Does Home Health Care increase the probability of 30-day hospital readmissions? Interpreting coefficient sign reversals, or their absence, in binary logistic regression analysis.

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Abstract

Data for 30-day readmission rates in American hospitals often show that patients that receive Home Health Care (HHC) have a higher probability of being readmitted to hospital than those that did not receive such services, but it is expected that when control variables are included in a regression we will obtain a "sign reversal" of the treatment effect. We map the real-world situation to the binary logistic regression model, and we construct a counterfactual probability metric that leads to necessary and sufficient conditions for the sign reversal to occur, conditions that show that logistic regression is an appropriate tool for this research purpose. This metric also permits us to obtain evidence related to the criteria used to assign HHC treatment. We examine seven data samples from different USA hospitals for the period 2011-2017. We find that in all cases the provision of HHC increased the probability of readmission of the treated patients. This casts doubt on the appropriateness of the 30-day readmission rate as an indicator of hospital performance and a criterion for hospital reimbursement, as it is currently used for Medicare patients.

Keywords: control variables, treatment effect, causality, ignorability, consistency.

1 Introduction and motivation.

The "direction of influence" is of prime importance in the way we explore and try to explain real-world phenomena, since it reflects a fundamental qualitative feature that is crucial on its own and distinct from the strength of a relationship. For example, the direction of influence is critical in assessing government policy –sometimes more so than the policy's actual quantitative impact. In many cases, a negative stance against a contemplated government action is based on the argument that the policy will have the opposite effect from the one intended. Due to the existence of confounding relationships between variables, it is conceivable that empirical studies may produce the wrong direction of influence if these confounders are not taken properly into account. Therefore, the ability of our models to allow for and be able to reflect a "coefficient sign reversal" when we feed them with all relevant data is important. Dawes (1979) discusses the basic finding that statistical models are better than experts in predicting, but it is the experts that have the knowledge and the responsibility to choose wisely the variables to include in a statistical model (those that have "conditionally monotonic" relations with the outcome, as he puts it, meaning an unambiguous direction of influence). Our argument here is that a statistical model can also be used to test for preconceptions and professional biases these same experts may have.

The 30-day re-hospitalization or readmission rate (henceforth simply "RA risk") has been an indicator closely tracked for American hospitals, even more so in recent years after the Affordable Care Act provided for monetary penalties for those hospitals that have "too many" Medicare patients readmitted in this window.[1] Our specific concern in this article is the effect on RA risk due to the provision of Home Health Care (HHC) to some patients after a hospitalization. Such services typically include skilled nursing, physical therapy

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[1]See *https://www.cms.gov/medicare/medicare-fee-for-service-payment/acuteinpatientpps/readmissions-reduction-program.html* where we read "Section 3025 of the Affordable Care Act added section 1886(q) to the Social Security Act establishing the Hospital Readmissions Reduction Program, which requires CMS to reduce payments to IPPS hospitals with excess readmissions, effective for discharges beginning on October 1, 2012. The regulations that implement this provision are in subpart I of 42 CFR part 412 (412.150 through 412.154)." It was further clarified in the final applicable rule that the readmission refers to the 30-day period, see *https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/AcuteInpatientPPS/HRRP-Archives.html*. Website accessed August 27, 2018.

and occupational therapy visits. The additional care and monitoring such patients receive would be expected to result in favorable outcomes, including lower readmission rates.

As it turns out, patients receiving HHC readmit at a higher rate than others do. In technical terms, the unadjusted RA Risk of the treated group is higher than the unadjusted RA Risk of the untreated group. This fact is routinely attributed to greater severity of illness and more acute need for care, and control for such factors should logically be expected to reverse the direction of the estimated effect of HHC on readmissions. But in study after study, as we will detail in a later section, the effect of HHC provision on RA risk is invariably estimated as being positive, increasing the RA risk even when controls are introduced in the relation. In light of such counterintuitive empirical results we believe it is important to develop concepts that allow for a deeper understanding of why the data are telling us the opposite of what we expect. The purpose of this paper is to develop such an interpretative framework by revisiting the well-known binary logistic regression model and showing how it can be fruitfully used for the task.[2]

First, we need to rationalize the fact that, in the absence of controls and risk-adjustment, the direction of influence is estimated as having the opposite sign from the anticipated one. To this end, we note that in the case of RA risk and HHC services, the treatment, although deemed beneficial, is not expected to spectacularly reverse the situation of the treated subjects. In other words, controls are jointly more influential than the treatment in affecting the outcome, and this is why we are not surprised when patients treated with HHC services re-admit at higher rates than untreated patients. But we do expect to detect the treatment's beneficial influence after controlling for the relevant factors, through a coefficient sign reversal.

As always, of importance for the results are the control variables that are selected in the attempt to bring to the surface this sign reversal. But the situation we focus on is an example of *directed* treatment. Treatment is not assigned randomly to subjects in order to statistically assess its effectiveness –on the contrary, individuals are included in the

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[2]This framework will be more generally applicable since there are many other real-life situations where we expect a reversal of the estimated direction of influence of the treatment after controlling for relevant factors. Remedial tutoring to weak students vis-a-vis their course results after treatment, or suicide rates of patients in psychotherapy are two such examples.

treatment group purposefully, based on certain criteria that to one or the other degree stem from official guidelines and operational procedures, or at least from professional experience that is common consensus. This is an important and helpful aspect of the real-life activity under study since it permits us a much higher degree of certainty when selecting the factors we should control for: they should be those that, at least on paper or in theory, guide the treatment assignment process. This mitigates the threat of having "omitted variables" distorting the obtained results.

The flip-side is that, if faced with unexpected and counterintuitive empirical findings, we cannot as easily invoke the "omitted confounders" argument to disqualify these results, since we just asserted that the choice of controls was adequate. In such a situation, if we insist that the empirical findings are misleading and that other omitted variables exist that may confound the relationship between the treatment and the outcome to the point of a "wrong sign", we essentially accept that *the treatment was not assigned according to the criteria that were supposed to be used*, since we have already included in the control vector those variables that guide the treatment assignment "officially".[3]

The framework that we will develop allows us to also obtain indirect statistical evidence on whether other considerations than the severity of the health condition of the patients has affected the decision to assign HHC treatment. And if such evidence surfaces, it will raise the question why the criterion of treatment assignment deviated from what it should have been. Given that the RA risk affects hospital reimbursement for Medicare patients, such a finding could point to the possibility of an attempt to "game the system" for financial reasons: instead of providing HHC treatment to the patients that need it most, we assign it to those in better shape for which the "mild" HHC treatment will have a better chance of averting a hospital re-admission, reducing the overall re-admission rate of the hospital and avoiding financial loss. This would be a case of using resources to "go for an easy win," a well-known *strategic* temptation when gains and losses are involved.[4] A coefficient sign-

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[3]We must mention, though, that analyses based on health claims and administrative data do not take into account socioeconomic factors like race or ethnicity that, "officially" or not, may play a role in the phenomenon we study.

[4]In fact, we could reasonably argue that the CMS financial disincentive justs strengthens the already existing goal of the hospitals to keep discharges from coming back, in light of their limited resources.

reversal would abolish such concerns (statistically); a persistent counterintuitive empirical finding (no sign reversal) would keep them alive. But with our methodology, we can examine such concerns even if the sought-after coefficient sign reversal does not happen. And if we do not obtain evidence that the likely answer to the counterintuitive empirical findings is the existence of omitted variables, we will then be healthily forced to accept them at face value and reconsider our preconceptions about Home Health Care services and their expected effects on the health of patients and their hospital readmission rates.

We organize the rest of this paper as follows: in Section 2 we analyze the phenomenon under study and we show why the binary logistic regression model is especially suitable for its estimation and analysis. In Section 3 we examine the logistic regression model variants with and without control variables; we show their interrelation and clarify what each model really tells us about the treatment variable and its effect on the outcome. In Section 4 we formally obtain necessary as well as sufficient conditions for a treatment coefficient sign reversal to occur, and we also show how the "readmission excess probability" measure (or "excess RA risk") before and after treatment can further illuminate the situation. In Section 5 we present and discuss results from seven data samples that address the relationship between Home Health Care and 30-day readmissions. The Appendix contains some simple mathematical derivations and elaborations on results presented in the main text.

2 The causal relation between RA risk and Home Health Care services and the binary logistic regression model to study it.

Statistical control for the effect of other factors in a treatment-effect relation may be implemented in a number of ways; Propensity Score Analysis (PSA) in its various incarnations comes readily to mind. But we chose binary logistic regression because it matches naturally to the situation under study, which we now present in some detail.[5]

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[5]CMS applies also hierarchical logistic regression, see Krumholz et al. (2006) and Krumholz et al. (2011).

Both a patient's discharge from an acute-care hospital and their readmission will happen if health professionals perceive sufficient change in the patient's health. We can summarize these decision-making processes as depending on the value of a health-risk index that is some sort of weighted average of various health indicators that are selected and jointly assessed based on medical knowledge and experience. But the calculation of its value is not typically done or recorded formally and so the health-risk index is a latent variable. Suppressing the patient/observation index i, let y_d^ℓ denote this health-risk index, with subscript 'd' reflecting the time of discharge. Let $\mathbf{z}$ be the vector of the main health indicators used to construct the health-risk index (our vector of controls), and let u_d represent unmodeled influencing factors. Assuming linearity, we can then write, standing at the timepoint of discharge,

$$y_d^\ell = \beta_0 + \mathbf{z}_d'\gamma + u_d\,.$$

Let T be the binary HHC treatment variable, taking the value '1' if HHC treatment was assigned at discharge. At the time of readmission (or 30 days after discharge if no readmission has occurred), which we will represent by the subscript 'r', the value of the latent health-risk index can be expressed as

$$y_r^\ell = \beta_0 + \beta_1 T + \mathbf{z}_r'\gamma + u_r\,,$$

where u_r represents unforseen health incidents and other influencing factors *after* discharge. Since we have included a constant term, we can assume inconsequentially that $E(u_r) = 0$.

By design, at discharge the value of the health-risk index y_d^ℓ has fallen below the critical level for hospital care. If readmission occurs, the health-risk index value has exceeded the critical value. If readmission did not occur, y_r^ℓ remained below the threshold, which for convenience can be set as being equal to zero (again, the existence of a constant term makes this modeling choice harmless).

We are interested in the conditional probability of readmission, so we should look at the y_r^ℓ relation. Defining the binary variable $y = I\left\{y_r^\ell > 0\right\}$ which represents whether the patient was readmitted to the hospital ($y = 1$) or not, the probability of readmission

conditional on the regressors is

$$\Pr\left(y = 1 \middle| \{T, \mathbf{z}_r\}\right) = \Pr\left(y_r^\ell > 0 \middle| \{T, \mathbf{z}_r\}\right) = 1 - \Pr\left(u_r \le -\beta_0 - \beta_1 T - \mathbf{z}_r'\gamma\right).$$

But the above is nothing else than the formal foundation of the binary logistic regression model.[6] We obtain the model if we assume that $u_r \middle| \{T, \mathbf{z}_r\} \sim \Lambda\left(0, \pi^2/3\right)$ where $\Lambda(g) = (1 + e^{-g})^{-1}$ is the standard Logistic distribution function, with mean zero and variance $\pi^2/3$. Finally, due to the symmetry properties of the distribution, we have

$$\Pr\left(y = 1 \middle| \{T, \mathbf{z}_r\}\right) = \Lambda\left(\beta_0 + \beta_1 T + \mathbf{z}_r'\gamma\right),$$

which is what the binary logistic regression model estimates, typically using maximum likelihood estimation and the likelihood of the observed binary y. Since the above model includes control covariates, we will call it the "extended" model.

The model requires that the controls are measured at readmission (or 30 days after discharge), but we usually have data on the controls at the point of discharge. But the controls are either historical data related to the patient that cannot change (like number of past hospital admissions, or length of stay during the last hospitalization), or binary variables reflecting the presence or absence of a series of illnesses which in all likelihood cannot change status in the very short period of time between discharge and maximum 30 days later. So $\mathbf{z}_r = \mathbf{z}_d = \mathbf{z}$, and the available data can be used with the model.

Regarding the treatment variable T, the decision to assign or not the HHC treatment is certainly influenced by the value of the latent health-risk index at discharge: so T is associated with the controls $\mathbf{z}$ but it cannot be determined completely by them, and for the following reason: at discharge, the value of the health risk index is below zero, otherwise the discharge would not happen. We can say that the decision whether to assign HHC treatment or not will depend on "how close to zero" it is. This requires an assessment mechanism that goes beyond the controls, and it is also represented by the component u_d. So it is *not* the case that $T = T(\mathbf{z})$, but rather that $T = T(y_d^\ell)$. It follows that the dependence between treatment and controls is imperfect, and so that the treatment will *not* be ignorable (see Rosenbaum and Rubin, 1983).

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[6]We note that this foundation for the binary logistic regression is invoked more frequently in econometrics rather than in biostatistics or in medicine.

Non-ignorability is anticipated in non-randomized situations, as is our case, and the general impression is that treatment ignorability is essentially equivalent to "regressor exogeneity" in least-squares linear regression (see e.g. the discussion in Guo and Fraser (2010), pp 30-35). It is implied that if ignorability is absent, then a model like logistic regression will produce estimates that will be biased and inconsistent and so more elaborate setups are needed (and have been developed). But this is not necessarily the case. As discussed above, in the situation we examine both the treatment variable and the controls are determined at discharge from the hospital, i.e they are predetermined with respect to the model disturbance u_r. At the same time u_r represents unforeseen health incidents or even other unmodeled influencing factors (e.g. shared community resources for non-hospital patient care), which in any case operated after discharge. Combined, these lead to the independence of the error term u_r from the regressors, which in turn justifies the use of the logistic regression model, being the main condition for the desirable properties of the maximum likelihood estimator, especially consistency, irrespective of the fact that the treatment is not ignorable.

Another important assumption in treatment effects estimation is the "stable unit treatment value" assumption (SUTVA). As Heckman (2005) p.11, puts it, SUTVA can be decomposed in two exclusion assumptions: first it excludes any effect of the treatment assignment mechanism on *potential* outcomes. In our case where the dependent variable is binary, the potential outcomes are the values 0 and 1. Given the degree of uncertainty still surrounding medicine and healthcare, no method of HHC assignment can make the binary dependent variable (hospital readmission) either zero or one with certainty, eliminating the other potential outcome, and so this component of SUTVA appears easily satisfied. Second, SUTVA excludes "social interactions and general equilibrium" effects, namely, it does not allow for the treatment effect to depend on whether other patients did or did not receive the treatment. In our case "social interactions" translate to the health of a person being influenced by the health of another: absent uncontrolled contagious diseases, such an influence most likely does not exist. A possible "general equilibrium" influence one could think of, is a congestion effect in the assignment of HHC treatments, a case where some patients should have been assigned HHC treatment according to medical protocols, but

weren't because available resources were fully employed in treating other patients. This is an issue to be determined per case, but we note that this part of SUTVA is more easily acceptable in a model as ours, where the treatment variable is binary. One could imagine that partial congestion effects may exist in community-based care, where scarcity of resources could affect the *intensity* of prescribed HHC. Having a binary treatment variable makes any existing congestion issues much less likely to affect the model.

The mapping of the real-world situation to the logistic regression model makes also clear that the "control" variables are inherently part of the causal model, and a logistic regression model specified with only a constant and the treatment variable as regressors (which we will call the "basic" model), does not appear to be anything but a badly misspecified model carelessly ignoring the control variables. Nevertheless, we will see in the next section that it has a useful supporting role to play alongside the extended model.

3   The basic and extended logistic regression models and their interrelation.

Contrary to the approach used in papers that examine the matter of coefficient sign reversal in linear regression (see next section), in the logistic regression context the usual conceptual approach is to start thinking from a model without the control variables and then proceed to include them (because we contemplate an entangled relation between the outcome and the treatment that we attempt to disentangle through the control variables). Introducing the patient/observation index i, we specify first the simple logistic regression model which is our "basic" model, since it does not contain any control variables,

$$P(y_i = 1|T_i) = \Lambda\left(b_0 + b_1 T_i\right), \quad \Lambda\left(g\right) = \left(1 + e^{-g}\right)^{-1}, \quad i = 1, ..., n. \tag{1}$$

We denote by n_1 the number of observations in which $T = 1$ and by n_0 the number of observations in which $T = 0$, $n_0 + n_1 = n$. In this model, the "marginal effect" of the binary variable T on the dependent variable, denoted by $m_B\left(T\right)$, is the change in the probability of the outcome when T takes the value unity compared to when it takes the value zero:

$$m_B\left(T\right) = P\left(Y = 1|T = 1\right) - P\left(Y = 1|T = 0\right), \tag{2}$$

where the constant term is understood to also exist. We show in the Appendix that

$$\hat{m}_B(T) = \Lambda\left(\hat{b}_0 + \hat{b}_1\right) - \Lambda\left(\hat{b}_0\right) = \frac{1}{n_1}\sum_{T_i=1} y_i - \frac{1}{n_0}\sum_{T_i=0} y_i, \tag{3}$$

and that it always holds that sign $\{\hat{m}_B(T)\} = $ sign $\left\{\hat{b}_1\right\}$.

We will use these shortly. Consider now the extended model, where we enhance the specification by a collection of control variables $\mathbf{z}$. We assume that the propensity scores remain bounded away from zero and from unity (because otherwise we would have in the sample patients for which assignment of treatment or not would be a certain event, contaminating the inference we are after), and that conditioning on T does not restrict the joint support of $\mathbf{z}$ (namely, that assignment or not of treatment does not imply that some combinations of values of the control variables are excluded). Medical knowledge is (still) incomplete, and this makes medical decisions inherently stochastic, so it is only natural to accept these assumptions. The extended model was arrived at previously and it is

$$\Pr(y_i = 1|\{T_i, \mathbf{z}_i\}) = \Lambda\left(\beta_0 + \beta_1 T_i + \mathbf{z}_i'\gamma\right), \quad i = 1, ..., n. \tag{4}$$

Note that we changed the notation for the constant term and the treatment coefficients, to indicate that we allow for the possibility that their true values are different from the corresponding true values in the basic model. The point here is that the basic model is not a "wrong" specification, a misspecified model that leads to mistaken results. Specifically, we prove in the Appendix that we can express the estimated marginal effect of T on the probabilities of y in the basic model, *using estimates derived from the extended model*:

$$\hat{m}_B(T) = \Lambda\left(\hat{b}_0 + \hat{b}_1\right) - \Lambda\left(\hat{b}_0\right) = \frac{1}{n_1}\sum_{T_i=1} \Lambda\left(\hat{\beta}_0 + \hat{\beta}_1 + \mathbf{z}_i'\hat{\gamma}\right) - \frac{1}{n_0}\sum_{T_i=0} \Lambda\left(\hat{\beta}_0 + \mathbf{z}_i'\hat{\gamma}\right). \tag{5}$$

Equation (5) holds exactly as a matter of algebra, and it clarifies what the "marginal effect" in the basic model essentially estimates. It is an intuitive probability difference, the average readmission *excess* probability between readmission rates of patients treated with HHC ($T = 1$) and of those untreated ($T = 0$), or "posterior excess RA risk" for short. This metric averages *over any possible control* factors because the relation in (5) does not depend on the choice of the control variables.[7] Since sign $\{\hat{m}_B(T)\} = $ sign $\left\{\hat{b}_1\right\}$, we see then that

[7]This also implies that the basic model can be thought of as modeling the "average" unit of the

this sign tells us whether the treated group is more likely (positive sign), or not (negative sign) to be readmitted than the untreated group, *after* treatment has been administered, and *without* reference to what were the related probabilities before treatment. It reflects a comparison between the treated and the untreated cohorts, and since in our model the treatment is not ignorable, this comparison cannot be considered as providing us with the *direction of influence* of the treatment on the outcome. It only represents how the situation between patient groups morphed after treatment, without revealing whether the assignment and the carrying out of treatment increased or reduced the probability of readmission of the treated. So it is methodologically irrelevant and misleading to debate whether the sign of the treatment coefficient in the basic model is "wrong or right", "reasonable or not", with respect to the apparent direction of influence, since this is not what this sign reveals: it simply indicates whether the treated group of patients was more likely to be-readmitted after treatment than the untreated group. It is an inter-group comparison after treatment, not a comparison of the same group before and after treatment (and with the treatment being non-ignorable, the two cannot coincide). But in the extended model, the coefficient β_1 represents the effect of the treatment, and so its sign does indeed reflect the direction of influence of the treatment on the outcome.

The analysis implies that the basic model without controls is not a causal model, and so $\hat{m}_B(T)$ is not a true "marginal effect." This happens because treatment is not ignorable, and with ignorability absent, the causal, "before and after" model is the extended one. But $\hat{m}_B(T)$, re-interpreted as "posterior excess RA risk", remains a meaningful descriptive statistic. Moreover, when the treatment variable is binary, the coefficient $\hat{b}_1$ from the simple model estimates consistently the log-odds ratio, and so if its value is statistically zero we obtain up-front evidence as to whether the dependent variable is unconditionally independent from the treatment. We see therefore that the cheaply computed basic model has valuable information to offer.

Returning to the extended model, we want also an expression that has always the same sign as $\hat{\beta}_1$. This is the "average treatment effect on the treated", a known quantity in

population. Such "averaging" over health matters would certainly be met with strong objections from health professionals.

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Treatment Effects literature:

$$A\hat{T}T \equiv \frac{1}{n_1} \sum_{T_i=1} \left\{ \Lambda \left(\hat{\beta}_0 + \hat{\beta}_1 + \mathbf{z}'_i \hat{\gamma} \right) - \Lambda \left(\hat{\beta}_0 + \mathbf{z}'_i \hat{\gamma} \right) \right\}, \tag{6}$$

with $\text{sign} \left\{ A\hat{T}T \right\} = \text{sign} \left\{ \hat{\beta}_1 \right\}$. This measures the change in RA risk of the treated group of patients due to treatment. If we add to and subtract from (6) the factor

$$(1/n_0) \sum_{T_i=0} \Lambda \left(\hat{\beta}_0 + \mathbf{z}'_i \hat{\gamma} \right),$$

and re-arrange, we obtain, using (5),

$$A\hat{T}T = \hat{m}_B(T) - \hat{E}_p, \tag{7}$$

where

$$\hat{E}_p \equiv (1/n_1) \sum_{T_i=1} \Lambda \left(\hat{\beta}_0 + \mathbf{z}'_i \hat{\gamma} \right) - (1/n_0) \sum_{T_i=0} \Lambda \left(\hat{\beta}_0 + \mathbf{z}'_i \hat{\gamma} \right). \tag{8}$$

The composition of $\hat{E}_p$ is revealing: its first term, $(1/n_1) \sum_{T_i=1} \Lambda \left(\hat{\beta}_0 + \mathbf{z}'_i \hat{\gamma} \right)$, is an estimated average *counterfactual* probability: what the probability of readmission would have been for those that did receive treatment, *if they had not*. The second term in $\hat{E}_p$ is the estimated average probability of readmission of those individuals that did not receive treatment. So $\hat{E}_p$ measures the *excess RA risk* between the treated and the untreated group *prior to treatment*, based on the selected controls. It is the "prior excess RA risk" and from (8) we see that the average treatment effect on the treated $A\hat{T}T$ can be expressed as the change in the excess RA risk before and after treatment.

We also note that if the subsamples of treated and untreated patients were perfectly balanced with respect to all covariates, by eq. (8) we would have $\hat{E}_p = 0$ and in such a case by eq.(7) we would get $A\hat{T}T = \hat{m}_B(T)$, already in the sample. The population equivalent would be to have $\text{plim} \hat{E}_p = 0$, namely that the (unobservable) counterfactual probability of readmission related to the treated group equals at the limit the (observed) readmission probability of the non-treated group. Such a situation would imply that although conceptually the controls are an integral part of the causal model, balancing/matching would render them statistically redundant and we could estimate only the basic model for the purpose at hand, ignoring the controls. This is nothing else than the starting point of Treatment Effects theory: if the treated and the untreated cohorts are otherwise identical

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(i.e. as regards the controls), to estimate the treatment effect we can use the probability related to the untreated group in place of the unobserved counterfactual probability related to the treated group. When, by the nature of the situation under examination, the two subsamples are not and cannot be balanced, the extended model is what we need to estimate. An analogous situation holds for the property of ignorability: when it does not hold, we need to estimate the extended model.

We close this section with Table 1 that summarizes the interrelations between the two models and the measures we have considered.

Table 1. Interrelations of models and measures.
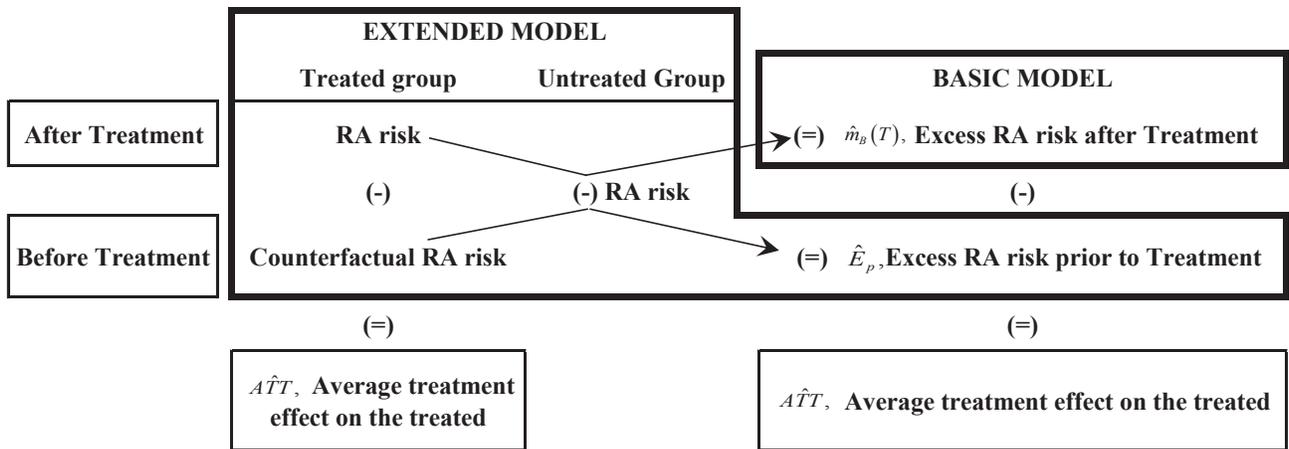
The important thing to note here is that the basic model lives only in the after-treatment stage (and it is only under special conditions like ignorability or matched subsamples that it can be used to estimate the treatment causal effect). In contrast, the extended model, by conditioning on covariates can by design provide results for the situation both before and after treatment, results that will be reliable as long as the estimator is consistent.

4 Necessary and sufficient conditions for a coefficient sign reversal and the excess RA risk measure.

In principle, it is conceivable that the inclusion of control variables in a relation can lead to a sign reversal of the treatment coefficient. In our case these covariates record some aspect

of the patient's medical and clinical history. Suppose that a patient was admitted to hospital for a broken bone, while suffering from osteoporosis. The orthopedic nature of their illness would certainly increase the probability of being assigned HHC for physiotherapy to restore muscle health and mobility after discharge. At the same time, suffering from osteoporosis increases the likelihood of readmission because the patient is prone to broken-bone accidents. So while the HHC treatment per se lowers the readmission probability by improving the myoskeletal health, statistically its relation with the outcome is confounded by the pre-existing condition of osteoporosis: control variables do not just perform a sub-sample balancing act –they affect the dependent variable directly. Including this control in the relation is expected to decompose influences and reveal the beneficial effect of the treatment.

Algebraic rules and conditions governing coefficient sign reversals have been established for ordinary least squares (OLS) and instrumental variables (IV) estimation methods, i.e. for linear regression, starting with Leamer (1975) and continuing with Visco (1978), McAleer et al. (1986), Oksanen (1987), Visco (1988) and Giles (1989). The methodological approach there was to initially consider a comprehensive model, and then examine under which conditions the exclusion of a regressor could reverse the sign of one of the remaining ones (Knaeble and Dutter, 2016, go the other way and also provide a more statistically inclined treatment of the matter). For the non-linear binary choice regression model, the effects of exclusion/inclusion of covariates have been studied with respect to the consequences on the properties of the estimator in an "omitted variables" context; see for example Gail et al. (1984), Yatchew and Griliches (1985), Robinson and Jewel (1991) and Wooldridge (2002), ch 15.

We were unable to find in the literature a study that examines the conditions under which a sign reversal of a coefficient estimate can occur in logistic regression.[8] We therefore determine necessary as well as sufficient conditions for a sign reversal between the estimates $\hat{b}_1$ and $\hat{\beta}_1$. These conditions are compactly expressed through the inequality

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[8]We nevertheless point to the property of "collapsibility" of logistic regression coefficients, see Guo and Geng (1995). If a coefficient is collapsible with respect to a covariate, it cannot change value (let alone sign) as the value of the covariate changes. The necessary and sufficient condition for this property is the mirror image of the first condition for a sign reversal that we derive here.

$$\text{sign}\left\{\hat{\beta}_1\right\} \cdot \text{sign}\left\{\hat{b}_1\right\} < 0. \tag{9}$$

Using the results of the previous Section, we have,

$$\text{sign}\left\{\hat{\beta}_1\right\} \cdot \text{sign}\left\{\hat{b}_1\right\} < 0 \Leftrightarrow \hat{ATT} \cdot \hat{m}_B(T) < 0 \Leftrightarrow [\hat{m}_B(T)]^2 < \hat{E}_p \cdot \hat{m}_B(T). \tag{10}$$

Expression (10) is a necessary and sufficient condition but it also incorporates three necessary conditions for a coefficient sign reversal, the inspection of which should show that the logistic regression model does not impose any artificial restriction but will allow the sign reversal to appear in the extended model, if it is indeed the case that treatment reduces the RA risk when we adjust using controls (allowing for sufficient sample size and statistical power of course).

The first necessary condition is that at least one control variable must be associated with both the treatment and the dependent variable. In other words, we must at least have one "confounder in the classical sense" (see for example Hauck et al., 1991). If either association is missing we obtain $\hat{E}_p \to_p 0$ (see Appendix), i.e. prior excess RA risk is zero in the population and inequality (10) is never satisfied at the limit. This necessary condition has intuition: if not even one control is associated with both the treatment and the dependent variable there is no basis to expect that the introduction of controls will alter the relation between them so much as to reverse its direction. The condition can be checked prior to any estimation step, by looking at the correlation matrix between the outcome, the treatment and the controls. While the correlation matrix detects only linear dependence, experience says that it is very rare to have pure non-linear association accompanied by zero linear dependence.[9]

The second necessary condition is that we must have $\beta_1 \neq 0$. This requires that the presence of the control variables does not make Y and T independent, not even mean-independent: the treatment should not be ignorable conditional on the controls, for a sign reversal to be able to occur. If the treatment is ignorable, i.e. if $\beta_1 = 0$ then we will have $\hat{ATT} \to_p 0$ and so again inequality (10) cannot hold at the population level. In practice we

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[9]Moreover for binary variables, general/non-linear association measures like Spearman's rho and Kendall's tau become identical to Pearson's correlation coefficient.

will learn this after estimating the extended model and obtaining the statistical significance of $\hat{\beta}_1$. This condition also is logical: if the treatment becomes negligible in the presence of controls, any sign reversal in the sample will be spurious.

Note how the two necessary conditions "keep one another in check": we require that the controls are associated with the treatment (first condition), but "not so much" that the treatment becomes ignorable (second condition). For both conditions we obtain statistical evidence, prior to and after estimation.

The third necessary condition of a sign reversal that comes out of eq.(10) says that, if a sign reversal occurs, the excess probability metrics $\hat{E}_p$ and $\hat{m}_B(T)$ will necessarily have the same sign. This implies that the *treatment coefficient* can change sign only if the *excess RA risk* does *not*. If, for example, prior to HHC treatment the treated patients are more likely to be readmitted, then they "have" to remain so after the treatment, if a sign reversal is to be able to occur. This may sound counterintuitive and an artificial restriction imposed by the model, but we elaborate on it in the Appendix to show that any such impression is a case of misleading intuition. Essentially, this condition reflects the requirement that the treatment effect must not be strong, in order for a sign reversal to possibly occur when control variables are present.

Finally, *given* the satisfaction of the necessary conditions, the sufficient condition for a sign reversal is an inequality between absolute values,

$$|\hat{m}_B(T)| \ < \ \left|\hat{E}_p\right| . \tag{11}$$

The posterior excess RA risk $\hat{m}_B(T)$ must be smaller in absolute value than the prior excess RA risk $\hat{E}_p$. So a sign-reversal of the treatment coefficient will always go together with a narrowing of the distance between the treated and the untreated group of patients, as regards their readmission probability. We detail in the Appendix one example to showcase the logic behind this condition.

We conclude that the logistic regression model does not impose any artificial restrictions on the data, and so it is a valid tool also from a technical point of view for examining the effects of the treatment variable on the outcome, including the possibility of a coefficient sign reversal.

But the usefulness of the excess RA risk metric extends further. By examining all possible combinations of signs and relative magnitudes, we obtain six different sub-cases. In practice, the two cases where no coefficient sign reversal occurs can meaningfully and usefully be separated into two subcategories. This breakdown has to do with the assignment criteria for HHC treatment, and whether they align with the patients' readmission probabilities prior to treatment. This additional inference has important consequences from the point of view of the administration procedures actually followed, contrasted with those that "should" be followed. To our knowledge it has not been discussed or measured previously in the literature. We tabulate these six cases in Table 2 that we tried to make self-explanatory.

TABLE 2. The six sub-cases based on the possible combinations of the change in readmission excess probability.

Case	Inequality	Excess RA risk before and after treatment		Extended model	Basic model	Sign reversal	Treatment assignment	Effect on readmission prob.
A	$0 < \hat{m}_B(T) < \hat{E}_p$		It remained positive after treatment but reduced.	$A\hat{T}T$ & $\beta_1 < 0$	$b_1 > 0$	Yes	The patients for HHC treatment were chosen in alignment with their readmission probability.	Treatment reduced the probability of readmission although it did not make it negative, so it remained higher than that of the non-treated patients.
B	$\hat{m}_B(T) < 0 < \hat{E}_p$	Excess RA risk was positive before treatment.	It turned *negative* after treatment.	$A\hat{T}T$ & $\beta_1 < 0$	$b_1 < 0$	No		Treatment reduced the probability of readmission of the treated *below* the readmission probability of the non-treated.
C	$0 < \hat{E}_p < \hat{m}_B(T)$		It increased after treatment.	$A\hat{T}T$ & $\beta_1 > 0$	$b_1 > 0$	No		The treated group had *higher* probability of readmission after treatment than before treatment.
D	$\hat{E}_p < \hat{m}_B(T) < 0$		It remained negative after treatment but reduced in absolute value.	$A\hat{T}T$ & $\beta_1 > 0$	$b_1 < 0$	Yes	Chosen for HHC treatment were patients with *lower* readmission probability.	The treatment *increased* the readmission probability of the treated, but it remained below that of the non-treated.
E	$\hat{m}_B(T) < \hat{E}_p < 0$	Excess RA risk was negative before treatment.	It remained negative after treatment, increasing in absolute value.	$A\hat{T}T$ & $\beta_1 < 0$	$b_1 < 0$	No		The treatment reduced this probability.
F	$\hat{E}_p < 0 < \hat{m}_B(T)$		It became *positive* after treatment.	$A\hat{T}T$ & $\beta_1 > 0$	$b_1 > 0$	No		The treatment *increased* the readmission probability of the treated, *above* that of the non-treated.

We decided to separate the six cases in two subgroups according to whether the patients selected for HHC treatment had on average higher probability of readmission or not prior to treatment, because this directly informs on the criteria and the procedures used to assign HHC. Based only on treatment coefficients signs, case E is the same as case B, and case F is the same as case C. But they differ importantly as regards the sign of the excess RA risk before treatment.

If we obtain any one of the cases D, E, F, that are characterized by negative prior excess RA risk, we have statistical evidence that the patients selected for treatment were those that on average had a *lower* probability of readmission before treatment. As discussed also in the Introduction, such an occurrence can be rationalized as follows: since Home Health Care, by its nature, is not expected to dramatically affect the health status of a patient, it may be the case that, after all, it is assigned to people with less severe illnesses so that they may have a better chance for favorable outcomes compared to patients with more severe health issues. This is the source of statistical evidence as to whether the financial disincentives related to 30-day readmission may lead to actions that attempt to game the system while jeopardizing the health of the patients. So computing prior excess RA risk $\hat{E}_p$ and inspecting its sign provides important information.

Table 2 is the interpretational tool that translates the results of the logistic regression model in real-life terms. We proceed now to present and discuss some empirical evidence.

5 Home Health Care and 30-day readmission rates: results from seven data samples.

5.1 Data, Limitations, and Empirical Results.

We examined data samples, stripped of patient identifiers, from seven acute-care hospitals of different types and from different USA states. Some basic characteristics of these data samples are provided in Table 3. We decided against separating the samples per year, for reasons of statistical power. We note that all the data fall inside the period in which the Affordable Care Act provision for penalizing excessive readmission rates was in effect.

So we will not be able to detect any change in operational procedures that this provision of the law may have had on the assignment of HHC, but also, we are protected from inappropriately mixing the effects of fundamentally different financial/legal environments vis. our object of study.[10]

For the samples in Table 3, the 30-day readmission rate averages 10.0%, ranging from 7.6% to 11.5% for any individual hospital.[11] The proportion of patients receiving HHC averages 23%, ranging from 15% to 42%.

TABLE 3: Basic characteristics of the data samples.

Data Sample a/a	Type of Hospital	State	Years	n_1 Treated with HHC	n_0 Untreated	$n = n_1 + n_0$ Sample Size
1	Urban for-profit	CA	2014-16	5,259	22,932	28,191
2	Urban for-profit	CA	2012-17	14,910	20,531	35,441
3	Urban for-profit	CA	2015-17	2,977	14,447	17,424
4	Suburban for-profit	LA	2012-17	5,319	27,037	32,356
5	Urban non-profit	MA	2012-17	4,829	14,113	18,942
6	Suburban non-profit	MD	2012-17	6,803	37,845	44,648
7	Suburban non-profit	PA	2011-15	12,571	38,468	51,039
			Total	52,668	175,373	228,041

Notes: **Case Mix Index**: sample mean = 1.34 while US hospitals' mean = 1.38 (*T-test value for difference in means* = .29, *p* = .77). **Beds**: sample mean = 279 while US hospitals' mean = 190 (*T* = 1.27, *p* = .20).

Regarding the external validity of the data, we note that we obtained them not through a rigorous random sampling procedure but as "convenience samples" of US hospital populations. While we have to acknowledge the possibility that this may impact the degree to which the samples are representative of the population of interest, the Case Mix Index as well as number of beds show that these hospitals are statistically in the range of the

[10]If a change has occurred, mixing data from the periods before and after could simply offset the quantitative footprints of different behaviors offering a misleading picture of "no-change."

[11]These rates are on the low side for USA hospitals, but not dramatically so given that the sample excludes many high-risk patients discharged to skilled nursing facilities.

corresponding mean values for US hospitals.[12] Moreover, absence of random sampling in the strict sense does not imply the existence of any systematic criterion for observation selection and none was employed. Therefore we expect that selection bias is absent.

Another concern could be the fact that most likely, batches of observations per hospital represent patients for whom the same clinician made the decision whether to assign HHC treatment at discharge or not. Could this affect the statistical independence of observations in each sample? We argue that it essentially doesn't, since here the "clinician" is the "deterministic black box" that takes as inputs the patients' heath indicators and provides a binary decision on HHC treatment. To the degree that health indicators of patients are independent random variables, the HHC decision mechanism does not affect the independence of observations.

We also followed the lead of many hospital administrators in including in our analysis only cases where patients were discharged home, and not to hospice care. The latter are, for the purposes of this analysis, very different from those discharged home either with or without HHC. They clearly would be out of place in a study that addresses the extent to which HHC benefits the patient's health compared to those that don't receive such treatment (since the "treatment" in their case is qualitatively different). With this choice we also deal with the criticism that statistical control, when used to "equalize" markedly divergent subpopulations, may lead to nonsensical results.[13]

Regarding the choice of controls, extensive experience in modeling RA risk has shown that our two primary controls that are based on past utilization usually account for 60-75% of the variance that can be accounted for in this rather unpredictable outcome. We believe that our additional controls -numbering about 30 for most samples- do an adequate job at accounting for the rest, since they are certainly considered by healthcare professionals

[12]The Case Mix Index is a common indicator of the severity of patient diagnoses, used by hospitals to track level of patient needs and by CMS to allocate funding. We obtained these metrics from Kaiser Health News (a major provider of US healthcare-related information) and CMS itself. See also *https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/AcuteInpatientPPS/Acute-Inpatient-Files-for-Download-Items/CMS022630.html*. Webside accessed May 12, 2019.

[13]CMS models consider together patients that were discharged to hospice care, and other dispositions, but it is not clear whether and how they weight differently each patient subgroup in their formulas for hospital penalties.

when assessing the health-risk of a patient and whether to assign HHC services or not.[14]

For five of the seven samples we also did a fairly exhaustive search for interaction effects involving the predictors listed in the note to Table 4 as well as others. Pooling the data provides adequate statistical power, and so we primarily conducted these searches by screening via chi-squared automatic interaction detection (CHAID), then testing any promising interactions in a holdout sample, and further testing via regression. In no case was an interaction found to contribute as much as 0.003 to any model's test-sample pseudo-R-squared, nor did we come across in prior related literature any major roles played by interactions that would be applicable to our models. Although co-existence of illnesses could be expected to have a visible effect on the health risk index and so on the readmission probability, it appears that the leading controls here (recent prior admissions and length of stay) already capture such effects.

Careful selection of covariates may reduce the possibility of omitted variable bias, but this is not the only threat against the internal validity of a model. Other issues, like heterogeneity, functional form, clustering, can affect its validity. For example in our case, in each of the samples approximately 20% of patients had data that appeared in more than one record, reflecting multiple hospitalizations. The fact that we did not account for this clustering constitutes a limitation. But as we will see, the results obtained agree with multiple previous studies that employed different models, specifications and estimation methods, mitigating against model misspecification.

We examined the correlation matrix of outcome, treatment and controls for each sample, in order to determine whether the necessary condition is met that there exist controls that are confounders. This was indeed the case, with leading confounders in all cases being Number of Prior Admissions in the Last 12 Months and Length of Stay, as expected. These past-utilization indicators are known to be important RA predictors; see for example Donzé et al. (2013). We also note that our knowledge of the hospitals included in our empirical study tells us that it is unlikely that any discernible degree of congestion in HHC treatment

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[14]The set of control variables we have used shares many variables in common with those used by CMS and by their Hospital Readmission Reduction Program vendors that have developed thousands of diagnosis- and hospital-specific readmission equations, each incorporating roughly 50 variables. Many of these variables, like our controls, are binary indicators of presence or absence of certain high-risk diagnoses.

existed.

We report the estimates that concern the purpose of this paper in Table 4. As a valida-
tion check we have also performed PSA in some of our samples (observation matching and
sample stratification balancing on propensity scores), and no sign reversal was obtained.[15]

TABLE 4: Results from the basic and extended logistic regression models.

Dependent variable: RA (30-day re-hospitalization, binary) Treatment variable: HHC (Provision of Home Health Care, binary).								
	Basic model	Extend-ed model	Prior excess RA Risk	Posteri-or excess RA Risk	Average treatment effect on the Treated		Probability of readmission	
Data Sample a/a	$\hat{b}_1*$	$\hat{\beta}_1*$	$\hat{E}_p$	$\hat{m}_B(T)$	$\hat{ATT}$	Sub-case per Table 1	**Untreat ed**	**Treated** before treatment	**Treated** after treatment
1	.493	.223	.030	.056	.025	C	.104	.134	.159
2	.287	.232	.007	.028	.021	C	.095	.102	.123
3	.374	.183	.021	.040	.019	C	.104	.125	.144
4	.914	.254	.054	.080	.027	C	.062	.116	.143
5	.584	.419	.014	.050	.036	C	.072	.086	.122
6	.298	.117	.018	.029	.011	C	.097	.115	.126
7	.975	.245	.074	.106	.031	C	.078	.153	.184

*All *p*-values of the treatment coefficients < .01.

Controls included in the Extended model. Some samples: Admission Priority, Admission Service, DNR status. All samples:
No. of Admissions in Prior 12 Mos, Length of Stay, Presence/Absence of 12-29 Diagnoses: Lymphoma, Metastatic Cancer,
Paralysis, DM Without Chronic Complications, DM With Chronic Complications, Liver Disease, Weight Loss, Coagulopathy,
Congestive Heart Failure, Peripheral Vascular Disease, Rheumatoid Arthritis/Collagen Vascular Disease, Solid Tumor
Without Metastasis, Anemia, Fluid/Electrolyte Disorders, COPD, HTN With Complication, AMI, Obesity, HTN, Palpitations,
Cerebral Infarction, PN, Renal Failure, Respiratory Failure, AFIB, GI Bleed, GI Obstruction, Osteoarthritis, Sickle Cell
Anemia.

5.2 Discussion and Conclusions (extended version).

Results for all hospitals are qualitatively the same: in the presence of controls, the mag-
nitude of the treatment coefficient reduced, sometimes substantially, but no sign reversal
was obtained. Also, all samples match case C of Table 2. To describe the results in a

[15]Kurth et al. (2005) compared the extended ("multivariable") logistic regression model to four different
ways of adjustment using PSA on the same data set. Initially they obtained visibly different results between
all four models (even in between PSA-based ones). When they excluded from the data set patients with
very low propensity scores (below 0.05) the results from all five models became similar.

chain-of-events order, the excess RA risk prior to treatment was positive, $\hat{E}_p > 0$ and this tells us that on average HHC treatment was assigned to patients with higher probability of re-admission prior to treatment. Second, because $\hat{\beta}_1 > 0 \Rightarrow \hat{ATT} > 0$, we learn that the HHC treatment *increased* the probability of readmission of the treated patients. Then $\hat{b}_1 > 0 \Rightarrow \hat{m}_B(T) > 0$ just reflects that fact that the probability of readmission of the treated group after treatment was higher than the corresponding probability of the untreated group, an unavoidable result given the previous two.

The finding that $\hat{E}_p > 0$ tells us that HHC was assigned in alignment with the readmission excess probability of the patients. We do not argue that the excess RA risk is the sole factor for such a decision. But the important thing here is that we find no evidence (on average) that HHC was assigned to patients with lower probability of re-admission presumably with possible intent to engender favorable outcomes in terms of the 30-day readmission metric, at the expense of more needy patients. Thus we provide statistical evidence that the negative incentive to reduce the 30-day readmission rate introduced by the Affordable Care Act did not lead to changes to the HHC assignment procedures and decision rules in ways that the health interests of the patients may have been compromised.[16] To our knowledge, this is the first time that an empirical result on this issue is being reported in the literature.

But it is the result $\hat{\beta}_1 > 0 \Rightarrow \hat{ATT} > 0$ that is troubling: the provision of HHC treatment appears to have *increased* 30-day RA risk, in all samples, and in the presence of numerous control variables and confounders. The last three columns of Table 4 give directly the (average) readmission probabilities so that the reader has a clear quantitative view of the matter. The first column of the three is the probability of readmission for the patients that did not receive HHC treatment. The next one is the counterfactual probability of readmission for the HHC-treated group (what their probability of readmission was before receiving the HHC treatment). The last column gives the RA risk of the treated group after receiving treatment. The difference of the last two equals the column $\hat{ATT}$, i.e. the increase of RA risk for the treated group: it ranges from one percentage point (sample 6)

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[16]Berenson et al. (2012) argue that in any case this financial disincentive is not so strong as to offset the financial gains of a hospital from a hospitalization. The overall effects of financial (dis)incentives on healthcare decisions are a complex and important issue, but it is beyond the scope of this paper.

to three-and-a half (sample 5). While such increases may appear "small", in relative terms (not shown in Table 3) they range from an increase of 9.6% (sample 6) to 41.9% (sample 5), that cannot be considered negligible.

It is not the first time that this finding occurs in the literature, and under different research methodologies and model specifications. Weinberger et al. (1996) obtained the same result examining 1396 "severely ill" patients from nine Veterans Affairs Medical Centers. Madigan (2008) found high readmission rates for patients with heart failure under home health care. Madigan et al. (2012) found a U-shaped effect of home-visit frequency on 30-day readmission probabilities. Bradley et al. (2013), in a study based on response questionnaires from 571 hospitals for the period 2010-2011 and patients with heart failure, found that "*some strategies, which seem to link hospitals and outpatient care more closely and have been recommended by quality alliances, were associated with higher [30-day readmission rates].*" Leppin et al. (2014), in a meta study of 47 trials from the period 1990-2013, found on weighted average a reduction in the 30-day readmission probability from the provision of HHC, but a closer look at their results shows that 95% confidence intervals included also the possibility of the opposite result in 35 of these studies.

Our contribution to the empirical literature is not just one more measurement study that supports this result. We do so while at the same time verifying statistically that the assignment of HHC services appears to follow expected medical guidelines that always aim to improve the health status of the patients. Yet HHC again increases the probability of 30-day readmission, and this is a result that appears robust to model (mis)specification and estimation methodologies.

Could we attribute this result to the "level/intensity" of Home Health Care? A connection between this aspect of HHC and readmission rates has been documented in the literature. For example, Harrison et al. (2002) in a randomized control trial found that more comprehensive home care lead to reduced readmissions compared to "standard" such services. Tinetti et al. (2012) also compared two different models of home health care ("usual" and "restorative") and found a similar effect. Wang et al. (2016) in a massive study using 2011-2012 data found that increasing HHC quality led to small but measurable reductions of RA risk. Verhaegh et al. (2014) found that "high intensity" post-discharge

care plans are needed to lead to a reduction in readmission rates for chronically ill patients. Analogous conclusions were reached by Jones et al. (2017). Crucially, these studies compare subgroups of patients that have received different forms of HHC, and so the desirable "negative effect" of higher-intensity HHC essentially means lower readmission rates compared to patients that received HHC of lower intensity. They do not measure whether the readmission probability increased compared to what it was before the assignment of (any version of) HHC. But their findings do suggest that empirical studies where the HHC variable is graded and not modeled as binary may have new insights to offer as regards RA risk.

Should we then contemplate a direct negative effect of HHC on the patients' health? Changes in the industry such as shortages in staffing, reliance on per diem staff, or lack of primary care nurses and therapists may have impacted negatively the quality of HHC. But it is difficult to imagine that, in professions so regulated and with a strong culture for patient safety, quality would dip so low that Home Health Care would become, *on average*, directly detrimental to the health of the patients (and across hospitals in so many regions of the country). Nevertheless, studies that include quality indicators for HHC would certainly be a worthwhile avenue for further research.

This leaves the "administrative/professional" aspects of HHC as possible avenues through which RA risk increases. Weinberger et al. (1996) hypothesized that increased monitoring of the treated patients may uncover previously undetected medical problems. Also that "... *greater access to primary care providers could have improved communication and, in turn, increased readmissions.*" Ma et al. (2018) in a meta-study of related research documented the reasons for 30-day readmissions of patients receiving HHC. Looking at them one can reasonably conjecture that many of these health conditions would have been less frequently detected and/or lead to a readmission, if it weren't for health professionals monitoring the patient. Bradley et al. (2013) characterized the increased RA risk result as "paradoxical" but argued that *"Reducing the informational and logistical barriers to hospitalization may increase readmissions when the practice is designed to reduce readmissions."* An indication that quality matters also in administrative matters is the study by Schoonover et al. (2014) that found that increased complexity in home medication regimens increased RA risk. We

would add the "professional bias" in using hospital services: healthcare professionals may initiate more frequently than the patients themselves a re-hospitalization in order to avoid a potential health incident at home, where the dangers to the patient's health and survival would be greater.

It appears that we have here a clear case of a "molar treatment package" ("molar" with the meaning used in physics), namely an example of the fact that a "treatment" is unavoidably a complex bundle of many components, and the treatment effect that we can estimate is the net effect of all these components together.[17] The HHC treatment appears to have at least two main such components, the "clinical actions" component and the "monitoring" component, that exert opposite influences on the RA risk. Persistent statistical evidence tells us that the second force is stronger than the first.

But this means that the statement "Home Health Care is beneficial to the health of a patient *and* it increases the probability of hospital readmission" *is not a contradiction in terms.* The direct clinical actions improve the health of the patient but the more frequent, expert and sensitive monitoring of the patient as well as a closer association of the patients to the healthcare and hospital system appear to induce and facilitate readmissions at a higher rate.

In turn this makes the use of the 30-day readmission rate as a measure in hospital reimbursement schemes questionable: If interventions like Home Health Care are beneficial to the patients and at the same time lead to an increase in readmission rates of the treated, by using the RA risk as a financial stick as is currently used, we penalize the hospitals that prescribe HHC to benefit their patients. Joynt and Jha (2012) describe perfectly the conflict here when they write *"In fact, there are several factors influencing [increasing] readmission rates that we would not want hospitals to change."* Certainly, operational metrics used to implement policy are usually blunter as instruments than a scientific point of view would tolerate. But when a metric penalizes behavior that it apparently purports to encourage, it underscores the difficult dilemmas of healthcare: "Better health outcomes at lower cost" may at times be an unattainable combination and in such a case the tendency is to avoid the discussion about which of the two targets we would want society and its agents to

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[17]See the discussion in Cook et al. (2002), p.54.

prioritize.

6 Appendix.

6.1 The Basic Logistic regression model.

Estimated by maximum likelihood, the model in (1) has the log-likelihood

$$\ln L = \sum_{i=1}^{n} \{y_i \ln \Lambda (b_0 + b_1 T_i) + (1 - y_i) \ln [1 - \Lambda (b_0 + b_1 T_i)]\}$$

. The maximum likelihood estimator satisfies the first-order conditions

$$\hat{b}_0 : \sum_{i=1}^{n} y_i = \sum_{i=1}^{n} \Lambda \left(\hat{b}_0 + \hat{b}_1 T_i \right) = \sum_{T_i=0} \Lambda \left(\hat{b}_0 \right) + \sum_{T_i=1} \Lambda \left(\hat{b}_0 + \hat{b}_1 \right), \qquad (12)$$

$$\hat{b}_1 : \sum_{T_i=1} y_i = \sum_{T_i=1} \Lambda \left(\hat{b}_0 + \hat{b}_1 \right). \qquad (13)$$

Subtracting (13) from (12) we obtain

$$\sum_{T_i=0} y_i = \sum_{T_i=0} \Lambda \left(\hat{b}_0 \right). \qquad (14)$$

The RHS of equations (13) and (14) do not contain variables that vary with the index, so we have

$$\frac{1}{n_1} \sum_{T_i=1} y_i = \Lambda \left(\hat{b}_0 + \hat{b}_1 \right), \quad \frac{1}{n_0} \sum_{T_i=0} y_i = \Lambda \left(\hat{b}_0 \right), \qquad (15)$$

from which we obtain the expression for the marginal effect,

$$\hat{m}_B (T) = \Lambda \left(\hat{b}_0 + \hat{b}_1 \right) - \Lambda \left(\hat{b}_0 \right) = \frac{1}{n_1} \sum_{T_i=1} y_i - \frac{1}{n_0} \sum_{T_i=0} y_i. \qquad (16)$$

As a mathematical fact, any expression of the form

$$\left(1 + e^{-\xi - h} \right)^{-1} - \left(1 + e^{-\xi} \right)^{-1} = \Lambda (\xi + h) - \Lambda (\xi),$$

will have the sign of h, so we get sign $\{\hat{m}_B (T)\}$ = sign $\left\{ \hat{b}_1 \right\}$.

6.2 The Extended Logistic regression model.

For the extended model of eq.(4), the first-order conditions that are satisfied by the maximum likelihood estimator for the constant and the treatment coefficient become

$$\hat{\beta}_0 : \sum_{i=1}^{n} y_i = \sum_{i=1}^{n} \Lambda\left(\hat{\beta}_0 + \hat{\beta}_1 T_i + \mathbf{z}_i'\hat{\gamma}\right), \tag{17}$$

$$\hat{\beta}_1 : \frac{1}{n_1} \sum_{T_i=1} y_i = \frac{1}{n_1} \sum_{T_i=1} \Lambda\left(\hat{\beta}_0 + \hat{\beta}_1 + \mathbf{z}_i'\hat{\gamma}\right), \tag{18}$$

Working as in the basic model, we can obtain

$$\frac{1}{n_0} \sum_{T_i=0} y_i = \frac{1}{n_0} \sum_{T_i=0} \Lambda\left(\hat{\beta}_0 + \mathbf{z}_i'\hat{\gamma}\right). \tag{19}$$

Note that the left-hand sides of equations (18) and (19) include only data (the dependent variable), and are also present in equation (16) of the basic model. Inserting their right-hand-sides in the latter, we obtain

$$\hat{m}_B(T) = \Lambda\left(\hat{b}_0 + \hat{b}_1\right) - \Lambda\left(\hat{b}_0\right) = \frac{1}{n_1} \sum_{T_i=1} \Lambda\left(\hat{\beta}_0 + \hat{\beta}_1 + \mathbf{z}_i'\hat{\gamma}\right) - \frac{1}{n_0} \sum_{T_i=0} \Lambda\left(\hat{\beta}_0 + \mathbf{z}_i'\hat{\gamma}\right).$$

This is eq. (5) of the main text.

6.3 Some notes on the necessary and sufficient conditions for a sign-reversal in binary logistic regression.

The first necessary condition for a coefficient sign reversal in the main text states that at least one control variable must be associated with both the treatment and the dependent variable. If all controls are independent from the outcome variable we have $\gamma = \mathbf{0}$ which from (8) leads to

$$\hat{E}_p \to_p E\left[\Lambda\left(\text{plim}\hat{\beta}_0 \mid T = 1\right)\right] - E\left[\Lambda\left(\text{plim}\hat{\beta}_0 \mid T = 0\right)\right] = \Lambda\left(\text{plim}\hat{\beta}_0\right) - \Lambda\left(\text{plim}\hat{\beta}_0\right) = 0$$

If all controls are independent from the treatment, then

$$\hat{E}_p \to_p E\left[\Lambda\left(\text{plim}\hat{\beta}_0 + \mathbf{z}_i'\text{plim}\hat{\gamma} \mid T = 1\right)\right] - E\left[\Lambda\left(\text{plim}\hat{\beta}_0 + \mathbf{z}_i'\text{plim}\hat{\gamma} \mid T = 0\right)\right]$$

$$= E\left[\Lambda\left(\text{plim}\hat{\beta}_0 + \mathbf{z}_i'\text{plim}\hat{\gamma}\right)\right] - E\left[\Lambda\left(\text{plim}\hat{\beta}_0 + \mathbf{z}_i'\text{plim}\hat{\gamma}\right)\right] = 0 \ .$$

We used the probability limits of the estimators in the expression and not the true coefficients, to indicate that this result is unaffected by a possible misspecification that would make the estimators inconsistent. In such a situation, any non-zero finite sample value of $\hat{E}_p$ is due to small-sample variability.

The third necessary condition states that if a sign-reversal occurs, the excess probability metrics $\hat{E}_p$ and $\hat{m}_B(T)$ will necessarily have the same sign. This may appear counter-intuitive and artificial. For example, one is immediately inclined to object and ask: *what if the treatment has very strong beneficial effects?* We should then expect, this thinking would go, that it is possible that the positive prior excess RA risk, $\hat{E}_p > 0$ was reduced so much due to treatment, that it turned negative, $\hat{m}_B(T) < 0$. The logistic regression model appears not to allow for that, since the requirement $\text{sign}\left\{\hat{E}_p\right\} = \text{sign}\left\{\hat{m}_B(T)\right\}$ is a necessary condition for a sign reversal. But in reality the "objection" above is a case of misleading intuition, itself misguided by the dramatic effect the term "sign reversal" conveys. The fact is that, as we already mentioned in the Introduction, a sign reversal of the treatment coefficient is an indication of a weak treatment effect, not of a strong one. If the treatment has a strong effect (in either direction), the introduction of controls will not be able to reverse that. The model certainly allows for the case $\hat{E}_p > 0 > \hat{m}_B(T)$, if the treatment effect is beneficial and strong, but here we will not observe a coefficient sign reversal but we will have $\hat{\beta}_1 < 0$, $\hat{b}_1 < 0$ (this is case B of Table 1).

Given the necessary conditions, we obtained also as a sufficient condition that a sign-reversal of the treatment coefficient will always go together with a narrowing of the distance between the treated and the untreated group of patients, as regards their readmission probability. This again may appear counter-intuitive, and this time the misconception comes if we ignore the chain-of-events by thinking first in terms of the results of the basic model and then of the results of the extended model. For if we start with positive readmission excess probability $\hat{E}_p > 0$, and say, we also obtain $\hat{\beta}_1 < 0$ (cases A or B of Table 1), it means that the treatment reduces the probability of readmission, so that *after* treatment, the readmission excess probability will be *lower*, and we will have $\hat{E}_p > \hat{m}_B(T) > 0 \Rightarrow \hat{b}_1 > 0$ (case A of Table 1). If the treatment effect is very strong, then $\hat{m}_B(T)$ will go below zero

(case B of Table 1), and then we will not have a coefficient sign reversal, as discussed just previously. We leave the ruminations for the other possible scenarios and sign combinations to the interested readers that want to persuade themselves further.

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