure measurement is an important predictor in the relationship between childhood obesity and later cognition. Two thirds of the studies that looked at weight status when measured at younger ages of exposure (11 years of age or younger) detected non-significant effects. Conversely, all but one study (the study by Fletcher and Lehrer[53]) that looked at weight status when measured at older ages of exposure (12-17 years of age) observed insignificant negative effects. Thus, it seems there is clearer evidence of a negative effect on cognitive performance for children with obesity measured in adolescence, while there is weaker evidence of such a negative effect when obesity is measured for younger children. We explain the nuances of this finding in the Discussion.

3.1.2 | Educational attainment

Three studies investigated the impact of childhood obesity (measured at ages ranging from 9 through 17 years) on later educational attainment (measured at ages ranging from 12 through 30 years). As per cognitive performance, both exposure and outcome variables varied across studies, which makes it difficult to compare the effect sizes of associations. Two studies measured exposure at age 12 years: one included both continuous (pounds and BMI) and categorical (overweight including obese) outcomes, reporting significant negative effects for both males and females, but stronger for the latter.[60] The other used overweight and obese categories separately, finding significant negative effects only for White and Asian-American females with overweight and obesity.[57] The third educational attainment study used obesity measured at age 7 to 9 years as the exposure of interest, without differentiating by sex, and did not find significant effects.[31] Educational attainment measures vary across studies, including students' grade retention, university completion and probability of graduating from high school, which may contribute to the heterogeneity of findings.

3.1.3 | Labour market outcomes

Only one study included in this review measured the effect of childhood obesity on later labour market outcomes.[58] This study used data from US individuals who were 12 to 16 years of age at exposure, reporting significant negative effects for both males and females. However, while White females face wage penalties before reaching the overweight threshold, White males only face wage penalties for having severe obesity as children. More than 40% of White females faced a wage penalty from their weight in adolescence, compared with only 8% of White males.[58]

Although we were only able to include one study in this outcome category, this study appears to support the wider findings that significant negative effects are generally observed when the exposure is measured at older ages (age 12 to 16 years) and that the effects are more pronounced for females. Our findings conform to the results of the review by Gondek et al,[37] who find that poor physical health in early adulthood tends to be related with unemployment. While excluded from our review due to the age of weight exposure, other studies find that individuals with overweight and obesity are subject to lowered labour market outcomes.[22,23,63] While this corroborates our findings regarding age of exposure measurement, we cannot rule out the fact that duration of overweight or obesity may be the driver of this finding rather than the age of exposure.

3.2 | Geographical differences

Of the 15 studies (12 papers) based in the United States, eight reported significant negative effects ranging all of the aforementioned outcomes. All of the seven studies that reported a nonsignificant

TABLE 3 Sample characteristics of studies

First author (year)		Sample size	Sex of sample (% female)	Period considered (waves of data used)	Sample obesity prevalence at baseline[a]	Sample age at baseline (years)	Sample age at outcome (years)	Result[b]
Cognitive performance	Afzal (2015)[c]	672	50%	1988-1994 (three waves)	7% OB	5 (2-8)	14	N.S.
	Afzal (2015)[d]	1,991	49%	1994-2000 (three waves)	12% OB	5.3 (2-8)	14	N.S.
	Averett (2010)	20,856 child-years (only White/Black)	48%	1986-2002 (not specified)	Not specified	6	13	Negative
	Bisset (2013)	1,959	50%	2002-2005 (four waves)	7% OV	4	~8	N.S.
	Black (2015)[61]	4,983	49%	2004-2012 (three waves)	17%[e] OV 6% OB	8/9	12/13	Negative
	Capogrossi (2013)	21,260 observations	49%	1998/9-2006/7 (three waves)	12% OV 13% OB	~6	~14	N.S.
	Chen (2012)	409	48%	2002-2008 (six waves)	17%[e] (OV and OB)	~5 (first graders)	~12 (seventh graders)	N.S.
	Ding (2009)	893	53%	1999-2003 (two or 3 waves)[f]	8% OB	16	18	Negative (F only)
	Fletcher (2011)	1,684 biological siblings	51%	1994/1995-2001/2002 (three waves)	7% OB	12	18	N.S.
	Kaestner (2009)[c]	~2,200	50%	1986-2004 (three waves)	15%[e] OV 17%[e] OB	9 (7-9)	12	N.S.
	Kenney (2015)	3,362	49%	1998/1999-2006/2007 (two waves)	21% OV 20% OB	~10 (fifth graders)	13.8 (eighth graders)	N.S.
	Kranjac (2015)	5,034	45%	1998-2007 (seven waves)	21% OV 19% OB	5.7	14	Negative ([g] older, OB>OV)
	Lu (2014)	8,690	51%	2001-2003 (two waves)	17% OV	~12 (seventh graders)	~15 (ninth graders)	Negative ([g]for F)
	Sabia (2007)	5,129	52%	1994-1996 (two waves)	15% OV 10% OB	14	17	Negative (F White and M non-White only)
	Sabia (2015)[c]	11,822	52%	1994/1995-2007/2008 (two waves)	12% OV 9% OB	14	17	Negative (F only)
	Shi (2018)	~5,000	49%	2007/2008-2012/2013 (three waves)	29% OV 8% OB	8	13	Negative ([g] older, OB>OV)
	von Hinke (2012)	3,001	51%	2000/2001-2005/2006 (three waves)	10%-18%[e] OV	11	14	N.S.
	von Hinke (2016)	4,844	51%	2002/2003-2005/2006 (two waves)	10%-18%[e] OV	11	14	N.S.

(Continues)

TABLE 3 (Continued)

First author (year)		Sample size	Sex of sample (% female)	Period considered (waves of data used)	Sample obesity prevalence at baseline[a]	Sample age at baseline (years)	Sample age at outcome (years)	Result[b]
Educational attn.	Kaestner (2009)[d]	~2,200	50%	1986-2004 (three waves)	33% OV	9 (7-9)	12	N.S.
	Okunade (2009)	8,388	53%	1994/1995-2001/2002 (three waves)	18% OV 10% OB	12	17	Negative ([g] for non-African American F)
	Sabia (2015)[d]	11,822	52%	1994/1995-2007/2008 (two waves)	12% OV 9% OB	14-17	27-30	Negative
LMO	Pinkston (2017)	2,533 (White)	42%	1996-2009 (three waves)	31% (OV and OB)	12-16	24-30	Negative ([g]F)

Abbreviation, LMO, labour market outcome.
[a]OV, overweight; OB, obese.
[b]N.S., nonsignificant findings; F, females; M, males.
[c]First study in paper.
[d]Second study in paper.
[e]Study author did not provide information, so it is approximated by review author if it was possible.
[f]Author contacted study author for information.
[g]Stronger association for specified group.

effect used weight measurements at a young age of exposure (under age 12). Of the US papers that reported significant negative effects, we again observe that females with overweight and obesity experience stronger negative effects compared to their male peers. The two studies based in the United Kingdom and the study based in Canada conclude nonsignificant effects of obesity on cognitive performance outcomes. All three of these studies examined the effect when weight status was measured at younger ages of exposure (ranging between 4 and 11 years of age), and only one of these studies disaggregates by sex.

Both studies based in Taiwan examined the effect on cognitive performance but reached different conclusions. While the study that reported nonsignificant results used exposure when measured at a younger age (age 5 to 6 years), the study that reported a significant negative effect used exposure when measured at an older age (age 12 years). The evidence from Taiwan contributes to our hypothesis that age of exposure measurement is an important predictor of future negative outcomes. Lastly, the two studies based in Australia, both of which used the LSAC dataset, reported significant negative effects of childhood obesity on cognitive performance. Different to most other countries, they obtained these results for exposure measured at younger ages. However, a separate study concluded that 66% of LSAC children with obesity at ages 4 or 5 years remained with obesity at age 10 or 11 years,[64] suggesting that children with obesity in middle childhood were likely to remain with obesity in adolescence and therefore experience the negative effects.

3.3 | Sex as an effect modifier

Existing evidence consistently shows that sex may be an effect modifier in the relationship between childhood obesity and education outcomes. Specifically, females experienced larger negative effects compared with their male peers. Sixteen studies disaggregated their analyses by sex. Of these 16 studies, seven studies reported nonsignificant effects, though all of these studies had used a young age of exposure that could explain these nonsignificant effects. More importantly, nine of the sex-stratified studies reported significant effects, and of these, the majority (n = 6) reported that this effect was either more significant, larger or only observed by females.

Five studies presented their results with and without disaggregating by sex, which allowed us to further investigate the effect of sex. Three of these studies reported no significant effect, though all three used young exposure ages. The other two of these studies reported significant negative effects for the full sample then found differential negative effects when disaggregating by sex. The study by Ding et al[17] found an overall significant negative relationship between obesity and GPA, but when disaggregated by sex, the effect completely attenuated for males while the significant negative effect became slightly larger for females. The other study that reported significant negative results found that the effect became larger and more significant for females when disaggregating by sex.[56] Our findings regarding the differential impact of sex on the relationship of interest is in line with the findings of the reviews by Cohen et al[39] and Martin et al.[36] Martin et al[36] concluded that the association between childhood obesity and academic achievement varied by sex, age and school subject. This review showed a significantly negative association between overweight and maths achievement for adolescent females, but not for younger females and males.[36] The review Cohen et al[39] found a negative association between educational attainment and obesity, with 'stronger social patterning among women.'[39 (p989)]

3.4 | Differences by race and ethnicity

Seventeen papers used data from countries with racially diverse populations—including the United States, United Kingdom, Australia and Canada—and 14 of these addressed potential differences by race and ethnicity. For example, Pinkston[58] decided to limit analyses to only White females and males due to issues of sample size, explaining that non-White respondents had too many missing observations. Von Hinke et al[32,33] limited their sample to only White participants due to the restrictions specified for the use of genetic instruments. Referred to as population stratification, this practice is used to avoid bias from the fact that certain races systematically inherit different frequencies of genetic variants.[32,33] Four studies based on US data presented results stratified by race and/or ethnicity, while the rest merely controlled for those characteristics. Averett and Stifel[28] reported significant negative effects for both White and African American children with overweight. Okunade et al[57] reported results separately by ethnicity and race and found that overweight and obesity were negatively related with on-time high school graduation, specifically for White, Asian American and Hispanic American females, but not for African American females, with no significant effects observed in males regardless of race. Finally, Sabia[59] found a significant negative association between obesity and GPA, but only for White females and non-White males. Only Capogrossi and You[51] reported no significant effects for either White or minority students. It is clear that the magnitude of effects changed according to participants' race, sex and subject exam. However, this evidence is limited and hardly generalizable.

3.5 | Study quality (risk of bias)

Results of the risk of bias assessment are available in Table S3. Two of the three low-risk papers reported significant negative results. Low-risk papers used objective procedures to secure child exposure weight and outcome measurements. These papers also verified low risk of selection bias by showing that the outcome difference was not present at baseline or was appropriately controlled for (i.e., by regressing the child's baseline cognitive performance score in the equation). Additionally, these papers appropriately adjusted for both known and 'extra' confounders related to children's mental health. Low-risk papers also recognized and appropriately addressed issues of cohort attrition bias. As all three of these low-risk papers used weight exposure as measured between about ages 5 and 10 years, future studies should investigate using appropriate methods with later ages of exposure.

Most of the evidence in this review was at medium risk of bias, representing 12 papers, covering 14 studies. Of these studies, only four reported a significant negative effect. However, nine out of the 10 studies with a nonsignificant effect examined weight status at a young age of exposure, using ages as young as age 4 years. Studies of medium risk were less likely to have ascertained objective measurements of child weight, controlled for or demonstrated that the outcome of interest was not present at baseline, controlled for the extra confounders, and were more likely to have not controlled for attrition bias.

Finally, four of the papers, representing five studies on both cognitive performance and educational attainment outcomes, were classified with a high risk of bias. This was mainly due to the subjective measurements of exposure and/or outcome variables and because the respective outcome was not adequately controlled for at baseline. All five of these studies reported results disaggregated by sex; three of the studies report significant effects only for [White] females with the overweight and/or obesity, not their male counterparts. The other two high-risk studies concluded that there are significant negative effects for both male and females, though one of these studies only concludes that non-White males experience these negative effects.[59]

4 | DISCUSSION

To our knowledge, this is the first systematic review of the effects of childhood obesity human capital focusing on studies adopting a robust causal inference design. This review also uses the NOS to measure potential risk of bias of the included studies. The review reveals a number of key features of the relationship between childhood obesity and the outcomes of interest, which largely explain the heterogeneity of findings of previous studies and reviews on the subject. These features are discussed in the remainder of this section.

4.1 | Effect of age at exposure measurement

When accounting for age at exposure measurement, the literature assessed in this review shows a relatively clear pattern: the older the child was when exposure was measured, the more likely they were to experience detrimental impacts later in life. Individuals with overweight or obesity measured at age 12 years and older generally experienced significant negative effects, while children with overweight or obesity at younger ages had less often detrimental consequences on their human capital outcomes (mostly education) later on. The only exception to this trend is the study by Fletcher and Lehrer set in the United States.[53] This study first documented that overweight at age 12 years was significantly related with negative cognitive performance at age 18 years. However, when the genetic instruments were added, results became insignificant.[53] A potential explanation of why this study ultimately reported insignificant effects may be attributed to the small sample size (ranging from 469 to 1,684 per analysis); an extremely large sample size is needed for proper use of a genetic instruments, otherwise the analysis lacks the power to detect significant effects.[53,65]

The finding that children with overweight or obesity measured at older ages, such as in early- or mid-adolescence, are more likely to suffer negative effects also resulted from some of the previous reviews.[39] One possible explanation is that the impacts of childhood obesity are cumulative over time; a hypothesis also embodied in what is commonly referred to as the 'life course' approach.[66] In a cumulative impact perspective, studies measuring outcomes in adolescence have better chances of capturing a meaningful effect than those focusing on younger children, as detrimental effects of overweight and obesity might have built up over a longer period of time. Another potential explanation arising from this review may support a 'critical age' hypothesis, in which effects are more likely to be experienced at a particular point in time during childhood—here at around age 12 years.[67] Critical period hypotheses generally state that there are certain stages in people's lives that have specific impacts on the rest of their lives.[67] In context, age 12 years may be a critical period for students' educational experiences, and having obesity at this period may set them on a negative pathway. Ultimately, we do not have enough evidence to support either theory. Based on our reviewed papers, we can only say that when exposure to overweight or obesity is measured during the adolescent years, we are more likely to find significant relationships with later life outcomes. However, as the papers do not report whether the child started to experience overweight and obesity around the time of exposure measurement (thus adolescence), or if they had lived with overweight and obesity for longer spells, we cannot disentangle whether it is adolescence as a critical age of exposure, or the cumulative experience of obesity during, which drive detrimental effects, later in life.

4.2 | Measurement of childhood obesity

Childhood obesity was defined, standardized and measured in a variety of ways. This made it difficult to compare point estimates and probably contributed to the inconsistency in findings. Children's exposure weight status should be defined consistently with the hypothesized pathways leading to the outcome of interest. For example, if children with obesity experience reduced educational attainment through stigmatization and discrimination in the classroom, a dichotomous obese/nonobese variable may be a more consistent predictor than continuous BMI. The results of this review suggest that a correct and consistent specification of the exposure weight status variables for childhood overweight and obesity is essential in elucidating robust effects.

Further, though the majority of studies used the Centre for Disease Control's (CDC) 2000 or the International Obesity Task Force's (IOTF) 2000 definition for childhood overweight and obesity, not all did. Overweight is defined as having an age- and sex-adjusted BMI *z*-score greater than or equal to the 85th percentile; obesity is when it is greater than or equal to the 95th percentile. The main consequence of using different definitions is that the prevalence of obesity and overweight in the sample would change depending on the definition chosen. With a more restrictive definition, only the children with the higher BMI would be considered overweight or obese, meaning that the effects are more likely to be ascertained, but the reduced prevalence makes the results less likely to be statistically significant. However, when the prevalence of obesity is low, regardless of the definition used, those who have obesity may be more stigmatized, which may contribute to explaining why studies detected significant effects even with relatively low obesity rates, such as 6% in the study by Black et al[50] and 8% in Ding et al.[17] Many studies and reviews overlook the prevalence of obesity at exposure, but considering the sample's baseline obesity prevalence is important in interpreting results.

4.3 | Generational differences

Generational differences between cohorts are a cause of heterogeneity across studies. There is a 42-year gap in the year of birth of some of the cohort members in the reviewed studies. Not only does the prevalence of childhood obesity change, but also the social norms and expectations placed on cohort members' outcomes (e.g., educational attainment). As a result, the relationship between exposure and outcome of interest may be significantly different for a child in the NLSY79 cohort, born in 1957, compared with a child from the ECLS-K or ALSPAC cohorts, born in the early 1990s.

4.4 | Theoretical frameworks

The studies assessed in this review relied on different theoretical frameworks, and most were not informed by any frameworks, which likely contributed to inconsistencies in methods and results. Kaestner and Grossman[31] used a standard economic model of child quality and concluded that childhood obesity was not significantly associated with cognitive performance nor educational attainment outcomes, instrumenting children's weight status with lagged weight to obtain a model consistent with their production function. They argued that other studies reached different conclusions because those studies 'used empirical specifications that were not consistent with common theoretical formulations of the education (human capital) production function.'[31 (p660)]

5 | CONCLUSION

This systematic literature review provides evidence that childhood obesity can negatively affect human capital development. This evidence is disproportionately based on US data, educational outcomes and entirely on data from high-income countries. Our evidence is also limited by the type of study outcomes we found, which may mean our results are not generalizable for all dimensions of human capital.

Although this review includes only studies adopting robust causal inference approaches, limitations and risk of bias remain widespread in the evidence reviewed.

Only about half of the cognitive performance studies reported significant negative effects. However, a number of clear trends emerge when disaggregating and comparing findings within and across studies. Detrimental effects of childhood obesity on education are mostly apparent when obesity is measured and assessed in adolescence, and they affect girls most strongly and consistently.

The priority for future research is now to elucidate the pathways that may explain the above findings. There is also a need for robust studies that have follow-up measurements later in life. Is adolescence a critical time for obesity to produce its negative effects on education and future employment? Or, does disadvantage cumulate over time and become detectable only in adolescence? And, what makes girls most vulnerable to the effects of obesity? Is it inner characteristics interacting synergistically with obesity, or features of the environment that create pressures hindering social capital development? These are some of the questions that need to be answered in order to translate the findings of this review into policies that may mitigate the impacts of obesity in childhood and prevent disadvantage from occurring in the first place, thus averting social costs that are currently widespread, increasing and unaccounted for.

CONFLICTS OF INTEREST

None of the authors have anything to declare.

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AUTHOR CONTRIBUTIONS

FS, MCH and ABS conceptualized the study. ABS developed and implemented the search strategy, abstract screening, full text review, data extraction and data analysis. MCH and EA conducted the double reviewing and contributed to the development of the Risk of Bias assessment. ABS, MCH and FS contributed to the writing and editing of the manuscript. FS provided oversight on all aspects of the review.

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ORCID

Alexa Blair Segal https://orcid.org/0000-0003-3051-4347
M. Carmen Huerta https://orcid.org/0000-0002-7700-3993
Elisabetta Aurino https://orcid.org/0000-0001-5814-2704
Franco Sassi https://orcid.org/0000-0001-9773-2117

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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