A Multilevel Event History Model of Social Diffusion: Medical Innovation Revisited

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This article presents a multilevel event history model of social diffusion and applies it to Coleman, Katz, and Menzel’s (1966) data on the adoption of tetracycline by physicians. The simplest form of a multilevel model allows a random intercept. In the present application of this simple model to the Medical Innovation data, structured for an event history analysis, the physicians are nested in city and time. Random intercepts capture effects of contextual conditions that are shared by event history cases with the same city–time status. The intercepts also reflect any baseline internal contagion effects, that is, the proportion of physicians in the city–time network who have adopted the drug at time \( t - 1 \).

Here, I show that Van den Bulte and Lilien’s (2001) finding of an important contextual effect of drug firms’ marketing effort is misleading. I also show that the social network in which physicians are situated significantly contributes to their adoptions, controlling for baseline internal contagion effects and individual-level characteristics of physicians, which have been emphasized in investigations of these data.

Keywords: contagion, diffusion, social networks

1. INTRODUCTION

Coleman, Katz, and Menzel (1966) examined the contribution of networks of interpersonal contact, based on discussion, advice, and friendship relations, to the diffusion of a medical innovation—the adoption of tetracycline, a new antibiotic. Four communities of physicians, located in three small cities and one substantially larger city, were investigated. A physician’s adoption of the antibiotic was ascertained from the prescription records of local pharmacies, which were monitored during three consecutive days each month over a period of 17 months after the introduction of the antibiotic. Upon
the first appearance of a prescription, the prescribing physician was
defined as an adopter during all of the remaining time periods of the
study. Since the antibiotic proved to be efficacious, the assumption
is reasonable that a physician’s first prescription of the antibiotic
implied continued employment of it. A small fraction of physicians
adopted the antibiotic in the first month and, by the 17th month a
substantial fraction of monitored physicians had adopted it. Coleman
et al.’s analysis supported their hypothesis that the physicians’
contact networks contributed to the diffusion of the drug. Subsequent
analyses of the data have provided mixed support for this claim (Burt,
1987; Marsden and Podolny, 1990; Strang and Brandon Tuma, 1993;

Van den Bulte and Lilien’s (2001) analysis of the
Medical Innovation data presents the most startling conclusion among the
analyses that been conducted thus far. They conclude that “prior
evidence of social contagion gained from the Medical Innovation study
by Coleman et al. (1966) is an artifact arising from omitting the
effect of marketing efforts” (Van den Bulte and Lilien, 2001, p. 1411).
Van den Bulte and Lilien’s conclusion is based on the following
formalization of the marketing efforts of the drug companies that were
promoting the adoption of tetracycline:

\[
M^{(t)} = p^{(t)} + (1 - \delta)M^{(t-1)},
\]

where \(M^{(t)}\) is the time \(t\) measure of marketing effort, \(0 \leq \delta \leq 1\) is a
scalar constant, and \(p^{(t)}\) is the amount of published advertising in
month \(t\) in hundreds of pages, \(t = 1, 2, \ldots, 17\) and \(M^{(0)} = 0\). Hence,

\[
M^{(1)} = p^{(1)}
\]

\[
M^{(2)} = p^{(2)} + (1 - \delta)M^{(1)} = p^{(2)} + (1 - \delta)p^{(1)}
\]

\[
M^{(3)} = p^{(3)} + (1 - \delta)M^{(2)} = p^{(3)} + (1 - \delta)p^{(2)} + (1 - \delta)^2p^{(1)}
\]

\[
M^{(4)} = p^{(4)} + (1 - \delta)M^{(3)} = p^{(4)} + (1 - \delta)p^{(3)} + (1 - \delta)^2p^{(2)} + (1 - \delta)^3p^{(1)},
\]

and so on. Two marketing effort measures were computed: one
measure for the marketing effort of Lederle, the major pharmaceutical
company that promoted the antibiotic, and another for the marketing
effort of all other competing firms. Van den Bulte acknowledges
(personal communication, 2008) that this formalization of the firms’
marketing efforts is crucial to their conclusion. Upon request, Van
den Bulte provided the data upon which the 2001 analysis was based,
including the \(p^{(t)}\) measures and the corresponding values of \(M^{(t)}\).
These scores are presented in Table 1. Note Lederle’s nearly constant
level of published advertising.
Table 1 The Basis of Van den Bulte and Lilien’s (2001) Measure of Marketing Effort

<table>
<thead>
<tr>
<th>Time t</th>
<th>Lederle $p^{(t)}$</th>
<th>Other competitors $p^{(t)}$</th>
<th>Lederle $M^{(t)} (\delta = 0.25)$</th>
<th>Other competitors $M^{(t)} (\delta = 0.25)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.00</td>
<td>0.0000</td>
<td>0.0000</td>
<td>0.0000</td>
</tr>
<tr>
<td>2</td>
<td>0.06</td>
<td>0.0000</td>
<td>0.0600</td>
<td>0.0000</td>
</tr>
<tr>
<td>3</td>
<td>0.06</td>
<td>0.0000</td>
<td>0.1050</td>
<td>0.0000</td>
</tr>
<tr>
<td>4</td>
<td>0.06</td>
<td>0.1200</td>
<td>0.1388</td>
<td>0.1200</td>
</tr>
<tr>
<td>5</td>
<td>0.06</td>
<td>0.1600</td>
<td>0.1641</td>
<td>0.1900</td>
</tr>
<tr>
<td>6</td>
<td>0.08</td>
<td>0.0800</td>
<td>0.2030</td>
<td>0.2225</td>
</tr>
<tr>
<td>7</td>
<td>0.09</td>
<td>0.0800</td>
<td>0.2423</td>
<td>0.2469</td>
</tr>
<tr>
<td>8</td>
<td>0.06</td>
<td>0.0800</td>
<td>0.2417</td>
<td>0.2652</td>
</tr>
<tr>
<td>9</td>
<td>0.06</td>
<td>0.0200</td>
<td>0.2413</td>
<td>0.2189</td>
</tr>
<tr>
<td>10</td>
<td>0.06</td>
<td>0.0200</td>
<td>0.2410</td>
<td>0.1842</td>
</tr>
<tr>
<td>11</td>
<td>0.06</td>
<td>0.0800</td>
<td>0.2407</td>
<td>0.2181</td>
</tr>
<tr>
<td>12</td>
<td>0.06</td>
<td>0.0900</td>
<td>0.2405</td>
<td>0.2536</td>
</tr>
<tr>
<td>13</td>
<td>0.06</td>
<td>0.1900</td>
<td>0.2404</td>
<td>0.3802</td>
</tr>
<tr>
<td>14</td>
<td>0.06</td>
<td>0.3600</td>
<td>0.2403</td>
<td>0.6451</td>
</tr>
<tr>
<td>15</td>
<td>0.06</td>
<td>0.2800</td>
<td>0.2402</td>
<td>0.7639</td>
</tr>
<tr>
<td>16</td>
<td>0.06</td>
<td>0.2925</td>
<td>0.2402</td>
<td>0.8654</td>
</tr>
<tr>
<td>17</td>
<td>0.06</td>
<td>0.3050</td>
<td>0.2401</td>
<td>0.9540</td>
</tr>
</tbody>
</table>

Van den Bulte and Lilien (2001) estimate the parameter $\delta$ with a grid search for the $\delta$ value that produced the highest model likelihood “in a model that did not feature social network exposure variables” (p. 1424), but that did include variables pertaining to the individual characteristics of those physicians who were at risk of adopting (their professional age, whether they held a chief or honorary position, the number of journals they read, and their scientific orientation) and an indicator variable for summer months. Estimating the models with all of these individual-level variables, the summer indicator variable, and a social contagion variable (several contagion measures were entertained in separate models), they found significant effects of social contagion. Estimating the models with all of these individual level characteristics, the summer indicator, a social contagion variable, and the two measures of marketing effort (1), one for Lederle and the other for all other competing firms combined, they found no significant effects of social contagion, a significant effect of the Lederle’s marketing effort, and no significant effect of the combined marketing effort of other competitors. These findings are reproduced in Table 2 with the data provided to me.
Van den Bulte and Lilien's findings are based on a discrete time event history model of the form

\[ P(y_i^{(t)} = 1 \mid y_i^{(t-1)} = 0) = \left[ 1 + \exp \left( \beta' x_i^{(t-1)} + \alpha \sum_j w_{ij} y_j^{(t-1)} \right) \right]^{-1}, \quad (2) \]

where \( y_i^{(t-1)} = 0 \) is a physician \( i \) who is at risk of adopting at time \( t - 1 \), \( P(y_i^{(t)} = 1 \mid y_i^{(t-1)} = 0) \) is the hazard rate for the physician's adoption of tetracycline, \( \sum_j w_{ij} y_j^{(t-1)} \) is a social contagion measure, and \( x_i^{(t-1)} \) is an array of control variables. The analysis pools 125 physicians in the four cities studied by Coleman et al. (1966). The units of analysis are 947 physician-months involving all instances of physicians at risk of adopting tetracycline in a particular month.

The Medical Innovation data have served as a useful platform for exploring alternative models of social diffusion. It would be unfortunate if Van den Bulte and Lilien's (2001) findings misleadingly put an end to investigations of this data set. From a nearly constant monthly level of advertising pages for Lederle, Van den Bulte and Lilien constructed a variable for Lederle that rises monotonically over time to an asymptote in a fashion that mimics the overtime curve of cumulative adoptions in the pooled populations of physicians. Their formalization of Lederle's marketing effort generates a variable that is
a close approximation to the partial sum of a geometric series, that is,

\[ M^{(t)} = p + (1 - \delta)p + (1 - \delta)^2p + \cdots + (1 - \delta)^{t-2}p + (1 - \delta)^{t-1}p = p\left[1 + (1 - \delta) + (1 - \delta)^2 + \cdots + (1 - \delta)^{t-2} + (1 - \delta)^{t-1}\right] = p\left(\frac{1 - (1 - \delta)^{t-1}}{\delta}\right), \]

(3)

for a constant \( p^{(t)} = p, \ t = 1, 2, \ldots \). With their estimate of \( \delta = 0.25 \), arbitrarily selected values of \( 0 < p < 1 \), for example, \( p = \{0.001, 0.01, 0.03, 0.06, 0.12\} \), suffice to eliminate contagion effects based on (3). Reducing the amount of monthly advertising from six pages to three pages or to one page or to one-tenth of a page per month also eliminates the detection of a contact network contagion effect.

Thus, it is not evident that a contextual effect of advertising exists and, if it does, it is not evident that it accounts for the observed network contagion effect. Below I describe a parsimonious test of these effects. Contextual effects may be based on city-level conditions that differ across the four cities or on time-level conditions that differ across the months in which the physicians’ adoptions were monitored. City–time contextual effects will generate variation in the intercepts of a suitably nested random intercept model. In the absence of such variation, city–time contextual effects may be dismissed.

2. A RANDOM INTERCEPT EVENT HISTORY MODEL

Physicians’ time \( t \) responses are nested within cities and time periods. A credible approach to these data is to locate those conditions that are common to physicians in the city–time intercepts for this nesting. These common conditions include Van den Bulte and Lilien’s marketing effort measures. From (2), at each time \( t \), we have

\[ P(y_i^{(t)} = 1 | y_i^{(t-1)} = 0) = \left[1 + \exp\left(\beta_0 + \beta_1 M_1^{(t-1)} + \beta_2 M_2^{(t-1)} + \sum_k \beta_k x_{ik} + \alpha \sum_j w_{ij} y_j^{(t-1)}\right)\right]^{-1}, \]

(4)

where \( M_1^{(t)} \) is the marketing effort measure for Lederle and \( M_2^{(t)} \) is the marketing effort measure for the other competitors at time \( t \). The values of these marketing effort measures vary across time periods and are constants within time periods. Since both measures are
constants at time $t - 1$, we have

$$P(y_i^{(t)} = 1 \mid y_i^{(t-1)} = 0) = \left[ 1 + \exp \left( \beta_0^{(t-1)} + \sum_k \beta_k x_{ik} + \alpha \sum_j w_{ij} y_j^{(t-1)} \right) \right]^{-1}. \quad (5)$$

Here, the intercept $\beta_0^{(t-1)}$ represents the net contributions of all contextual conditions that are common to the physicians in the pooled four cities in month $t - 1$. If the fit of this model does not improve upon the fit of the reduced model with a fixed intercept, then there is little reason to suppose that variation of marketing effort is an important factor in the account of physicians’ responses.

Model (5) nests physicians in time. With an elaborated city–time random intercept model, the intercepts also include city-level contextual effects. The intercepts of a city–time random intercept model also include baseline internal contagion effects, that is, $\sum_j w_{ij} y_j^{(t-1)}$ where $w_{ij} = \frac{1}{1 - n_c}$ for all $i$ and $j$ ($i \neq j$), $w_{ii} = 0$ for all $i$, and $n_c$ is the number of physicians in city $c$’s network. This baseline internal contagion value is the proportion of physicians in a particular city who have adopted the drug at time $t - 1$. With such an approach, contact-network contagion is disentangled from baseline-internal contagion. The contact-network contagion test $H_0 : \alpha = 0$ evaluates whether, within a city at a particular time, the variation among the physicians in their weighted averaging of the time $t - 1$ adoptions and nonadoptions of other physicians is associated with the hazard rate of their adoption of the drug at time $t$. The weights that are involved in this weighted averaging are not the weights of the baseline internal contagion model, but weights that are determined by the structure of the contact network in which the physicians are embedded. Thus, in the detection of a contact-network contribution, a city–time nesting simultaneously addresses effects of contextual conditions and baseline internal contagion contributions to adoption. With this elaborated nesting, if the fit of the nested model with random intercepts does not improve upon the fit of the reduced model with a fixed intercept, then there is little reason to suppose that the hazard rate of physicians’ adoption of tetracycline depends on city–time variation of contextual conditions, including marketing efforts and the proportion of previous adopters in a city.

3. FINDINGS

My analysis of Van den Bulte and Lilien’s (2001) measure of marketing effort suggests that their null finding on network contagion
effects is an artifact of the construction of their measure of marketing effort. The monthly level of drug firms’ advertisements in professional journals is a contextual condition that applies to all physicians at risk of adoption in a given month; as such, marketing effort is one condition, among other contextual conditions, that may contribute to variation of hazard rates among physicians in different city–time contexts. The analysis of a suitably nested model with random intercepts provides a parsimonious test of whether or not there is warrant for entertaining hypotheses concerned with such variation. Such warrant must be suspect if the analysis indicates no significant difference of fit between a model that allows the intercepts to vary across contexts and a model that does not allow for such variation. As shown below, the hazard rate of adoption is near zero for at-risk physicians who are not directly exposed to any adopters, regardless of the time and city context.

In the network-contagion component of their model, Van den Bulte and Lilien consider four measures of physicians’ contact-network exposure to the time \( t - 1 \) adoptions and nonadoptions of other physicians. Two of these measures employ weights based on structural cohesion in the physicians’ contact network, and the other two measures employ weights based on structural equivalence in the network (see Van den Bulte and Lilien, 2001, on the detailed construction of these measures). In the event history dataset, the measures are correlated as follows

\[
\begin{align*}
\text{BURTSE1} & \quad 1 \\
\text{BURTCOH} & \quad 0.725 \quad 1 \\
\text{TOTSP} & \quad 0.822 \quad 0.704 \quad 1 \\
\text{TOTCOH} & \quad 0.859 \quad 0.687 \quad 0.739 \quad 1 \\
\end{align*}
\]

where BURTSE1, BURTCOH, TOTSP, and TOTCOH are the acronyms for the four contact network contagion variables in Van den Bulte and Lilien’s data set. Here, I take each measure as providing a credible approach to network contagion and employ their mean values, denoted as CNET, in the analysis. Cronbach’s \( \alpha \) for this measure is 0.922.

Table 3 presents the findings obtained for the event history city–time random intercept model

\[
\ln \{ P(y_i^{(t)} = 1 \mid y_i^{(t-1)} = 0) \} = \beta_0 + \beta_1 x_{si} + \cdots + \beta_6 cnet_{si} + u_s \tag{6}
\]

for \( s = 1, \ldots, 68 \) city–times, with \( i = 1, \ldots, n_s \) physicians in city–time \( s \). The estimate \( \beta_0 + u_s \) is the predicted intercept for the physicians in city–time \( s \). The summer indicator and marketing effort
### TABLE 3 Estimate of Network-Contagion, Controlling for Physicians’ Personal Characteristics, with Random Intercepts Based on a Nesting of Time Periods Within Cities, in Comparison to the Estimate Obtained with a Fixed Intercept

<table>
<thead>
<tr>
<th></th>
<th>Random intercept†</th>
<th>Fixed intercept</th>
</tr>
</thead>
<tbody>
<tr>
<td>Professional age</td>
<td>−0.132∗</td>
<td>−0.132∗</td>
</tr>
<tr>
<td>Professional age²</td>
<td>−0.098∗∗</td>
<td>−0.098∗∗</td>
</tr>
<tr>
<td>Chief or honorary position</td>
<td>−0.945∗</td>
<td>−0.945∗</td>
</tr>
<tr>
<td>N of journals (log)</td>
<td>0.854***</td>
<td>0.854***</td>
</tr>
<tr>
<td>Scientific orientation</td>
<td>1.030****</td>
<td>1.030****</td>
</tr>
<tr>
<td>CNET††</td>
<td>0.967***</td>
<td>0.967***</td>
</tr>
<tr>
<td>Intercept</td>
<td>−3.807****</td>
<td>−3.807****</td>
</tr>
<tr>
<td>LL</td>
<td>−308.370</td>
<td>−308.370</td>
</tr>
</tbody>
</table>

†p < .10; ‡p < .05; ***p < .01; ****p < .001 (two-sided).

††LR test vs. logistic regression: chi2(2) = 4.2E−12, Prob > chi2 = 1.0000.

CNET is measured as the mean of the four network contagion measures employed in Van den Bulte and Lilien’s analysis, i.e., TOTCOH, BURTCOH, TOTSP, and BURTSE1 in Table 2, for which Cronbach’s α is 0.922.

*Note:* These estimates are based on Stata 10’s xtmelogit procedure.

Scores have been dropped as variables, since both are contextual variables with scores that are shared by all at-risk physicians located in a particular city–time setting. The contributions of these constants are captured by the random intercepts. The included variables are the remaining individual-level variables in Table 2 (professional age, professional age squared, chief or honorary position, logged number of journals, and scientific orientation) and CNET (the mean of the four contact network contagion measures).

The network-contagion variable has a significant effect. The fit of the model, LL = −308.37, does not differ from the fit of the reduced null model with a fixed intercept. Indeed, the estimated random effects $u_s$ are trivial with values that range from $−2.30E−16$ to $4.00E−16$. With the estimated $\beta_0 + u_s \approx −3.807$ for all $u_s$ ($s = 1, \ldots, 68$), the baseline hazard rate of adoption is near zero regardless of physicians’ city–time settings. Hence, we may constrain the intercepts with no loss of fit and dismiss the hypothesis that there are contextual differences among the city–time settings that significantly raise or lower the hazard rates for the physicians within these settings. Given trivial variation of the intercepts, the estimates...
obtained for the reduced fixed effects logistic model is
\[
\ln\{P(y_i(t) = 1 | y_i(t−1) = 0)\} = \beta_0 + \beta_1age_i + \cdots + \beta_6cnet_i,
\] (7)
which assumes an intercept that is constant across cities and times, are identical to those obtained for the nested random intercept model.

The above findings support the null model’s assumption of no significant variation among the city–time intercepts. The findings are inconsistent with the hypothesis that mass marketing efforts (based on advertising in professional journals) had a monotonically increasing impact over time on the hazard rate of physicians’ adoption of tetracycline. The findings are consistent with the existence of contact-network contagion that is net of baseline internal contagion and those individual-level variables included as controls.

4. CONCLUSION
My findings support the original conclusion of Coleman et al. (1966) that physicians’ networks of interpersonal contact with other physicians contributed to physicians’ adoption of tetracycline. Moreover, my findings strengthen Coleman et al.’s conclusion in showing that a contact network effect is maintained in a model that allows a potential baseline internal contagion contribution to adoption, that is, the proportion of physicians in a city who have adopted the drug at time \(t−1\).

Mainly as a matter of convenience, I have employed a summative measure of the contact network role in adoption—CNET—based on the four measures employed in Van den Bulte and Lilien’s (2001) analysis. Two of these measures emphasize features of structural cohesion and two emphasize features of structural equivalence in the contact networks of the physicians. Previous analyses have been addressed to the relative merits of a structural cohesion versus structural equivalence approach to specifying network effects on social diffusion. However, since each of these two approaches draws on different but correlated structural features of contact networks, it may be useful in the future to treat them as multiple measures that in combination provide a more reliable discrimination of the contact network environments in which actors are situated. I do not discount the theoretical importance of pursuing a more exact specification of how contact networks contribute to diffusion. I suggest only that it is equally important to more firmly and impartially establish that there is a contact network contribution prior to advancing assertions that some structural features of the contact network are more important than others.
ACKNOWLEDGMENTS

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REFERENCES


