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# Social induction dynamics of the causal social $R_0$ on clinical weight loss: Randomized trial evidence of social propagation from Amman, Jordan

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## Abstract

An unresolved debate persists in public health, concerning a distinction between communicable diseases that are causally linked to microbes or viruses and "non-communicable diseases" that are not. We offer evidence in support of the view that public health categories need rethinking; that more precise and granular ones are needed; and that "non-communicable" and "non-infectious" can be tautological misnomers. The scientific literature suggests an underlying socially "infectious" phenomenon and we put forth a fundamental Social Contagion Hypothesis: that social networks constitute a propagating force; that this force can be quantified in socio-biological terms; and that it can be leveraged for public health. Yet a major question still remains unanswered: if weight loss, like biological pathogens, cascades through host populations, and can be mitigated through interventions targeting risky social behaviors, then can a social reproductive number (Social $R_0$) for metabolic and cardiovascular disease be estimated and modulated? We present the first known evidence from a randomized trial, demonstrating the epidemiological dynamics of socially infectious diseases. We find that health propagation can achieve epidemic proportions (including changes in weight, $1<R_0=1.3$, $p<0.01$); and that public health systems can intervene to modify the $R_0$ value thereby potentially managing, preventing, or reversing social infections at epidemic scale (yielding population-averaged *comparative* $R_0$ ratios, $244<R_c<368$, $p<0.01$, for weight change). To facilitate adoption of the methodologies, macros and code for use with various statistical software packages are included. The results indicate that the social induction of health interventions are not only possible, but that propagation can both be isolated causally via trial design and be quantified over time. This bodes enormous promise for developing and quantifying future self-sustaining public health interventions.

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## Author summary

Public health debate persists concerning the distinction between communicable diseases caused by microbes or viruses and "non-communicable diseases" that are not. We offer evidence that more precise and granular public health categories are needed; and that "non-communicable" and "non-infectious" can be tautological misnomers. The literature suggests an underlying socially "infectious" phenomenon and we hypothesize that social networks constitute a propagating force which can be quantified in socio-biological terms and leveraged for public health. But if weight loss, like biological pathogens, cascades through host populations, and can be mitigated through interventions targeting risky social behaviors, then can a social reproductive number for metabolic and cardiovascular disease be estimated and modulated? We present the first known evidence from a randomized trial, demonstrating the epidemiological dynamics of socially infectious diseases. We find that health propagation can achieve epidemic proportions (including changes in weight); and that public health systems can intervene to potentially manage, prevent, or reverse social infections at epidemic scale. The results indicate that the social induction of health interventions are not only possible, but that propagation can be both isolated causally via trial design and quantified over time. This bodes enormous promise for developing and quantifying future self-sustaining public health interventions.

## Introduction

World economic order is undergoing a great transformation (Polanyi 2002 [1]) akin to that brought about by the first and even second and third industrial revolutions. Humanity now finds itself in the midst of a fourth (Schwab 2017 [2]), an age punctuated by artificially intelligent machines capable of replacing large quantities of intellectual and physical labor. In this new frontier we find what is for many perhaps axiomatic: complexity is ever increasing. What is needed now is not only new calculus and math (Bar-Yam 2019 [3]); we also need new variables to understand interdependencies, contingencies, and contradictions arising from this emerging epoch.

A central achievement of the first three industrial revolutions was to advance global public health in the face of ever-increasing complexity: peace, new energy sources, and technology made human civilizations wealthier, urbanized, and vastly more connected. At the same time, industrial processes also made networked societies sicker, necessitating the need for revolutions in medicine for the treatment of smallpox, typhus, and tuberculosis (Jackson 2003 [4]). From vaccines to sanitation and from germ-theory to the invention of the $R_0$ (Wu, Dordain & Bolden 2020 [5]), industrial transformation gave rise to the modern fields of epidemiology and public health.

Social complexity is increasing now at paces that decades ago would have been unthinkable, and a century ago, unimaginable. Meme stocks pumped up artificially by Reddit users demonstrates the power of social networks over the stock market (Smith 2021 [6]). Bank run cascades were induced by Twitter influencers resulting in the collapse of Silicon Valley and Signature Banks, a bailout of depositors, and the sale of UBS to its competitor (Cookson et al. 2023 [7]; FDIC 2023 [8]; Merced, Farrell & Sorkin 2023 [9]).

A marked feature of this industrial revolution is the race for meaningful new variables that can help modulate contagion effects between the biological and socio-economic worlds. The spread of vaccine misinformation on Facebook constitutes just one example: social

propagation dynamics in an online network meld into biological disease dynamics in the physical world before once again recombining with the digital world. "Social infectiousness" has, in a very real sense, become less of a metaphor and more about molecules.

The coupling of social and biological contagions have been observed to have both deleterious and salutary effects, exhibiting dynamics that are not present when these systems are "uncoupled" (Bauch & Bhattacharyya 2012 [10]; Bauch & Galvani 2013 [11]). An important meta-review, for example, suggested that increased life-saving anti-retroviral usage also drives riskier behavior (Crepaz, Hart & Marks 2004 [12]).

Disaggregating the social-biological causal pathways underlying disease propagation presents unique challenges that are important to public health (Williams et al. 2013 [13]). So-called Non-Communicable Diseases are estimated to cost $47 trillion dollars between 2010–2030 and comprise 74% of annual global mortality as of 2022 (Bloom et al. 2012 [14]). Yet 80% of the disease burden (NCD Alliance 2022 [15]) is driven by largely preventable and "socially infectious" dietary, lifestyle, and substance use (World Health Organization 2023a [16]) risk factors that flow through networks (Ding et al. 2024a [17]). Adding further complexity, socially infectious conditions often worsen the likelihood of severe outcomes from biologically transmitted infection, and vice versa (Bowe, Xie & Al-Aly 2022 [18]; Li et al. 2022 [19]; World Health Organization 2020 [20]).

It well known that many "non-communicable" diseases are spread through biological pathogens, demonstrating—from a biological perspective—that conventional public health binaries are anachronistic and in need of revisions: cancers, liver cirrhosis, multiple sclerosis, rheumatic heart disease, long covid, encephalitis, and post-polio syndrome are several prominent examples. Furthermore, a whole host of "non-communicable autoimmune diseases have been linked to viruses, including Grave's, SLE, arthritis, and type-1 diabetes mellitus. See Smatti et al. 2019 [21] for further discussion. In the post-COVID-19 world, advances in etiology will no doubt uncover many other examples. Yet public health binaries are also being challenged from a sociological perspective. Studies have shown observational social network propagation effects on biological phenomenon (Fowler & Christakis 2010 [22]). A debate with the WHO remains unresolved regarding the transmissibility of "non-communicable" conditions and the validity of conventional public health categories. See for example, Ackland, Choi, and Puska 2003 [23].

This paper clarifies and disaggregates social network causal effects on clinical weight loss. We demonstrate that a Social $R_0$ value quantifies social network forces, including those that reach epidemic proportions; that these forces can be modulated and optimized by public health institutions, and importantly, that they are causally linked to changes in human biology.

## Intervention

The Microclinic Social Network Program was designed as a gamified, win-win public health program with the aim of facilitating "Managed Coopetition." That is to say forces of social cooperation and competition were optimized by a trained facilitator towards the primary goal of weight loss. In the randomized controlled trial (RCT) treatment group, subjects were partitioned into micro-clusters ("microclinics") which effectively constitute coalitions jointly completing the program: within classroom and field settings (e.g., grocery stores for dietary lessons or the gym for those in exercise), instruction was provided to a class consisting of several disjoint (micro-)clusters, each typically including two or three subjects. Significant individual progress toward weight loss was rewarded with achievement recognition each session, fostering competition both within and between clusters.

Whereas it was hypothesized that cooperation within and between clusters could arise as subjects (a) develop additional social ties/interdependencies, (b) are motivated by the progress of others, and (c) explicitly or implicitly recognize that in order to achieve individual progress, it behooves them to "train" in coordination with their own cluster; a sort of rivalry or "friendly," cooperative competition which has been termed coopetition (Cherington 2008 [24]) emerges. This coopetition was hypothesized to create towards to top, i.e. a weight loss cascade. The full trial methods, including a consort diagram, has been documented elsewhere (Ding et al. 2024b [25]).

## Methodology

One novel extension of the Susceptible-Infected-Susceptible (SIS) compartmental model of mathematical epidemiology (Hethcote 1989 [26]), the SISa model (Hill et al. 2010 [27]), augments the former with an additional, spontaneous/'automatic' infection term:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI + gI - aS = -\frac{\mathrm{d}I}{\mathrm{d}t}$$

Here, $S$ ($I$) denotes the number of susceptible (infected) individuals at time $t$; a constant (large and well-mixed) population, $N = S+I$, is assumed; along with constant rates of transmission, recovery and spontaneous infection, respectively, $\beta$, $g$, and $a$. The authors estimate these rates, additionally assuming two kinds of infection ('content' and 'discontent') and allowing the possibility of superinfection directly between such states; epidemiologically modelling emotions in a social network determined via the Framingham study (Dawber 1980 [28]) which included administration of the CES-D exam (Radloff 1977 [29]) for classifying subjects' emotional states. We adapt their methodology to clinical, survey and social network data from an RCT conducted in Amman, Jordan. A major difficulty is the classification of subjects' states: For weight change, we naturally identify 'lost' ('gained') with 'content' ('discontent'); but we have no CES-D or other diagnostic criteria to distinguish an intermediate, 'neutral' state. As such, much of the analysis and results center on sensitivity and robustness analyses of corresponding thresholds; absolute values of change below (above) which subjects are classified as 'neutral' ('lost' or 'gained').

Due to the supplementary spontaneous infection which distinguishes the SISa model, the basic reproduction ratio (van den Driessche and Watmough 2008 [30]) does not exhibit the thresholding behaviour common to classical compartmental models including SIS. Nonetheless, it may be estimated as follows:

$$R_0 \simeq \frac{\beta n}{g}$$

This is a first-order Taylor expansion of $1\text{-}exp(\text{-}\beta n/g)$, the cumulative distribution function of an exponential random variable corresponding to the interarrival time of a Poisson process with rate $\beta n/g$: It is the probability that, in a unit time interval (in the present case, one week), an 'infected' individual infects one of their ($n$) contacts. Here, $n$ is the average network degree. However, there is an additional complication due to the model being constrained to a social network; the estimated rates are specific to state transitions, and thus two $R_0$ values are computed, one each for 'loss' and 'gain': For each $i \in \{l, g\}$,

$$\text{Social } R_{0,i} \simeq \frac{\beta_i n_i}{g_{T,i}}$$

Here, $\beta_i$ is the slope estimated ($a_i$ being the corresponding intercept) via an ordinary least squares (OLS) regression of neutral-to-state $i$ transitions at time $t$ on the number of contacts in state $i$ at time $t$-$1$ (of which $n_i$ is the average); and the 'total' rate,

$$g_{T,i} = g_i + s_i$$

Here, $g_i$ is the intercept estimated via an OLS regression of state $i$-to-neutral transitions at time $t$ on the number of neutral contacts at time $t$-$1$. Denoting by $j$ the other infected state, and $\sigma_{ij}$ the intercept estimated via an OLS regression of state $i$-to-state $j$ transitions at time $t$ on the number of contacts in state $j$ at time $t$-$1$, the following are computed:

$$\alpha_j = a_j + \beta_j n_j$$

$$s_i = \left[\sigma_{ij} - g_i\left(1 - \frac{1 - e^{-\alpha_j}}{\alpha_j}\right)\right]_+$$

Here, $[.]_+$ denotes the positive part, max{0,.}.

Our novel procedure involves three key $R_0$ causal intervention effect measures. First, having so computed $R_{0,l}$ and $R_{0,g}$, our 'integrated' *net loss* $R_0$ value is computed. Furthermore, in contrast to an observational study which was only able to establish the existence of dis/content $R_0$ values incidentally (Hill et al. 2010 [27]), the present RCT data was partitioned into three arms (treatment groups), full ($A$) and partial ($B$) treatment and control ($C$), yielding values for each $x \in \{A, B, C\}$ and quantiles $q \in [0,0.9]$:

$$R_{0,x;q} \equiv R_{0,x;l,q} - R_{0,x;g,q}$$

Here, for each $i \in \{l, g\}$, $R_{0,x;i,q}$ is as above, with contacts restricted to lie within common arms ($x$). This measures a sort of 'profit' realized within the corresponding arm; the probability of loss (improvement) less that of gain (disimprovement).

More precisely, this weight loss net $R_{0,x;q}$ is interpreted as follows: Within arm $x \in \{A, B, C\}$, define the loss (gain) state via a previous-period weight decrease (increase) with magnitude exceeding quantile $q \in [0,0.9]$ of the collection of all such one-period weight change magnitudes, and neutral otherwise; then $R_{0,x;q}$ is the *expected number of subsequent neutral-to-loss transitions induced by contacts in the loss state, less that of neutral-to-gain transitions induced by contacts in the gain state, both taken relative to a 'contactless' case where state transitions are not influenced by such contacts*. As such, either term (unlike a classical, epidemiological $R_0$) may be *negative*, in case of negative rather than positive influence, where loss (gain) contacts may be expected to in fact *decrease* the incidence of neutral-to-loss(-gain) transitions. E.g., heavy drug (say, heroin) use might be an intuitive example, where (hypothetically) having familial drug users may actually reduce one's likelihood of becoming a user. As a concrete example, in case $R_{0,x;l,q} = 2$ and $R_{0,x;g,q} = 0.5$, $R_{0,x;q} = 1.5$, say at a threshold quantile corresponding do 5 lb weight gain/loss, 1.5 more neutral-to-loss than -gain transitions are expected to occur, respectively due to loss-(gain-)state contacts. Also, since $1.5>1$, over iterated time periods, such weight loss is expected to propagate as a contagion at an epidemic level.

Smoothed via moving average (with factor 0.75) and estimated [at the 95% level, propagating OLS coefficient estimates' standard errors via Monte Carlo simulations (Kroese, Taimre, and Botev 2011 [31]) and robustly estimating covariance via the Olive-Hawkins method (Olive 2004 [32])], these values are obtained and plotted for thresholds (demarcating the neutral/infected state transitions) between zero and the 90$^{th}$ percentile of the histogram of absolute weight changes (for the entire dataset).

Second, to compare the three arms [which again is beyond an observational scope (Hill et al. 2010 [27])], the following (intent-to-treat causal $R_0$, relative efficacy) ratios are proposed:

$$R_{c,AC;q} \equiv \frac{R_{0,A;q} - R_{0,C;q}}{|R_{0,C;q}|}$$

$$R_{c,AB;q} \equiv \frac{R_{0,A;q} - R_{0,B;q}}{|R_{0,B;q}|}$$

$$R_{c,BC;q} \equiv \frac{R_{0,B;q} - R_{0,C;q}}{|R_{0,C;q}|}$$

The numerator differences ensure that these ratios' signs correspond to treatment efficacy; positive for treatment-induced weight loss. And the denominator absolute value scales this as a multiple of the control without regard to its sign. More precisely, the reason for using the absolute value of the denominator, is that the sign of the numerator then indicates whether the intervention improved upon the control (positive) or not (negative). These values are similarly smoothed and estimated for thresholds up to the 90$^{th}$ percentile of the histogram of absolute weight changes.

Third and finally, a summary measure of the efficacies $R_{c,i;q}$ for each $i \in \{AC, AB, BC\}$, up to the $q^{th}$ percentile of the weight change histogram, is computed as a histogram [say, $p(\kappa)$]-weighted average of the preceding values:

$$\bar{R}_{c,i;q} = \int_0^q R_{c,i;\kappa} dp(\kappa)$$

Similarly, taking instead the arm-wise $R_{0,x;q}$ values as integrands, analogous summary measures are obtained:

$$\bar{R}_{0,x;q} = \int_0^q R_{0,x;q} dp(\kappa)$$

Finally, note that the longitudinal, repeated-measures RCT data are spread across 14 one-week intervals and follow-ups at 21.75, 24 and 28.25 months; but given that there are too little data within any single of these, the state transitions and contact counts are pooled across all to yield significant results. Also, the OLS regressions are controlled for over a hundred covariates, including age, gender, political unrest, Ramadan, preexisting health conditions such as pre/diabetes, hypertension and being overweight or obese, and several others obtained from survey data related to lifestyle habits including healthy eating, smoking and exercise.

The steps previously described are shown in Fig 1.

The computations of Fig 1A and 1B are combined diagrammatically in Fig 2A; likewise for all of Fig 1 in Fig 2B.

Ethics: This study was approved by the WIRB (USA, 20121224) and by the National Center for Diabetes, Endocrinology and Genetics (Jordan, 3/2011).

## Results

The study included 910 participants with randomization allocated with the ratio of 3:1:1, with resulting group sizes of n = 537, 186, and 187 in arms A, B, and C, respectively. Recall the

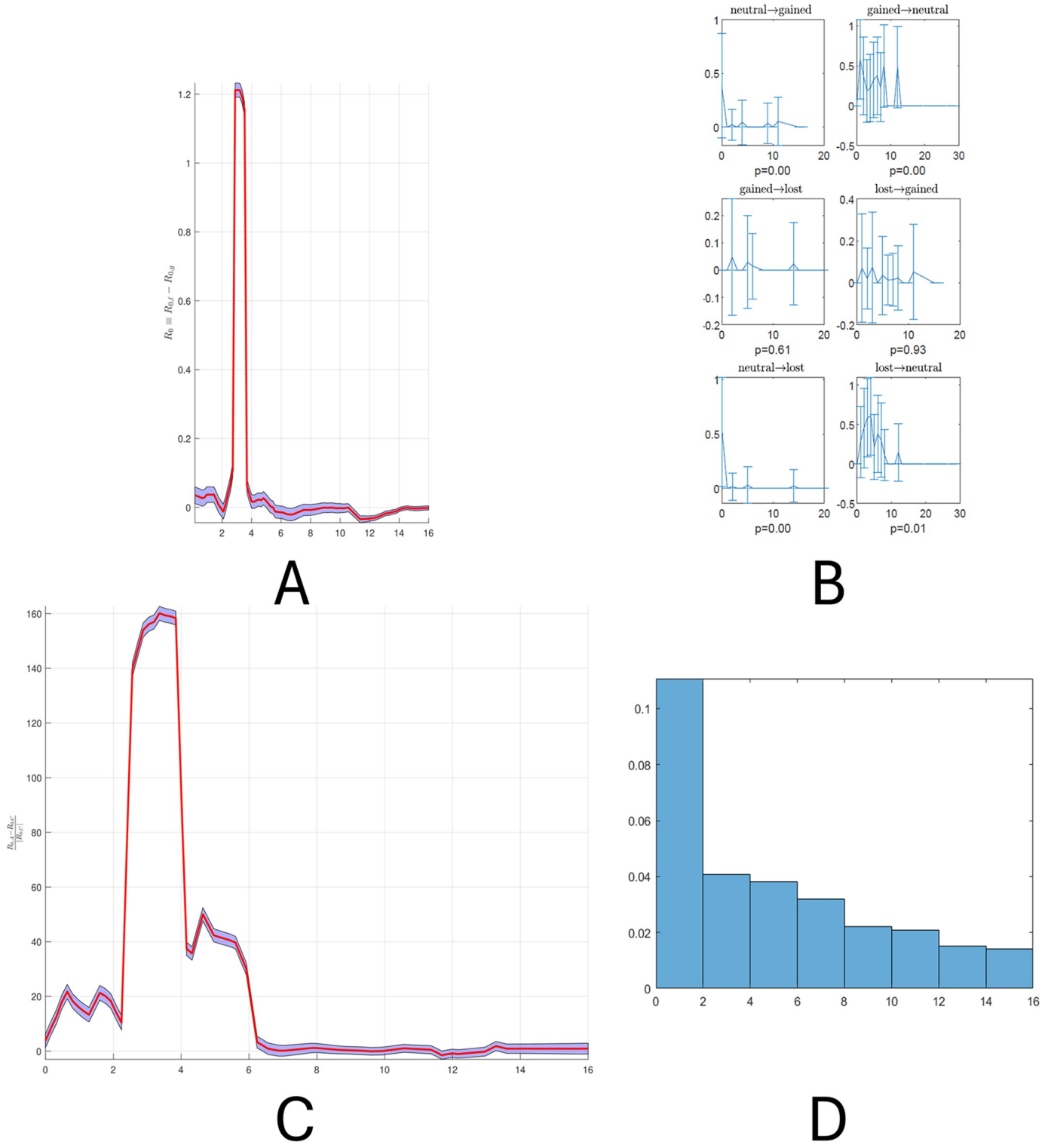


**Fig 1.** A. Specify a threshold ($x$-axis) for weight change above (below) which absolute value positive/negative changes are classified as gained/lost (neutral). B. Perform OLS on each of six non-self transitions between these three classes, for each subject eligible to so transition against their number of contacts likewise transitioning in the previous period. Coefficients from the four transitions involving loss (gain) are used to compute $R_{0,l}$ ($R_{0,g}$), which difference yields $R_0$ (previous panel $y$-axis). C. Performing the preceding steps for the treatment (control) group yields $R_{0,A}$ ($R_{0,C}$), which difference divided by the absolute value of the latter yields $R_c$. D. Numerically integrating $R_c$ up to the $q^{\text{th}}$ percentile of the histogram of all subjects' weight changes (shown), weighted by the latter, yields the so-weighted average, $\bar{R}_{c,q}$

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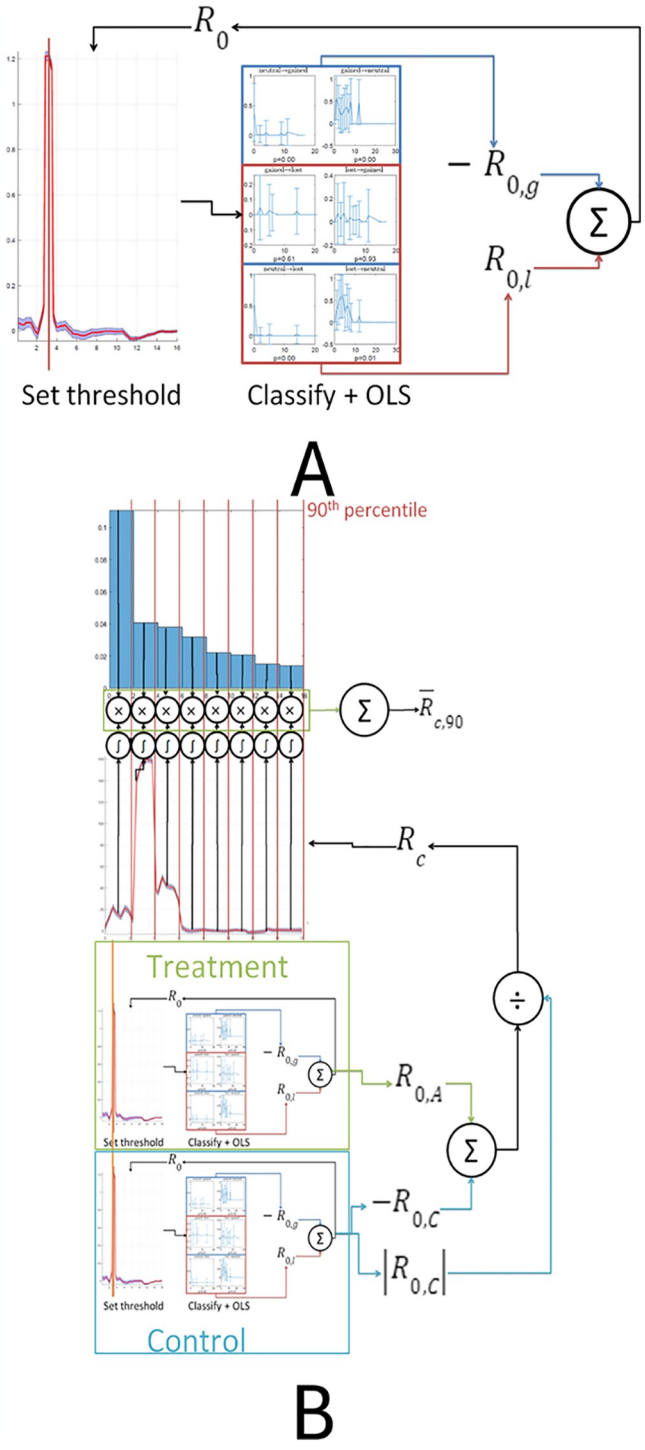


**Fig 2.** A. $R_0$ calculation: For a given threshold, subjects are classified as gained, lost or neutral; OLS regression is done for each of the corresponding pairwise transitions; and those not involving gained (lost) are combined to yield $R_{0,l}$ ($R_{0,g}$); the difference of which yields $R_0$. B. This procedure is separately applied to treatment and control subjects, respectively yielding $R_{0,A}$ and $R_{0,C}$; the difference of which, divided by the absolute value of the latter, yields $R_c$; which is then numerically integrated with respect to the histogram of (all subjects') weight changes to yield $\bar{R}_{c,q}$, e.g., in case $q = 90$, up to the 90th percentile.

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**Table 1. Baseline Participant Characteristics.**

| Characteristic, mean (SD) or % | Full MCP (n = 537) | Basic MCP (n = 186) | Control Group (n = 187) |
|---|---|---|---|
| Age, years | 54.2 | 56.6 | 56.2 |
| Women, (%) | 66.5 | 67.0 | 65.0 |
| Weight, kg | 85.9 | 85.0 | 86.0 |
| Height, m | 160.3 | 159.6 | 160.3 |
| BMI, kg/m$^2$ | 33.6 | 33.5 | 33.4 |
| Systolic blood pressure, mm Hg | 129.1 | 131.7 | 132.2 |
| Diastolic blood pressure, mm Hg | 81.3 | 81.0 | 81.3 |
| Mean arterial pressure, mm Hg | 97.2 | 97.9 | 98.3 |
| HbA1c (%) (SD) | 6.91 | 6.90 | 6.91 |
| Fasting plasma glucose*, mg/dL | 147.8 | 142.8 | 145.7 |

*baseline fasting glucose average of first and second weeks.

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defined weight loss net $R_{0,x;q}$: Within arm $x \in \{A, B, C\}$, define the loss (gain) state via a previous-period weight decrease (increase) with magnitude exceeding quantile $q \in [0,0.9]$ of the collection of all such one-period weight change magnitudes, and neutral otherwise; then $R_{0,x;q}$ is the *expected number of subsequent neutral-to-loss transitions induced by contacts in the loss state, less that of neutral-to-gain transitions induced by contacts in the gain state, both taken relative to a 'contactless' case where state transitions are not influenced by such contacts*. As a concrete example, in case $R_{0,x;l,q} = 2$ and $R_{0,x;g,q} = 0.5$, $R_{0,x;q} = 1.5$, say at a threshold quantile corresponding to 5 lb weight gain/loss, 1.5 more neutral-to-loss than -gain transitions are expected to occur, respectively due to loss-(gain-)state contacts. Also, since $1.5>1$, over iterated time periods, such weight loss is expected to propagate as a contagion at an epidemic level.

Table 1 collects trial participants' baseline characteristics.

As follows are tabulated the histogram-weighted averages of the proposed (*comparative*) ratios ($R_c$) for weight change, with entries italicized to indicate nonsignificance at the 95% level; the others are all significant:

Table 2 reports point and interval estimates for these weight loss net $R_{0;q}$ (full and partial) treatment effects (divided by the corresponding absolute control loss net values), averaged over quantiles up to $\bar{q} \in \{0.75, 0.8, 0.9\}$ and weighted by the histogram of absolute weight changes; with full treatment results seen to be consistent, and in particular for $\bar{q} = 0.9$ (replicated as the third column of Table 3): $\bar{R}_{c,90}$ weight loss is estimated to be 244 (95% CI [238,250]; $p<0.001$). Conversely, the effect of partial treatment is *not* consistent, with outliers

**Table 2. Weight loss histogram threshold-weighted average $R_c$ up to 75$^{th}$, 80$^{th}$, and 90$^{th}$ percentiles.**

| (Net of R(loss)–R(gain)); arms *i-C* | Histogram-weighted average $R_{c,i}$ (up to the listed percentile), $\bar{R}_{c,i,q}$ | | |
|---|---|---|---|
| *q* | 0 to 75$^{th}$ percentile | 0 to 80$^{th}$ percentile | 0 to 90$^{th}$ percentile |
| Body Weight; *A-C* | 368 [361,374] | 368 [361,374] | 244 [238,250] |
| Body Weight; *B-C* | 3.97 [2.31,5.64] | 3.97 [2.31,5.64] | -10.3 [-11.5,-9.11] |

Italicization denotes non-significance at the 95% level.

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**Table 3. Ratio of Social $R_0$ Reproductive Number (0-90$^{th}$ percentile) in Classrooms over all Intervention and Control Sessions, for Weight Loss.**

| (Net of R(loss)–R(gain)) | | | |
|---|---|---|---|
| | **Intervention Arm Social $\bar{R}_{0,A,90}$** | **Control Arm Social $\bar{R}_{0,C,90}$** | **Causal effect: Ratio of Social $\bar{R}_{c,90}$** |
| Body Weight | 0.311 [0.247,0.376] | *0.00344 [-0.0167,0.0236]* | 244 [238,250] |

Italicization denotes non-significance at the 95% level.

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between the 80$^{th}$ and 90$^{th}$ percentiles causing reversals for weight loss; and as such it is neglected in what follows.

As follows are tabulated the histogram-weighted averages of the proposed social ratios ($R_0$) for weight loss, with entries italicized to indicate nonsignificance at the 95% level:

Similar to taking net loss less gain weight changes, net full intervention against control differencing establishes treatment effect. Table 3 reports point and interval estimates for these weight loss net $R_{0,x;q}$ values, averaged over quantiles up to $\bar{q} = 0.9$ and weighted by the (within-arm) histogram of absolute weight changes: Full intervention [control] $\bar{R}_{0,90}$ weight loss is estimated to be 0.311 (95% CI [0.247,0.376]; $p<0.001$) [0.00344 (95% CI [-0.0167,0.0236]; $p = 0.738$)].

As to why the causal effect ratio reported in the third column of Table 3 doesn't equal or even approximate the naïve crude ratio of the values in the intervention versus control arms:

$$\bar{R}_{c;q} = \int_0^q R_{c;\kappa} dp(\kappa) = \int_0^q \frac{R_{0,A;\kappa} - R_{0,C;\kappa}}{|R_{0,C;\kappa}|} dp(\kappa) \neq \frac{\int_0^q R_{0,A;\kappa} dp(\kappa)}{\int_0^q R_{0,C;\kappa} dp(\kappa)} = \frac{\bar{R}_{0,A;q}}{\bar{R}_{0,C;q}}$$

The formula is more than just a crude ratio—as mathematical rules dictate that "the integral of a ratio is not the ratio of the integrals:" Both because $R_{c;\kappa}$ divides by the *absolute* value $|R_{0,C;\kappa}|$, and the numerator difference without that being the case would result in subtracting $q$ from the left-hand equalities. We believe our way is superior than a straightforward crude ratio as the right-hand pair of equalities (included as the fourth column of Table 3, with no significant results): The latter compares averages over the appropriate thresholds which 'blurs' their effect, whereas the former directly compares arms for each threshold, before averaging which avoids 'apples-to-oranges' comparisons. Intuitively, this avoids missing important facts, e.g., that peaks in Fig 3 occur for different thresholds; for which the intervention-vs.-control comparison is strong, and remains so even when averaged over all thresholds; whereas averaging before taking ratios smoothes the peaks and ends up artificially comparing distributional averages as though they correspond to equal thresholds, hence the comparatively unimpressive ratios that result from dividing the first two columns of Table 3.

For weight, a few notable trends are that, compared to arm *C*, the impact of arm *A* is consistent and one to two orders of magnitude beyond that of *B*, which also switches been positive and negative effect with $q$, possibly as arms *B* and *C* each had roughly half the number of subjects as *A*. Also, it is apparent that subjects between the 75$^{th}$ and 80$^{th}$ percentiles simply reinforced the histogram-weighted averages computed up to the former.

Clearly, this constitutes ample evidence of improved $R_0$ values (i.e., the average number of secondary 'infections' which experience weight changes above a given threshold, corresponding to each previously so-changed subject; loss net of gain), from control arm *C* to full

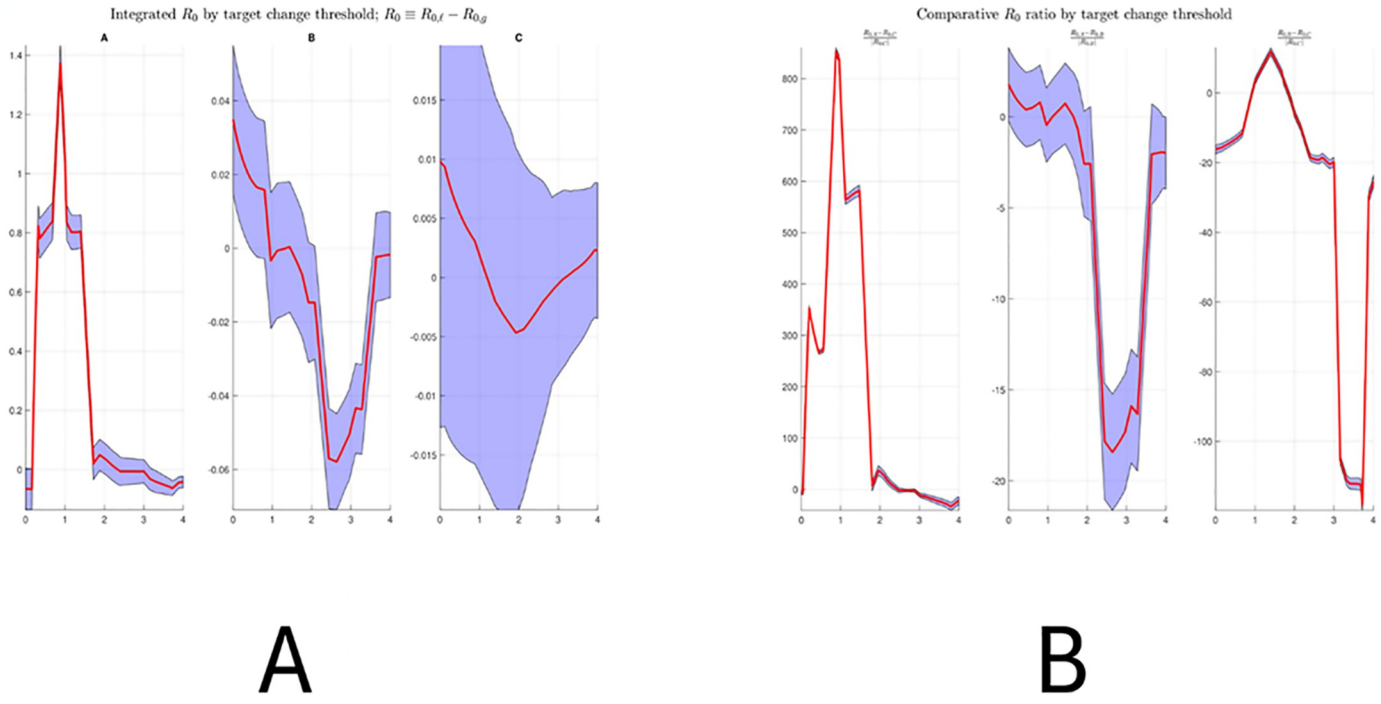


**Fig 3.** A. Weight change; $R_0$ values by arm: Most notable here is an *epidemic* value of $R_0$, loss net gain, in arm *A*: Separately from the 'automatic' infection (term *a*) which distinguishes the SISa model from its classical compartmental counterpart, SIS, it is significantly (at the 95% level, and likewise for all following results) observed in the trial (full treatment) arm *A* that for every subject having lost between 0.7–1 kg or more, on average in the following week, more than one (specifically, about 1.3) of their previously neutral social contacts also lose between 0.7–1 kg or more, *relative to the corresponding number of previously neutral social contacts that gain between 0.7–1 kg or more, for every subject having gained between 0.7–1 kg or more in the preceding week*. E.g., this situation could involve each 'loser' inducing 1.3 loser contacts and each 'winner' none, each loser and winner inducing 0.6 loser and winner contacts, or each winner inducing 1.3 winner contacts and each loser none: However, *net of gain*, each loser induces on average 1.3 loser contacts. 'Flanking' this threshold range by about 0.4 kg above and below, significant (nonepidemic) $R_0$ values of about 0.8 are observed in arm *A*, whereas elsewhere and in (control) arm *C*, no other comparable $R_0$ values are observed. For context, the (observational, and so not distinguished between treatment and control RCT arms) content net discontent R0 of another study (Hill et al. 2010 [27]) is 0.28+0.39 = 0.67; less but on the same order of magnitude observed. For (partial treatment) arm *B*, significant but smaller (by an order of magnitude) net values are observed, positively for thresholds up to about 0.4 kg, and negatively for those between 2–3.5; which help to establish the more 'eccentric' results tabulated above. To emphasize, relative to control (arm *C*), *full* treatment (arm *A*) has significantly and for a range of change thresholds, induced positive, *epidemic* weight loss net of gain!. B. $R_c$ values: Notable here is an anticipated jump for the same thresholds which yield epidemic social-network-induced weight loss net gain; namely, 0.7–1 kg. Though even for the broader range from about 0.3–1.4 kg in which $R_0$ values exceeding 0.8 are observed, and in fact, yet further between 0.1–1.7 kg, comparative $R_{c,AC}$ values of two orders of magnitude are computed: The conclusion is that relative to control arm *C*, the differential improvement in (full) treatment arm *A* is (significantly) two orders of magnitude greater than the absolute $R_0$ of weight loss net gain. In other words, not only does treatment induce epidemic-level improvements in weight loss net gain from the control, but as a multiple of the latter's magnitude, the degree of improvement is up to and above 800 times. Relative to *partial* treatment arm *B*, there is a *negative* deviation (an order of magnitude lower) for much higher thresholds (as seen in the corresponding $R_0$ plots, between 2–3.5 kg); but this is of little importance: Namely, because for such thresholds which exceed even the mean weight change in the entire dataset, the analysis becomes driven by outliers exhibiting extreme weekly weight loss or gain (all others being classified as neutral); and as such the histogram-weighted average $R_{c,AB}$ value, -1.28, is insignificant as it heavily discounts these outlying aberrations. The *B*-*C* comparison is seen to generally yield significantly negative $R_{c,BC}$ values where the $R_0$ values are significant in partial treatment arm *B*, and to be mixed elsewhere; which combined with the heavily right-skewed weight change histogram, results in the significantly negative $R_{c,BC}$ value, -10.28. But again, these two preceding results are secondary to the RCT design (emphasizing full rather than partial treatment or control); but in the latter case, may support a notion that when providing detailed health advice, it may better be only in the case of accompanying (group) activity to contextualize and pace the information acquisition in an effective manner.

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treatment arm *A*, as a multiple of the (absolute) control value, histogram-weighted averaged up to the 90$^{th}$ percentile of changes observed across all data. Furthermore, this effect is two orders of magnitude, indicating an enormous, (full-)treatment-induced improvement in social-network-propagated health behaviour.

Interestingly, there is significant *worsening* (of an order of magnitude) from control arm *C* to *partial* treatment arm *B*, suggesting that 'knowledge is insufficient:' Simply providing subjects with information on how to improve their health was less effective than doing nothing,

without the additional, social activities enriching this knowledge with action/instruction in a group setting. Information provided without that context could also be overwhelming or intimidating, resulting in this counterproductive effect; as opposed to subjects discarding the information without the 'enforced' group learning, in which case no difference from control should be expected. Though it should be emphasized that arms *B* and *C* were one-half the size of *A*, meaning this significantly negative *B*-*C* result should not be held in the same regard as the significantly positive *A*-*C* one (which is also an order of magnitude larger). Similarly, the insignificant *A*-*B* result is unremarkable, in particular, as the RCT was not designed to study this comparative effect of full vs. partial treatment; other than the intransitivity that, significantly, '$A>C$' and '$B<C$,' yet (insignificantly) '$A<B$!'

Fig 3 presents for weight change, plots of $R_0$ by arm and $R_c$ up to the 90$^{th}$ percentile of changes across the dataset; the histogram-weighted averages of the latter being tabulated previously.

## Conclusion

These results demonstrate the positive propensity for the propagation of weight loss in classrooms over time; cascading behavior that public health authorities can leverage.

In this paper we provide the first evidence of causal social-to-biological contagion from a randomized controlled trial based in low-income Jordanian communities in Amman, Jordan. Importantly, because the study was a 3-arm trial, we were able, for the first time, to differentiate between basic and full "dosing" effects of the social network intervention. The results suggest that social networks in and of themselves will not always yield propagative effects; they have to be engineered and managed to do so.

In addition to this dosing phenomenon, we made a second discovery. After performing an effect modification for weight loss based on gender, we found that males overwhelmingly drove weight "good" loss results, relative to controls, by a factor of 473. We hypothesize that this effect is due to modesty norms in Jordanian communities where unrelated males and females are mixed. Women typically wear long, black flowing *abayas* (robes), *hijabs* (head coverings), and sometimes even *niqabs* (full face coverings). This makes it more difficult to observe physical weight loss among females, possibly attenuating social contagion effects. Clearly, issues of gender equity need to be further studied and considered in future interventions in the Middle East.

The causal social network results presented here are also consistent with evidence presented elsewhere. In a Kentucky randomized trial, a vastly different rural setting, we used a novel ITT Social Induction Ratio to identify different correlations of weight loss trajectories in the intervention versus controls (Ding et al. 2024a [17]). In Jordan, we also identified a specific social induction mechanism as a "Follow-the-Leader Domino" effect, meaning that program participants gravitated towards the weight change trajectory of leaders, rather than former leaders or the average peer effect—and that this effect cascaded. Additionally, we were able to determine "good" versus "bad" contagion effects, i.e. weight loss versus weight gain.

But there is something more. As with Newton's proverbial falling apple, distinct social network forces upon human biology have been self-evident for millennia. Yet until now, it has not been determined if social networks are themselves wholly reducible to mere biological processes; or if these are independent forces that shape human biology.

Since participants were not only randomized but also controlled for genetic confounding using a novel social network matrix that measured social proximity and biological relationships, we can rule out reverse causation (homophily) or confounding. The evidence presented

here, together with our prior studies, may therefore constitute the first proof of a singular social to biological contagion effect.

Denis Noble famously averred, "Now they [genes] are trapped in huge colonies, locked inside highly intelligent beings, moulded by the outside world, communicating with it by complex processes, through which, blindly, as if by magic, function emerges" (Denis 2006, page 12 [33]). This paper attempts to demonstrate, in a generalizable way, what demystification of some of this "magic" might entail. In the years to come, we believe the task of biology will be not simply reductionist, but also integrative of complex physical and sociological systems.

## Policy implications

In some ways, the behavioral contagion described in this paper is both cutting-edge epidemiology and stone-age anthropology. The proverbial wisdom that bad company corrupts good morals has been around for millennia. Plato articulated his theory of *mimesis* (imitation) in the fourth century BCE (Plato 2008 [34]); and Ibn Khaldun's concept of *asabiyya* ("group feeling") laid a foundation for the modern field of social and economic history (Khaldun 2015 [35]).

Within the public health realm, scientists and the general public regularly acknowledge social contagion dynamics, referring to the drug, obesity, or diabetes "epidemics," perhaps without fully appreciating the category's implications. Referring to the spread of so-called "non communicable" diseases in "epidemic"–or even "pandemic" terms (Allen 2017 [36]; NCD Alliance 2016 [37]; Sheldon & Wright 2020 [38])–is a mathematical statement, implying an $R_0 > 1$.

As we have shown that a Social $R_0$ value could be calculated and proven to exist for weight, it follows that the category Non-Communicable Disease is a misnomer; and adding epidemic is a contradiction in terms. When combined with the forecited biological evidence of numerous NCDs caused by transmissible pathogens, we find that fundamental analytical, economic, and policy categories need to be radically reconsidered (World Health Organization 2023b [39]). This reckoning with basic analytical categories is especially salient in light of emerging evidence suggesting that our understanding of biological contagion itself—much less social contagion—is inadequate. A credible study showed that Alzheimer—a textbook "non-communicable" disease—is likely biologically infectious under certain conditions (Jucker et al, 2024 [40]).

The results presented in this paper, together with prior studies, clearly demonstrate the social transmissibility of so-called "non-infectious" diseases on clinical weight loss. This is not to say that conventional categories—biologically infectious versus biologically noninfectious—are totally misconceived, but that they are analytically blunt instruments, often obscuring powerful social network and/or biological mechanisms. Microbial infections are indeed spread via physical media, and need to be studied safely under a microscope. But they also can spread via social media, as COVID-19 disinformation wars demonstrate. Therefore, in a very real way, the digital and the biological world are entangled together in $R_0$ calculations (Delamater et al. 2019 [41]; Fu, Christakis & Fowler 2017 [42]) and should be studied using multidisciplinary tools.

### Limitations

There are several limitations to this study. Retention was similar to other peer studies (Diabetes Prevention Program Research Group et al. 2009 [43]), but not significantly better. Furthermore, while the present study is limited to its location in a low-income Middle Eastern community, we have found that the Microclinic Social Network Model, more broadly, induces

sociological and clinical changes in different geographical settings: from Lebanon to Gaza, from the West Bank to Jordan, and even from Kentucky to Kenya (Zoughbie et al. 2014 [44]; Zoughbie et al. 2009 [45]; Shahin et al. 2018 [46]; Ding et al. 2013a [47]; Prescott et al. 2013 [48]; Ding et al. 2013b [49]; Zoughbie et al. 2024 [50]; Zoughbie, Huddleston & Ding 2024 [51]).

Therefore, we believe the $R_0$ and causal social $R_0$ ratio we present in this paper are generalizable phenomenon which should be further studied and replicated.

## Supporting information

**S1 File.** Macro codes.docx legend, pages 1–2: "Macro code for computing the Social $R_0$ in Matlab". Macro codes.docx legend, pages 3–4: "Macro code for computing the Social $R_0$ in R". Macro codes.docx legend, pages 5–6: "Macro code for computing the Social $R_0$ in STATA" (DOCX)

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