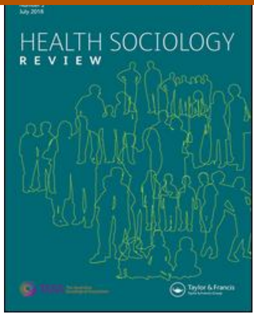


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# The (re)production of health inequalities through the process of disseminating preventive innovations: the dynamic influence of socioeconomic status

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**ABSTRACT**

Fundamental Cause Theory suggests the replacement of mechanisms that produce the persistent relationship between socioeconomic status and health over time. Understanding how this process operates is central to explaining the reproduction of health inequality. We use data from the Onco-barometer survey (2010) to test a set of hypotheses derived from FCT, Diffusion Of Innovations theory and the intersection between these theories to examine how socioeconomic inequality emerges and evolves across the cycle of diffusion of six relevant preventive practices in Spain: faecal occult blood tests, prostate-specific antigen tests, Papanicolaou tests, mammograms, cholesterol readings and blood-pressure checks. Because these preventive measures are characterised by differing rates of spontaneous knowledge and use amongst the Spanish population, we assume that they are at different stages in the diffusion cycle. Results suggest that SES has a dynamic influence according to the diffusion stage of each preventive measure. We argue that the conjunction of these theories offers a dynamic 'imagery' that can help to explain the generation and diminishment of inequalities. Moreover, this integration has the potential to bring 'social change' back into the study of health inequalities, which is essential to understanding equitable (and inequitable) returns produced by preventive innovations.

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Fundamental cause; health inequality; diffusion of innovations; preventive practices

## Introduction

Since the 1990s, sociological perspectives have been gaining ground in the study of health inequalities. In this context, Link and Phelan (1995) reformulate the 'social causation' arguments from a sociological viewpoint, thereby developing Fundamental Cause Theory (FCT). This theory revives the question of which social circumstances or factors put people 'at risk of risks' and calls for consideration to be given to the social contexts that determine specific risk factors. It also directs interest back to how the distribution

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of resources and knowledge across socioeconomic status (SES) groups can preserve health inequalities, even in the light of dramatic changes in specific proximal risk factors. Health inequalities are seen as being rooted in the pre-existing social stratification, which produces a specific distribution of resources (knowledge, money, power, prestige and beneficial social connections) amongst social groups. The main argument is that these resources shape whether individuals are aware of, have access to, can afford and are supported in their efforts to engage in health-enhancing behaviours. Consequently, when new health-enhancing capabilities emerge, those with greater access to flexible resources can benefit from them earlier and to a greater extent than can those with fewer flexible resources. For this reason, health inequalities are continually shaped (and re-shaped) as we develop more control over disease and death (Phelan & Link, 2005). FCT assumes a dynamic relationship between socioeconomic status and health-related events, given that new axes of inequality in health emerge as the old axes persist or diminish in importance.

The central role of flexible resources linked to socioeconomic status (SES) has been tested empirically by comparing situations in which they can be effectively deployed to other circumstances in which it is not possible to use them in a beneficial manner, as is the case when little or nothing is known about how to prevent disease and mortality (Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004; Rubin, Clouston, & Link, 2014). Moreover, empirical tests of FCT have also compared the evolution of health inequalities before and after the introduction of new technological innovations or new health-enhancing knowledge, to analyse how health inequality emerges, or whether the relationship between SES and specific health outcomes is shaped (or re-shaped). For example, this occurs with the emergence of life-extending treatments (e.g. HIV/AIDS medicines, statins to reduce cholesterol, the human papilloma virus vaccine, effective cancer screening or selective serotonin reuptake inhibitors in the case of suicide) (Chang & Lauderdale, 2009; Clouston, Rubin, Colen, & Link, 2014; Link, Northridge, Phelan, & Ganz, 1998; Polonijo & Carpiano, 2013; Rubin, Colen, & Link, 2010; Saldana-Ruiz, Clouston, Rubin, Colen, & Link, 2013; Tehranifar et al., 2009). In similar vein, recent research in 20 European countries has shown faster mortality declines amongst high-SES groups relative to low-SES groups for causes of death that are amenable to intervention, but not for non-amenable causes (Mackenbach et al., 2017).

Despite the efforts of these empirical studies, the specific underlying mechanisms that drive the process of reproduction of inequalities over time remain poorly understood. More specifically, the literature lacks any in-depth explanation of the time dimension of FCT – the evolution of inequalities over time. FCT would thus benefit from integration with another theoretical framework that offers a perspective that explains trends over time regarding the equity impact of health technologies. Diffusion of innovation (DOI) theory (Rogers, 1983) is well-suited to fill in the less-elaborated time dimension of FCT. Technological developments are key components of both theoretical frameworks. While FCT focusses on their unequal use and impact on health inequality, DOI theory highlights their adoption and diffusion over time. In particular, DOI theory predicts that technological innovations spread through the population in a predictable pattern (Rogers, 1983). Although this theory has been widely used in public health research (Greenhalgh et al., 2005; Haider & Kreps, 2004), only recently have scholars begun to use DOI theory models to explore the possible influence of changing patterns of health-related habits and the diffusion of preventive innovations on the magnitude of social inequalities (Centola, 2011; Elstad, 2013; Glied & Lleras-Muney, 2008; Wang, Clouston, Rubin, Colen, & Link, 2012).

The main objective of the current study is to elaborate on FCT by applying the theoretical principles of DOI theory to preventive healthcare use. We argue that FCT should assign in-depth consideration to the relevance of the diffusion contexts of preventive practices, in accordance with its proposition concerning the dynamic association between SES and health over time. More specifically, we test a set of hypotheses derived from FCT, DOI theory and the intersections between these theories to examine how socioeconomic inequality emerges and evolves across the cycle of diffusion for six relevant preventive practices in Spain: (1) faecal occult blood (FOB) tests; (2) prostate-specific antigen (PSA) tests, only for men; (3) Papanicolaou tests (Pap smears) and (4) mammograms, only for women; (5) cholesterol readings and (6) blood-pressure checks. Because these preventive practices are characterised by differing rates of spontaneous knowledge and use amongst the Spanish population, we assume that they are at different stages of the diffusion cycle.

## Theoretical framework and hypotheses

### *Fundamental causality propositions*

The main contribution of FCT has been the development of a theoretical framework that reveals paths that link socioeconomic status (SES) to health and that can account for the persistence of inequality in health despite temporal changes in the risk factors involved. Link and Phelan (1995) propose four conditions for determining whether SES is a basic cause of health inequality. The first two conditions are: (1) SES influences multiple disease outcomes, (2) through multiple risk factors. These propositions suggest that the persistence of the association between SES and health cannot be explained only by analysing contingent associations produced by particular risks and specific outcomes. Instead, these two propositions point to the relevant role played by changes in risks and health conditions and to the preservation of the association between SES and health beyond a particular risk profile at a given time. The third condition is as follows: (3) SES involves access to 'flexible resources' that can be used to avoid or minimise the consequences of a disease when it occurs. The concept of flexible resources thus plays a central role in the theory, as it highlights the differential access to money, knowledge, power, prestige and beneficial social relationships that influence the ability of individuals to avoid risk and adopt protective strategies in relation to diseases and treatments, as well as to possess knowledge concerning the risks associated with and changes in these diseases and treatments. The final condition holds that (4) the association between SES and health is reproduced over time through the replacement of intervening mechanisms. Changes in the incidence/prevalence of diseases, the risk of these diseases, knowledge about these risks and the effectiveness of treatments do indeed make it possible to apply the concept of the fundamental social causes of disease. This is because such changes give rise to new mechanisms, which replace or overlap the previous ones (Link & Phelan, 1995). Based on the four conditions posed by Link and Phelan (1995), our first assumption (H1) is that SES inequalities in preventive health practices manifest themselves in the uptake of multiple preventive measures – FOB tests, PSA tests, Pap smears, mammograms, cholesterol readings and blood-pressure checks – as societies gain control over diseases via new preventive knowledge.

Clouston and colleagues (2016) add a temporal context to FCT with their idea of the 'social history of disease'. Proceeding from the development of preventive knowledge and innovations, they consider health inequality in cause-specific mortality at a given time and place as a four-stage process. The first stage – natural mortality – is characterised by a lack of knowledge about prevention or effective treatment, and thus few, if any differences in mortality between people of different SES. During this stage, any SES differences that might exist do not always disadvantage those at the lower end. The second stage, which is characterised by the production of health inequalities, starts as a society develops new capacities for reducing the risk of mortality from a particular disease. In this stage, the benefits of new preventive capacities are unequally distributed across SES levels, such that health inequalities in mortality emerge for the specific cause in question. The third stage, in which health inequalities are reduced, begins as health-beneficial preventive innovations are extended. In this stage, the authors predict stabilisation in health inequalities, which can also begin to decrease in response to the more even distribution of the new life-saving knowledge throughout the population. The final phase is characterised by reduced mortality and disease elimination, with inequalities in mortality for the specific cause being greatly reduced and possibly even disappearing. According to the principles of FCT, however, the idea of 'social history of disease' suggests the reproduction of health inequalities over time. This is because new knowledge and possibilities for preventing cause-specific mortality will continue to emerge, such that new health inequalities also continue to arise and proceed through the four stages.

The observations presented above draw a substantial theoretical link to the DOI model proposed by Rogers (1983). One relevant empirical matter in this regard concerns how propositions of FCT operate across the diffusion of preventive practices. Such an exploration could elaborate the process described by Clouston and colleagues (2016) in greater depth.

### *Intersections with the diffusion of innovations theory*

DOI theory proposes a model in which new preventive technologies or health interventions follow an S-shaped adoption curve, which summarises the process by which these innovations spread over time within a given social system through personal contacts and social networks (Rogers, 2003). In the beginning, only a few people will adopt the innovation, followed by increased and accelerated adoption amongst the majority of the population as more and more people become acquainted with it. According to Rogers (2002), this rate of adoption suggests five distinct 'adopter categories': innovators, early adopters, early majority, late majority and laggards.

Given that people adopt innovations when they come in contact with other individuals like themselves who have already adopted them (Rogers, 1983), the spread of innovations through a population can be perceived in terms of social contagion (Centola, 2011; Centola & Macy, 2007). For the diffusion of 'complex' social contagions, which are costly, difficult or unfamiliar and typically include preventive practices, people require social reinforcement from multiple adopters in order to be convinced to adopt the innovations themselves (Centola, 2015; Centola & Macy, 2007; Guilbeault, Becker, & Centola, 2018). Social networks and their social composition (gender, education, occupation, ethnicity, neighbourhood and geographical region) thus play an important role in determining the way in which preventive practices and other complex contagions are likely to diffuse within a heterogeneous population (Guilbeault et al., 2018; Rogers, 2003). Empirical evidence has demonstrated that preventive measures diffuse more easily and rapidly within social systems characterised by a high degree of homophily (Centola, 2011) and interconnectedness (Rogers, 1983). Accordingly, earlier adopters share similar traits that differentiate them from later adopters, and they are more highly interconnected in the social system (Rogers, 1983).

Innovators and early adopters generally differ from later adopters in terms of their personal characteristics, and they tend to have had more years of education and higher social status (Rogers, 1983). As a consequence of these disparities amongst adopters, systematic social inequalities can emerge if different social strata vary in the pace at which they adopt a given innovation (Elstad, 2013). This situation entails several intersections with FCT, particularly regarding the basic assumption that access to and use of flexible resources can generate systematic differences in the early adoption of preventive practices and behaviours. Accordingly, people with higher SES are likely to take advantage of new health-enhancing opportunities as they emerge, or when specific healthy practices begin to be socially perceived as innovative. This highlights that innovations spread from high-status to low-status positions in a social-hierarchical process (Lindbladh, Lyttkens, Hanson, & Östergren, 1997). As a consequence, SES inequalities may emerge at the beginning of the diffusion process, reach a peak in the middle stages and gradually decline during the final stage as shown in Figure 1 (Elstad, 2013).

In this paper, we assume that DOI theory complements the propositions of FCT and that, together, they provide a theoretical framework for understanding the dynamic interaction process between the characteristics of innovations, potential adopters and the influence of contexts in which multiple sources of preventive knowledge, technologies and behaviours are diffused simultaneously. Following the fourth proposition of FCT,

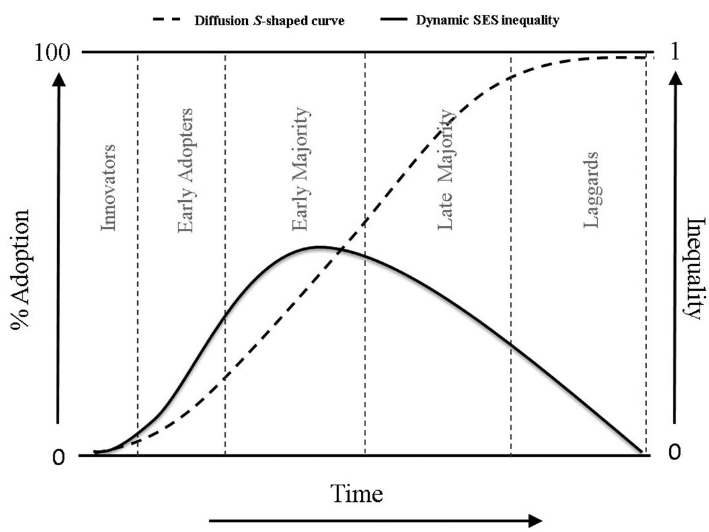


**Figure 1.** Proposed pattern of health inequality along the diffusion of innovation (DOI) model. Adapted from: Rogers (2003).

and in line with the arguments suggested by Elstad (2013) and by Clouston and colleagues (2016), our second assumption (H2a) is that the extent of SES inequalities in preventive health practices will vary according to the diffusion stage of each specific preventive measure. More specifically, we expect (H2b) that an early diffusion stage will be accompanied by the absence or emergence of SES inequalities, while intermediate stages will be accompanied by high inequalities and advanced stages by the reduction or absence of inequalities.

## Material and methods

### *Sample data*

We use data from the Onco-barometer survey, which was designed and implemented by the Centro de Investigaciones Sociológicas (CIS) and the Spanish Association Against Cancer (AECC) between November and December 2010. The Onco-barometer consisted of 7938 face-to-face interviews with members of the non-institutionalised Spanish adult population (aged 18 years or older) – excluding the autonomous cities of Ceuta and Melilla – concerning perceptions, general attitudes and knowledge about cancer, risk factors, treatments and healthcare assessment. Our analyses are restricted to adults between 50 and 69 years age, thus ensuring that we address only an age group for which all of the screenings and readings assessed are recommended. In addition, some health conditions increase the possibility of being involved in preventive care, sometimes even as part of the treatment. For this reason, people who had been diagnosed with cancer at some point in their lives ($N = 206$; 6.1%) and people with cardiovascular disease ($N = 429$; 12.8%) were omitted in analyses concerning cancer screening; blood-pressure checks and cholesterol readings, respectively. This resulted in the following weighed samples, following the exclusion of accumulated missing cases for each preventive practice: 1904 respondents for FOB tests, 889 men for PSA tests, 943 women for Pap smears, 965 women for mammograms and 1642 persons for both cholesterol readings and blood-pressure checks.

### *Variables*

The dependent variables are six preventive healthcare measures at different stages of the diffusion cycle: (1) FOB tests, used for colorectal cancer screening; (2) PSA tests for the detection of prostate cancer in men; (3) Pap smears and (4) mammograms for cervical and breast cancer screening (respectively) in women; and (5) cholesterol readings and (6) blood-pressure checks, which constitute two key practices for the prevention of cardiovascular morbidity and mortality. Respondents were asked the following question: 'Can you tell me whether you have participated in any of the following tests and checks in the past two years on your own initiative, on the explicit orders of a doctor or within an annual periodic review?' To simplify the analysis, we consider only two categories (1 = yes; 0 = no) for each dependent variable.

In Spain, FOB tests are performed at two-year intervals within the population between the ages of 50 and 69 years (Basu et al., 2018). Except for some regions that were in the pilot phase when the data from this research were compiled, most regions have provided FOB tests through an organised population-based colorectal cancer screening program during the 2000s. In contrast, PSA tests are not organised within any population-based preventive screening programme. Most major urology societies do not recommend such programmes, given the mixed evidence concerning the effectiveness of PSA tests in an asymptomatic population (Calonge et al., 2008). Such tests are nevertheless frequently used to explore the risk of prostate cancer. Pap smears to detect cervical cancer are recommended at intervals of 3–5 years for Spanish women between the ages of 25 and 69 years (Ministerio de Sanidad, Servicios Sociales e Igualdad, 2013). Since the mid-1980s, Pap smears have been implemented in most Spanish regions, albeit not in an organised screening programme, but in the form of opportunistic screening. Their use thus depends on the spontaneous initiative of individual women or their physicians. In contrast, the early detection of breast cancer through mammograms is organised in a population-based screening programme that systematically identifies all Spanish women between the ages of 50 and 69 years (in some regions, 45–69 years) and invites them to have a mammogram every two years (Basu et al., 2018). Cholesterol readings to screen dyslipidaemia are recommended for women 45 years of age and older and for men beginning at the age of 40 years (San Vicente Blanco et al., 2008). Blood-pressure checks to detect hypertension (and other medical conditions) are recommended every two years for people 40 years of age or older (Pérez, 2002).

The main independent variable in this study is socioeconomic status (SES). In order to avoid problems of multicollinearity due to the inclusion of both educational level and social class in the statistical models, we decided to use a composite indicator of SES. To this end, we performed a principal component analysis (PCA) including educational level and social class (See supplementary material for the operationalisation of both variables and PCA). This analysis yielded one extracted factor with a satisfying internal consistency (Cronbach's alpha = 0.72), which explains approximately 78% of the variance. The integrated metric indicator ranges between −1.53 and 2.14, with higher scores indicating higher SES.

In our analysis, we account for age (ranging from 50 to 69 years), gender (female or male) when applicable, work status (employed, unemployed or non-employed), marital status (married, separated or divorced, single or widower), self-perception of the risk of developing cancer in one's lifetime (high/very high or low/very low) and the presence of other chronic diseases other than cancer (no or yes). Finally, we consider whether respondents had any relatives or friends who had died of cancer or who had been diagnosed with cancer (no or yes), as contact with the disease through family or close friends can also play a relevant role in the regular use of checks and screenings (Lerman, Rimer, Trock, Balshem, & Engstrom, 1990; Rees, Martin, & Macrae, 2008).

### Statistical analyses

The statistical analyses were performed in three steps. First, for each preventive measure, we calculated both the rate of adoption and the rate of spontaneous knowledge amongst the Spanish population between the ages of 50 and 69 years. The rate of spontaneous knowledge was retrieved from the respondents' answers to the question concerning the cancer-detection techniques of which they were aware. Based on the rates of adoption and knowledge, we linked each preventive measure to a specific diffusion stage, following the logic that low rates imply an early diffusion stage, and *vice versa*. Second, we estimated the adoption rate for each preventive practice by age, gender (when applicable), work status, marital status, self-perception of the risk of developing cancer, and having relatives or friends who had either died from or been diagnosed with cancer. Third, to examine whether and how SES is associated with the adoption of preventive healthcare, we performed logistic regressions for each preventive practice. Regression coefficients were adjusted for age, gender (when applicable), work status, marital status, self-perception of the risk of developing cancer, presence of other chronic diseases and having relatives or friends who had either died of or been diagnosed with cancer. Odds ratios (OR) and the accompanying 95% confidence intervals (CI) are presented to facilitate interpretation. Finally, to explore how the effects of SES emerge and evolve along the diffusion cycle, we displayed the OR on a figure. Logistic models were computed using SPSS software version 22. Population weights according regional survey design were applied to arrive at valid estimations for the country as a whole.

## Results

The rate of spontaneous knowledge and the rate of adoption for each preventive practice are displayed in Table 1, along with the diffusion stage linked to each preventive measure based on these rates.

We determined that FOB testing was in an early stage of diffusion, as it had the lowest rates of both spontaneous knowledge (3%; 95% CI: 2.4–3.9) and adoption (9%; 95% CI: 8–10.4). This is not surprising, given that the first relevant screening programmes for colorectal cancer were not implemented until 2000 in Catalonia, followed by the Valencian Community in 2005 and Murcia in 2006. Other regions either started implementing FOB tests after 2006 or were in a pilot phase. Based on our results, we classified PSA tests as being in the early majority stage of diffusion, as only 12% of the men spontaneously indicated being aware of PSA tests as a screening method for detect cancer, and approximately 35% had used this type of screening in the past two years. Pap smears were classified as being at the end of the early majority stage or the beginning of the late majority stage. Of all female respondents, 23% spontaneously identified the Pap smear as a test for the early detection of cancer, and the rate of adoption in the past two years was 64%. With spontaneous knowledge and adoption rates of 53% and 81%, respectively, we classified mammograms in the late majority stage. Cholesterol readings and periodic blood-pressure checks were classified as being in a late stage of diffusion, as approximately 83% of all respondents between the ages of 50 and 69 years reported having had cholesterol readings taken, and 84% reported having had their blood-pressure checked within the two years prior to the survey.

The unadjusted adoption rates for each preventive measure according to SES are displayed in Table 2. Although the results reflect some SES differences in the adoption of FOB tests (SES I = 9.7% vs. SES IV = 5.9%), these differences were not statistically significant ($p = 0.205$). Prominent significant ($p < 0.001$) differences between the highest and lowest SES groups emerged for PSA tests (SES I = 43.7% vs. SES IV = 30.9%). The socioeconomic gradient was also evident in the case of Pap smears (SES I = 77.2% vs. SES IV = 59%; $p = 0.001$) and mammograms, albeit less pronounced (SES I = 87.7% vs. SES IV = 79.6%; $p = 0.021$). Very slight differences according to SES were observed for cholesterol readings and blood-pressure checks, but these were not significant ($p = 0.182$; $p = 0.351$).

**Table 1.** Diffusion stages of the preventive practices in the Spanish population.

| | Spontaneous knowledge amongst general population (%) | | | Spontaneous knowledge amongst population aged 50–69 years (%) | | | Rate of adoption amongst population aged 50–69 years in the past two years | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Prevalence | 95% CI | | Prevalence | 95% CI | | Prevalence | 95% CI | | Stage of Diffusion |
| FOB tests | 2.50 | 2.20 | 2.80 | 3.18 | 2.4 | 3.9 | 9.17 | 8.0 | 10.40 | early stage |
| PSA tests | 5.27 | 4.80 | 5.80 | 12.07[a] | 9.9 | 14.2 | 35.52[a] | 32.34 | 38.70 | early majority |
| Pap Smears | 13.00 | 12.30 | 13.70 | 23.29[b] | 20.7 | 25.8 | 64.23[b] | 61.30 | 67.10 | early majority/late majority |
| Mammograms | 36.03 | 35.00 | 37.10 | 53.43[b] | 50.4 | 56.4 | 81.50[b] | 79.20 | 82.80 | late majority |
| Cholesterol readings | No information | – | – | No information | – | – | 83.05 | 81.50 | 84.60 | late majority/laggards |
| Blood-pressure checks | No information | – | – | No information | – | – | 84.13 | 82.60 | 85.70 | late majority/laggards |

[a]Only men; [b]Only women.

**Table 2.** Unadjusted rate of adoption, by SES.

| | FOB tests | | | PSA tests | | | Pap Smears | | | Mammograms | | | Cholesterol readings | | | Blood pressure checks | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | % | 95% CI | | % | 95% CI | | % | 95% CI | | % | 95% CI | | % | 95% CI | | % | 95% CI | |
| SES | | | | | | | | | | | | | | | | | | |
| SES I (≥1) | 9.7 | 6.75 | 12.58 | 43.7 | 36.7 | 50.9 | 77.2 | 71.4 | 83.1 | 87.7 | 83.2 | 92.2 | 79.6 | 75.4 | 83.8 | 82.1 | 78.2 | 86.1 |
| SES II (1–0) | 10.3 | 7.3 | 13.4 | 39.6 | 32.7 | 46.6 | 71.2 | 65.2 | 78.6 | 88.7 | 84.0 | 93.3 | 84.6 | 80.7 | 88.5 | 85.6 | 81.8 | 89.4 |
| SES III (0–−1) | 8.4 | 6.5 | 10.2 | 24.4 | 20.4 | 28.5 | 62.8 | 61.2 | 70.5 | 81.5 | 77.8 | 85.2 | 82.5 | 79.8 | 85.3 | 82.3 | 79.6 | 85.1 |
| SES IV (≤−1) | 5.9 | 3.2 | 8.7 | 30.9 | 20.9 | 41.0 | 59.0 | 52.1 | 65.9 | 79.6 | 73.9 | 85.2 | 78.5 | 73.2 | 83.8 | 79.9 | 74.8 | 85.1 |
| Chi$^2$ test (Sig.) | 4.58 (0.205) | | | 28.49 (0.000) | | | 17.13 (0.001) | | | 9.69 (0.021) | | | 4.86 (0.182) | | | 3.27 (0.351) | | |

SES: quartiles of the composite index from principal component analyses. SES I represents the highest socioeconomic group and SES IV the lowest.

In Table 3, we present the results of our exploration of whether SES inequalities become manifest in the uptake of multiple preventive measures (in line with the propositions of FCT). In this regard, the results indicate that not all of the six preventive practices were subject to significant SES inequalities. More specifically, we could not confirm SES inequalities in the use of FOB tests (OR = 1.14; 95% CI: 0.96–1.35), blood-pressure checks (OR = 1.07; 95% CI: 0.96–1.20) and cholesterol readings (OR = 1.06; 95% CI: 0.95–1.18). In contrast, we observed that higher SES increases the likelihood of using PSA tests (OR = 1.50; 95% CI: 1.28–1.75), Pap smears (OR = 1.33; 95% CI: 1.14–1.55) and mammograms (OR = 1.31; 95% CI: 1.08–1.59).

The results presented in Table 3 are accompanied by Figure 2, in which we present the OR for SES and their accompanying 95% CI for each preventive practice. As indicated in this figure, the extent of SES inequalities in the adoption of preventive practices varies significantly according to the diffusion stage of each preventive measure – the higher the OR, the greater the influence of SES and, therefore, the greater the inequality in the adoption of the preventive practice. In line with our expectations, the influence of SES appeared to emerge for preventive practices in an early stage of diffusion (in our case, FOB tests), and it was prominent for measures that were classified as being in the early majority stage (PSA tests) and the intermediate (between the early and late majority) diffusion stage (Pap smears). As reflected by the results for mammograms in Spain, SES inequalities persisted in the late majority stage, despite the extensive rate of mammography adoption. Finally, in the last stage of diffusion (late majority-laggards), when the dissemination process is close to saturation (as was assumed for cholesterol readings and blood-pressure checks in Spain), we observed no significant SES differences in adoption.

## Discussion and conclusion

The primary aim of the current study was to elaborate on the substantial interconnections between FCT and DOI theory, given that a framework integrating the two theories could provide a more comprehensive understanding of the reproduction and persistence of health inequalities. In the study, we evaluated several of these interconnections empirically, by focussing on the influence of SES on the use of six different preventive practices in Spain, which neatly span the various stages of diffusion of knowledge and adoption.

According to the propositions of FCT, SES influences multiple disease outcomes through multiple risk factors. Our first assumption was therefore that the six preventive measures (FOB tests, PSA tests, Pap smears, mammograms, cholesterol readings and blood-pressure checks) would be subject to SES inequalities (H1). This was the case for the use of cancer screening tests (PSA tests, Pap smears and mammograms), but not for preventive practices at early diffusion stage, like FOB tests, and for cardiovascular diseases (blood-pressure checks and cholesterol readings). This result implies that associations between SES and preventive practices do not always exist at a specific place and time. Drawing on DOI theory, this finding could be explained in part by variations in the association between SES and preventive practices according to diffusion stage (Boscoe & Zhang, 2017; Clouston et al., 2017; Wang et al., 2012). This explanation is in line with the second hypothesis, which states that the extent of SES inequalities in preventive practices varies according to the diffusion stage of each preventive measure (H2a). Moreover, in line with Elstad (2013) and Clouston and colleagues (2016), we observed

**Table 3.** Influence of SES on the regular use of preventive practices at different diffusion stages.

| | FOB tests | | | | PSA tests | | | | Pap Smears | | | | Mammograms | | | | Cholesterol readings | | | | Blood-pressure checks | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | OR | 95% CI | | | OR | 95% CI | | | OR | 95% CI | | | OR | 95% CI | | | OR | 95% CI | | | OR | 95% CI | | |
| Age | 1.01 | 0.98 | 1.05 | | 1.07 | 1.03 | 1.11 | *** | 0.95 | 0.92 | 0.98 | *** | 0.99 | 0.95 | 1.02 | | 1.03 | 1.00 | 1.06 | * | 1.04 | 1.01 | 1.07 | ** |
| Women (ref. Men) | 0.78 | 0.55 | 1.09 | | – | – | – | | – | – | – | | – | – | – | | 0.73 | 0.55 | 0.96 | * | 0.93 | 0.71 | 1.23 | |
| Work status (ref. Employed) | | | | | | | | | | | | | | | | | | | | | | | | |
| Unemployed | 1.75 | 1.03 | 2.99 | * | 1.40 | 0.85 | 2.30 | | 1.69 | 0.94 | 3.04 | | 2.00 | 0.94 | 4.24 | | 1.02 | 0.67 | 1.56 | | 0.73 | 0.48 | 1.11 | |
| Non-employed | 1.28 | 0.83 | 1.98 | | 1.15 | 0.76 | 1.74 | | 1.18 | 0.81 | 1.72 | | 1.41 | 0.89 | 2.21 | | 1.26 | 0.90 | 1.76 | | 0.89 | 0.63 | 1.25 | |
| Marital status (ref. Married) | | | | | | | | | | | | | | | | | | | | | | | | |
| Separated or divorced | 1.01 | 0.54 | 1.89 | | 0.87 | 0.48 | 1.58 | | 1.02 | 0.55 | 1.86 | | 1.24 | 0.56 | 2.74 | | 0.86 | 0.52 | 1.42 | | 0.80 | 0.49 | 1.30 | |
| Single | 0.59 | 0.29 | 1.20 | | 0.56 | 0.32 | 0.99 | * | 0.85 | 0.48 | 1.50 | | 0.58 | 0.31 | 1.10 | | 0.73 | 0.47 | 1.14 | | 0.90 | 0.56 | 1.43 | |
| Widower | 0.72 | 0.36 | 1.46 | | 0.41 | 0.16 | 1.10 | | 0.94 | 0.61 | 1.45 | | 0.84 | 0.50 | 1.44 | | 0.74 | 0.45 | 1.20 | | 0.70 | 0.43 | 1.15 | |
| Self-perception of the risk of developing cancer in one's lifetime (ref. Low or very low) | 1.42 | 1.02 | 1.97 | * | 1.23 | 0.90 | 1.68 | | 0.73 | 0.55 | 0.98 | * | 0.92 | 0.64 | 1.33 | | 1.27 | 0.97 | 1.68 | | 0.87 | 0.66 | 1.14 | |
| Relative or friend died from cancer (ref. No) | 1.88 | 1.15 | 3.06 | * | 1.65 | 1.13 | 2.40 | ** | 1.18 | 0.82 | 1.72 | | 1.46 | 0.94 | 2.24 | | 1.32 | 0.97 | 1.78 | | 1.48 | 1.09 | 2.01 | * |
| Relative or friend currently has cancer (ref. No) | 1.47 | 1.05 | 2.06 | * | 1.42 | 1.04 | 1.93 | * | 1.16 | 0.88 | 1.55 | | 1.21 | 0.85 | 1.73 | | 1.31 | 1.00 | 1.72 | * | 1.29 | 0.98 | 1.69 | |
| Other chronic diseases or health problems (ref. No) | 1.04 | 0.75 | 1.45 | | 1.34 | 0.99 | 1.82 | * | 1.09 | 0.82 | 1.45 | | 1.20 | 0.84 | 1.71 | | 1.59 | 1.22 | 2.08 | *** | 1.76 | 1.34 | 2.31 | *** |
| SES | 1.14 | 0.96 | 1.35 | | 1.50 | 1.28 | 1.75 | *** | 1.33 | 1.14 | 1.55 | *** | 1.31 | 1.08 | 1.59 | ** | 1.02 | 0.89 | 1.17 | | 1.01 | 0.88 | 1.17 | |
| $R^2$ Nagelkerke | 3.9% | | | | 13.20% | | | | 6.00% | | | | 3.40% | | | | 4.60% | | | | 4.60% | | | |
| *N* | | 1904 | | | | 889 | | | | 943 | | | | 965 | | | | 1642 | | | | 1642 | | |

***$p < 0.001$; **$p < 0.01$; *$p < 0.05$.

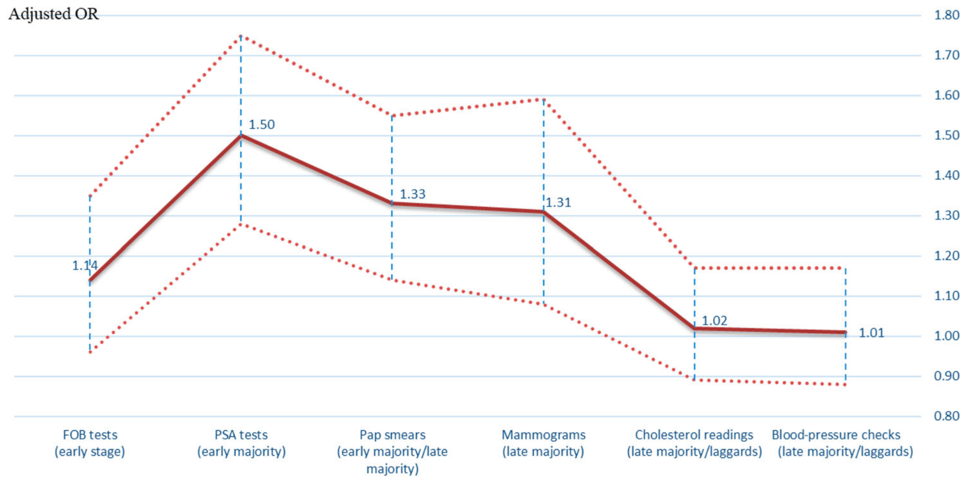


**Figure 2.** Trend of SES influence at different diffusion stages. The dotted line represents the 95% confidence interval of the OR.

that an early diffusion stage is accompanied by the absence or emergence of SES inequalities, with an intermediate stage accompanied by a high level of inequalities and an advanced stage with the reduction or absence of inequalities. The extent of SES inequalities in Spain for each preventive measure studied was in accordance with its diffusion stage: SES inequalities seemed to be emerging for FOB tests (early stage of diffusion), the greatest for PSA tests (early majority), smaller but existent for Pap smears and mammograms (intermediate early-late majority and late majority stage) and absent for cholesterol readings and blood-pressure checks (late diffusion stage). These findings suggest that the social institutionalisation of preventive practices can strongly reduce social disparities.

Our results have several theoretical and empirical implications for the persistence of social inequalities in health. First, we suggest that a theoretical framework integrating FCT and DOI theory could help to develop a more extensive and complete explanation for the 'health inequality paradox' in more generous and extensive European welfare states (Mackenbach, 2012, 2017). Despite improvements in their ability to control diseases by extending preventive policies and effective reforms aimed at the pre-distribution of health and focusing on the redistribution of resources, rather than on 'capabilities' (Abel & Frohlich, 2012), modern welfare states have not succeeded in reducing health inequality since the 1990s (Pega, Blakely, Carter, & Sjöberg, 2012). Moreover, most public health programmes have focused on 'downstream' interventions (e.g. effecting lifestyle changes) to reduce health inequalities. As suggested by FCT (Link & Phelan, 1995), however, it is crucial to address the underlying causes of the causes or 'upstream' social conditions (Marmot, 2005; Syme, 2002; Watt, 2007). Although DOI theory has been considered as a possible explanation for this apparent 'health inequality paradox', the argument has been advanced that, on its own, the theory may explain only the 'temporary widening of health inequalities when major improvements in population health occur that are mediated by behaviour change' (Mackenbach, 2012, p. 765). According to our interpretation, however, the interconnections between FCT and DOI theory overrule this argument. By introducing the idea of fundamental causality, the cycle described by

DOI theory could be repeated constantly over time, in line with the distribution of flexible resources linked to SES. This may help to explain the widening and diminishing of health inequalities because of the accumulated effect of disseminating multiple preventive practices, innovations and new health knowledge over time. The combination of FCT and DOI theory thus clearly reveals that existing explanations for the persistence of social inequalities in health tend to be too static in their focus on a 'paradox', as they tend to ignore the shifting relationship between inequality and the use of preventive innovations along the S-shaped curve predicted by DOI theory. Future research should therefore draw on the interconnections of FCT and DOI theory to adopt a more dynamic approach.

Second, variations between social groups and cohorts in the extent of inequalities in the use of preventive care reveal an explanatory shortcoming of FCT. More specifically, as observed by Masters, Link, and Phelan (2015, pp. 26–27), the explanatory core of FCT, which rests on the relevance of purposeful use of individual flexible resources, lacks any theoretical elaboration on how the effective benefits of these resources vary across differing social contexts. However, the conjunction with DOI theory adds a contextual and temporal dimension to FCT. This integration may help to improve the conceptualisation of the dynamic causal relationships between SES and health outcomes, in addition to enhancing the exploration of how this dynamic association is shaped by contemporary social changes/processes in the diffusion of relevant health knowledge. Further, given that the reproduction of health inequalities does not depend exclusively on resources at the individual level, the next theoretical step might be to identify and elaborate on the elements that shape the 'preventive diffusion context' at different levels (e.g. neighbourhoods, regions, countries). Particularly when studying complex contagions such as preventive practices, which require social reinforcement to diffuse, attention should be paid to network structures (i.e. who is connected to whom) (Centola, 2010; Centola & Macy, 2007; Rogers, 1983). Findings from an experimental study with artificially structured online communities have demonstrated that individual adoption of health innovations is much more likely to diffuse in clustered neighbourhood networks, due to social reinforcement of multiple neighbours (Centola, 2010; Guilbeault et al., 2018). This mirrors the importance of neighbourhoods' social cohesion and social capital for the likelihood of adoption (Kawashi & Berkman, 2014). It is, therefore, likely that connections exist between the different adopter categories and their neighbourhoods' cohesiveness. Exploring how preventive diffusion contexts and network structures contribute to equality in the adoption of multiple preventive innovations is, thus, a promising research area for advancing understanding of variations in health inequality across places and social strata.

Third, the diffusion model has been criticised for its inability to reveal any explanatory mechanisms beyond the imitation of behaviour (Mackenbach, 2012). The underlying idea is that an innovation is first adopted by high-status individuals (social 'leaders') and then by low-status individuals (social 'followers'), who imitate the behaviour of the 'leaders' (Lindbladh et al., 1997). This argument suggests that inequalities may be 'largely temporarily unavoidable as a consequence of modernisation' (Mackenbach, 2017, p. 16). Although imitation mechanisms could be operating in the process, the conjunction of FCT and DOI could open the door for investigating alternative mediating paths by exploring the role played by various 'meta-mechanisms' along the diffusion cycle of preventive innovations. In addition to the relevance of personal agency, Freese and Lutfey (2011) stipulate three meta-mechanisms that contribute to the reproduction of health inequalities: (1) spill-over effects between individuals through their social networks; (2) socially structured health preferences as part of a habitus, related to social positions (Bourdieu, 2002); and (3) institutional agency, in which institutions (e.g. the healthcare system and schools) contribute to the preservation of health inequalities to the extent that they treat individuals differently according to their social class, gender, race or age. We invite scholars to explore the relative importance of these meta-mechanisms along the diffusion cycle of relevant preventive innovations, thereby contributing to further theoretical development within the field.

Several limitations of the present study should be noted. First, we ignored differences in the ease of using various preventive practices, as well as in the ways in which they are provided and in how these differences affect their diffusion through a population. More specifically, it has been shown that preventive practices provided by specialists (Lorant, Boland, Humblet, & Deliège, 2002), which require considerable behavioural effort (Goldman & Lakdawalla, 2005) and which depend upon the spontaneous initiative of individuals or physicians (Palència et al., 2010; Walsh, Silles, & O'Neill, 2011; Willems & Bracke, 2018), enhance inequality more than is the case for preventive practices provided by general practitioners, which involve simple technologies and are organised in population-based programmes (e.g. organised cancer screening programmes). We also paid no attention to the potential accumulation of disadvantage across the lifespan and the timely initiation of preventive care (Missinne, 2015). Given that some preventive care takes place later in life, we could expect this care to be more unequal. We addressed this issue by limiting the sample to the same age categories. Future research could apply an integrated theoretical framework of FCT and DOI theory to a more complete exploration of these lifespan effects, in addition to considering potential cohort effects that might occur in response to social changes relating to the diffusion of preventive social practices. Second, our data did not allow us to consider the temporal diffusion of each preventive practice along the path suggested by the S-shaped curve. At a minimum, this would require a repeated cross-sectional design extended over a long period. Finally, this study focuses on the diffusion contexts of multiple preventive practices in one specific country. Future research should study the diffusion cycle of multiple preventive measures in cross-national comparative perspective, integrating questions regarding how specific characteristics of macro-diffusion contexts and institutional agency can influence the magnitude of health inequalities across countries or other relevant geographic areas.

In conclusion, our findings highlight the importance of the diffusion of innovative preventive technologies and its impact on the generation and reduction of inequalities in preventive care practices as greater control over diseases is developed (Phelan & Link, 2005). Taken together, FCT and DOI theory can provide an integrated framework with which to study health inequalities in the adoption of preventive practices from a temporal and comparative perspective. This integration has the potential to bring 'social change' back into the study of health inequalities, which is essential in order to understand transitions in health practices (Dixon & Banwell, 2009). The findings that we have reported suggest that, while social inequalities in *specific* preventive care practices tend to arise and disappear along the continuing process of technological innovations in cancer screening, they tend to be reproduced *overall*. This paradox requires our full attention.

## Disclosure statement

No potential conflict of interest was reported by the authors.

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