Online Technical Appendix

accompanying

“Opinion Leadership and Social Contagion in New Product Diffusion”

This Appendix reports on various checks documenting that our results are quite robust to variations in model specification.

Construction of network covariates

Table A-1 shows the results for some variants of Model 6 in Table 4, the one best supported by the data. The first column in Table A-1 just repeats the information of Model 6 in Table 4. The second column shows the results for the same model specification, but with Indegree, Outdegree and Contagion based only on referral ties. The third column shows the results for a model using only discussion ties. The fourth column is again based on the total network, but uses normalized social network weights w_{ij} such that $\Sigma_j w_{ij}$ equals unity (or zero for physicians nominating no peers at all), implying that physicians react to the proportion rather than the number of their peers who have adopted or are prescribing. The coefficients of these three alternative models are quite similar to those of the original model, and none fits better than the original. The one slight deviation worth noting is that, in the referral-only model, the coefficient of the interaction between self reported leadership and contagion is very similar to the one in the total network but its standard error is slightly bigger, which pushes the significance level slightly above 0.05.

Contemporaneous contagion

Since it is conceivable that contagion occurred within monthly periods, we also specified a model allowing for such simultaneous contagion. To this end, we used an instrumental variable approach which protects one's estimates from endogeneity bias (e.g., Anselin 1988; Land and Deane 1992; Manski 1993). We constructed the Volume Contagion variable as $\Sigma_j w_{ij} q_{jt}$ and regressed it on an intercept, dummies for LA and NYC, Volume Contagion at t-1, t-2 and t-3, and the network lagged detailing variable, i.e., the detailing to the nominees of the focal

physician at time t, t-1 and t-2 (Σ_j w_{ij} D_{jt-k}, for k = 0, 1 and 2). We then took the predicted values of this first-step regression (R^2 = 96%; all coefficients significant at $p < .01$), and used them as the instrumented values for contemporaneous Volume Contagion in the hazard model. As shown in column 5 of Table A-1, imposing simultaneous contagion while avoiding endogeneity bias leads to a slightly worse fit and does not change any of the coefficients or substantive conclusions.

Controlling for out-of-town contacts

By restricting the relevant networks to physicians practicing in specific zip code areas, our contagion variables do not encompass each and every colleague that the survey respondents nominated. Specifically, of all the people nominated by the survey respondents, we excluded 40 nominees in SF, 63 in LA and 80 in NYC. The excluded contacts received only one nomination on average, with 4 being the maximum, and accounted for 36% of all nominations. To the extent that our network definition is overly narrow, our contagion variables do not account for all the social influence experienced by the physicians whose adoptions we model. Since this may but need not affect our results, we checked that our results are robust to distinguishing between in-town and out-of-town contacts. Splitting the Outdegree into these two components did not improve model fit (Δ -2LL = 0.04) or affect the coefficients of substantive interest. Approximating Volume Contagion from out-of-town contacts by multiplying the number of out-of-town contacts by time (which is reasonable since Figure 4 shows that the in-town Volume Contagion variable increases linearly over time), and adding that new covariate to Model 6 did not significantly improve fit (Δ -2LL = 0.38) or affect the results either. Finally, allowing the effect of approximated out-of-town volume contagion to vary as a function of Indegree and Self-reported Leadership by adding the two relevant interaction terms did not improve model fit (Δ -

2LL = 2.54) or affect the results either. In short, our findings are robust to distinguishing between within-town and out-of-town contacts.

Excluding the flexible baseline hazard

Table A-2 shows the results for some additional variants of Model 6 in Table 4, the one best supported by the data. The first column in Table A-2 again shows the estimates of the latter model. The second column in Table A-2 pertains to the original model, but without time dummies. That model has a lower fit, but the loss in fit is small compared to the gain of 16 degrees of freedom (Δ -2LL = 10.40, $p > 0.05$). Unlike the original, the model without flexible baseline hazard exhibits a significant contagion effect for the average physician. Otherwise, the results are again very robust.

Unobserved heterogeneity and serial correlation

As is well known, unobserved heterogeneity induces spurious negative duration dependence in hazard models. This implies that it may create a downward bias in the contagion effect. Obviously, adding physician-specific fixed effects leads to biased estimates in a logit or probit hazard model of adoption or of any other non-repeated event (e.g., Chamberlain 1980). We therefore extended our model into a semi-parametric specification, featuring a flexible baseline hazard with monthly dummies and normally distributed random effects on the intercept.

We specified a hierarchical Bayes hazard model with a probit link function, and found that the convergence of the parameters within the Markov chain Monte Carlo (MCMC) routine was extremely poor, suggesting the model is overparameterized. When estimating the model without time dummies, we found that the estimated variance of the heterogeneity distribution tended to be determined entirely by the prior distribution of that variance, which indicates that there is no information in the data. Comparisons using Bayes Factors confirmed that unobserved

heterogeneity is not a concern. Similarly, empirical Bayes hazard models with a logit link function estimated using adaptive Gaussian quadrature in SAS NLMIXED led to variance estimates of 10^{-8}, the boundary value. All these results document the absence of detectable effects of unobserved heterogeneity. Given our rich set of covariates and since allowing for a flexible baseline hazard is known to make one's results robust to misspecification or even omission of the unobserved heterogeneity distribution, these results are not surprising (e.g., Butler, Baldwin and Johnson 2001; Dolton and van der Klaauw 1995 and 1999; Meyer 1990; Trussell and Richards 1985). In short, there is no evidence to reject the null hypothesis that our results are unaffected by unobserved heterogeneity.

We also checked for the presence of serial correlation within physicians. Extending Model 6 with an AR(1) structure, fixing the detailing carry-over rate to 0.44, and estimating the resulting model using the Generalized Estimating Equations method (GEE), led to an estimated AR(1) coefficient of 0.0015. Obviously, with such a small AR(1) value, the other coefficients and standard errors barely changed and all substantive conclusions were corroborated. Using the same procedure on the model without monthly dummies led to a similarly low serial correlation coefficient (-0.0004). All these results indicate the absence of serial correlation within physicians, which is consistent with the absence of unobserved heterogeneity.

Controls for possible time-varying endogeneity in detailing

It is conceivable that detailing decisions might have been revised after the launch of the product, making the time-invariant variables Past Drug 1 and Past Drug 2 ineffective controls for endogeneity in detailing and for omitted marketing effort variables. We therefore performed a robustness check using the 3-month moving total of the prescription volume for Drugs 1 and 2 (i.e., the sum of the volumes at t-1 through t-3) instead of their pre-launch total. As reported in

column 3 of Table A-2, this model fits slightly worse while all coefficients and statistical inferences remain unchanged.

Volume contagion after controlling for adoption and use contagion

As Table A-3 shows, extending models 3 and 6 in Table 4 with adoption contagion and use contagion do not change the main result about contagion being moderated by the influencers' prescription volume. Only volume contagion is significant in the main effects model ($p < .05$) and only the interaction between self-reported leadership and volume contagion is significant in the model with interactions ($p < .05$).

Fleshing out the volume contagion effect

That contagion is based on how much of the new drug one's network neighbors prescribe, rather than simply on whether they have adopted it or whether they wrote any prescription for it recently, could be driven by several processes. One possibility is that the volume matters because it is associated with the level of experience with the new drug, and experience in turn makes a colleague a more credible source of information and influence. However, there are alternative explanations as well.

One such alternative is that higher influence is associated with higher volume because the latter conveys enthusiasm about and commitment to the new drug. If this were the case, then one would expect that it is not the *number* of prescriptions written for the new drug but the *share* of the new drug in the source's overall category-level prescriptions that matters. To assess this alternative, we define $SOP_{jt\text{-}1}$ as the focal drug's share of prescriptions by network neighbor j at t-1, and compute it as the number of j's prescriptions for the focal drug at time t-1 divided by the number of j's prescriptions for all three drugs in the category at time t-1. We then define the *SOP*-based contagion variable as $\Sigma_j \, w_{ij} \, SOP_{jt\text{-}1}$. (Note, there is no point in multiplying *SOP* by

either $y_{jt\text{-}1}$ or $s_{jt\text{-}1}$ since $SOP_{jt\text{-}1} > 0$ requires $y_{jt\text{-}1} = 1$ and $s_{jt\text{-}1} = 1$.) Replacing the volume contagion variable with this SOP contagion variable in Model 6 in Table 4, and re-estimating the model leads to very similar coefficients (see Model 1 in Table A-4), including a significant negative interaction with self-reported opinion leadership, but a worse model fit. The difference in deviance (Δ -2LL = Δ BIC = 2.62) is large enough to favor the original volume contagion model (Raftery 1995).

Another possibility is that high prescribers of the new drug tend to be opinion leaders who not only have more network ties, but—being recognized experts—also exert more influence within each of these ties. If volume matters because it confers not experience with the new drug but expert status in general, then one would expect that it is not the *number* of prescriptions written for the new drug but the sociometric leadership of the prescribing peer that matters. To assess this alternative, we define indegree-weighted versions of the adoption and use contagion variables: $\Sigma_j \, w_{ij} \, K_j \, y_{jt\text{-}1}$.and $\Sigma_j \, w_{ij} \, K_j \, s_{jt\text{-}1}$, resp., where K is the *Indegree*. (Note, we do not create such variables for self-reported leadership, since the latter is measured only for survey respondents). Replacing the volume contagion variables with these sociometric leadership-weighted contagion variables leads to much worse model fit (Δ -2LL = Δ BIC = 8.54 and 7.58, resp.; see Models 2 and 3 in Table A-4) with the difference in deviance being large enough to markedly favor the volume contagion model (Raftery 1995).

Yet another possibility is that what matters is not experience with the new drug specifically, but more *general category-level experience*. To assess this alternative, we define two variants of general experience -weighted adoption and use contagion variables. In the first, we use the number of prescriptions written for the other two drugs in the category during the twelve months prior to the launch of the focal drug, and call the resulting variables "prelaunch weighted"

contagion (Models 4 and 5 in Table A-4). In the second variant, we use the number of prescriptions written in the previous month for all three drugs in the category as the weight, and call the resulting variables "category weighted" contagion (Models 6 and 7 in Table A-4). Replacing the volume contagion variables with these general experience-weighted contagion variables leads to much worse model fit (Δ -2LL = Δ BIC equals 6.80 or more) with the difference in deviance being large enough to markedly favor the volume contagion model in all four cases (Raftery 1995).

It is also conceivable that the volume contagion effect actually stems from "*detailing leakage*," where the amplification of the influence within ties stems from more detailing efforts being targeted towards heavy prescribers. We therefore also estimated models with detailing-weighted adoption and use contagion variables as Σ_j w_{ij} D_{jt-1} y_{jt-1} and Σ_j w_{ij} D_{jt-1} s_{jt-1}, respectively, where D_{jt-1} is the amount of detailing physician j received in the prior month. As columns 8 and 9 in Table A-4 report, these alternative models fit worse than the volume contagion model (Δ -2LL = Δ BIC = 5.28 and 3.82, respectively) and the detailing-weighted variables have no significant main effect or interaction effect with opinion leadership ($p > .10$). Hence, the original interpretation of genuine volume contagion seems best supported by the data.[1]

Finally, we also entertain the possibility that part of the volume contagion process operates through a *back-and-forth flow of patients* between physicians, where (i) i refers patients to j, (ii) j treats these patients and prescribes the new drug to some of those patients, (iii) some of those patients flow back to i, and (iv) i adopts so as not to change the regimen of those patients. While this contagion process through patient referral is conceivable, step (iii) from this sequence of

[1] We also assessed the possibility that not only the volume effect but the entire contagion effect is an artifact due to detailing leakage. Using the variable Σ_j w_{ij} D_{jt-1} instead of the volume contagion variable Σ_j w_{ij} q_{jt-1} leads to a model that fits markedly worse (Δ -2LL = Δ BIC = 6.22) and where detailing leakage has no significant main effect or interaction effect with self-reported leadership ($p > .10$).

events is quite unlikely in our data where both the discussion and referral ties in our data are extremely asymmetric. One implication, however, is testable, since the process implies that the practice size of *j* would influence his or her influence through referral ties. Because of the nature of the treatment, the usage rate does not vary within patients being treated, and a physician's category-level prescription volume is likely to be a good proxy for the number of patients treated. We therefore control for practice size in terms of category-level prescription. With $CQ_{jt\text{-}1}$ being the total category-level amount of prescriptions (for all three drugs) written by physician *j* in period *t*-1, and the network weight w_{ij} now being limited to referral ties only, we constructed yet other variants of the adoption and use contagion variables as (a) $\Sigma_j w_{ij} CQ_{jt\text{-}1} y_{jt\text{-}1}$ and (b) $\Sigma_j w_{ij} CQ_{jt\text{-}1} s_{jt\text{-}1}$. As columns 10 and 11 in Table A-4 report, these alternative models fit markedly worse than the volume contagion model (Δ -2LL = Δ BIC = 8.11 and 7.96, respectively) and the detailing-weighted variables have no significant main effect or interaction effect with opinion leadership ($p > .10$). Hence, the original interpretation of genuine volume contagion seems best supported by the data.

In short, the volume effect is most likely to due to *credibility based on experience* with the focal drug, rather than enthusiasm about the focal drug, expert status, category-level experience, amplification through detailing leakage, or back-and-forth flow from patients.

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Table A-1: Robustness Checks for Network Weights and Simultaneous Contagion

	Total Netw. (1)	Referral Netw. (2)	Discuss. Netw. (3)	Total Netw. Normalized (4)	Total Netw. Simultaneity (5)
Intercept	-3.88**	-3.70**	-3.88**	-3.72**	-3.94**
	(0.74)	(0.70)	(0.74)	(0.73)	(0.75)
Indegree	0.30*	0.51	0.52*	0.32*	0.29*
	(0.15)	(0.34)	(0.25)	(0.15)	(0.15)
Outdegree	0.08	0.12	0.10	0.10	0.08
	(0.06)	(0.12)	(0.08)	(0.06)	(0.06)
Self-rep. Leadership	0.42*	0.36*	0.44**	0.39*	0.43*
	(0.18)	(0.18)	(0.19)	(0.18)	(0.18)
Contagion - Volume	0.01	0.01	0.01	0.04	0.01
	(0.007)	(0.02)	(0.01)	(0.03)	(0.006)
Detailing Stock	0.41**	0.38**	0.41**	0.41**	0.40**
	(0.14)	(0.14)	(0.13)	(0.14)	(0.14)
Carry Over Effect	0.44*	0.47*	0.42*	0.42*	0.44*
	(0.20)	(0.24)	(0.19)	(0.21)	(0.21)
Indegree × Contagion	0.001	-0.02	0.002	-0.003	0.002
	(0.005)	(0.03)	(0.01)	(0.03)	(0.004)
Indegree × Detailing Stock	-0.05	-0.02	-0.11	-0.05	-0.05
	(0.04)	(0.09)	(0.07)	(0.04)	(0.04)
Self-rep. Leadership × Contagion	-0.01*	-0.01	-0.03**	-0.05*	-0.01*
	(0.005)	(0.008)	(0.01)	(0.02)	(0.005)
Self-rep. Leadership × Detailing Stock	-0.05	-0.06	-0.03	-0.05	-0.05
	(0.07)	(0.07)	(0.08)	(0.07)	(0.07)
LA Dummy	0.09	-0.01	0.09	0.02	0.11
	(0.42)	(0.41)	(0.42)	(0.42)	(0.45)
NYC Dummy	-0.27	-0.33	-0.31	-0.23	-0.25
	(0.43)	(0.42)	(0.43)	(0.44)	(0.44)
Solo Practice	0.01	-0.01	-0.01	-0.11	0.04
	(0.35)	(0.35)	(0.35)	(0.36)	(0.35)
University / Teaching Hospital	0.69	0.64	0.54	0.63	0.72
	(0.41)	(0.42)	(0.40)	(0.41)	(0.41)
Primary Care	-0.57	-0.48	-0.65	-0.48	-0.54
	(0.77)	(0.77)	(0.78)	(0.76)	(0.77)
Early Referral	-0.77	-0.86	-0.52	-0.73	-0.78
	(0.44)	(0.47)	(0.42)	(0.44)	(0.44)
Patients Managed	0.001	0.001	0.001	-0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.002	0.004	0.002	0.003	0.002
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01**	0.01**	0.01**	0.01**	0.01**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
LL	-225.48	-227.59	-225.99	-226.02	-225.76

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All Models 1-5 include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-2: Robustness Checks for Baseline Hazard and Time-varying Endogeneity

Variables	Total Netw. (1)	Total Netw. No Time Dummies (2)	Total Netw. Past Drugs as Moving Total (3)
Intercept	-3.88**	-4.62**	-3.96**
	(0.78)	(0.45)	(0.75)
Indegree	0.30*	0.22	0.31*
	(0.15)	(0.12)	(0.14)
Outdegree	0.08	0.06	0.07
	(0.06)	(0.05)	(0.05)
Self-rep. Leadership	0.42*	0.39*	0.44*
	(0.18)	(0.18)	(0.19)
Contagion	0.01	0.01*	0.01
	(0.007)	(0.007)	(0.007)
Detailing Stock	0.41**	0.37**	0.42**
	(0.14)	(0.12)	(0.13)
Carry Over Effect	0.44*	0.53**	0.48*
	(0.20)	(0.18)	(0.18)
Indegree × Contagion	0.001	0.003	0.001
	(0.005)	(0.005)	(0.005)
Indegree × Detailing Stock	-0.05	-0.03	-0.05
	(0.04)	(0.03)	(0.04)
Self-rep. Leadership × Contagion	-0.01*	-0.01**	-0.01*
	(0.005)	(0.005)	(0.005)
Self-rep. Leadership × Detailing Stock	-0.05	-0.04	-0.03
	(0.07)	(0.06)	(0.07)
LA Dummy	0.09	0.18	0.07
	(0.42)	(0.43)	(0.44)
NYC Dummy	-0.27	-0.15	-0.19
	(0.43)	(0.43)	(0.42)
Solo Practice	0.01	0.01	-0.07
	(0.35)	(0.34)	(0.35)
University / Teaching Hospital	0.69	0.74	0.67
	(0.41)	(0.41)	(0.41)
Primary Care	-0.57	-0.52	-0.56
	(0.77)	(0.77)	(0.77)
Early Referral	-0.77	-0.75	-0.72
	(0.44)	(0.44)	(0.45)
Patients Managed	0.001	0.001	0.001
	(0.001)	(0.001)	(0.001)
Past Drug 1	0.002	0.001	-0.001
	(0.004)	(0.004)	(0.013)
Past Drug 2	0.01**	0.01*	0.05**
	(0.004)	(0.004)	(0.01)
LL	-225.48	-230.68	-226.75

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. Models 1 and 3 include 16 monthly time dummies and so have a flexible baseline hazard rate. Model 2 does not.

Table A-3: Contagion through Adoption, Use and Volume

Variables	Main Effects	Interaction Effects
Intercept	-3.65**	-3.55**
	(0.71)	(0.76)
Indegree	0.14*	0.31*
	(0.07)	(0.15)
Outdegree	0.13	0.13
	(0.07)	(0.07)
Self-rep. Leadership	0.17	0.31
	(0.15)	(0.20)
Contagion, Adoption	-0.19	-0.12
	(0.28)	(0.40)
Contagion, Use	-0.02	-0.08
	(0.31)	(0.43)
Contagion, Volume	0.02*	0.02
	(0.009)	(0.009)
Detailing Stock	0.39**	0.43**
	(0.13)	(0.14)
Carry Over Effect	0.46*	0.45*
	(0.22)	(0.19)
Indegree × Contagion, Adoption		0.19
		(0.35)
Indegree × Contagion, Use		-0.21
		(0.36)
Indegree × Contagion, Volume		0.003
		(0.007)
Indegree × Detailing Stock		-0.05
		(0.04)
Self-rep. Leadership × Contagion, Adoption		-0.04
		(0.29)
Self-rep. Leadership × Contagion, Use		0.14
		(0.33)
Self-rep. Leadership × Contagion, Volume		-0.02*
		(0.008)
Self-rep. Leadership × Detailing Stock		-0.03
		(0.07)
LA Dummy	0.18	-0.09
	(0.41)	(0.36)
NYC Dummy	-0.36	-0.37
	(0.43)	(0.44)
Solo Practice	0.04	-0.09
	(0.35)	(0.36)
University / Teaching Hospital	0.72	0.73
	(0.41)	(0.42)
Primary Care	-0.61	-0.61
	(0.76)	(0.78)
Early Referral	-0.76	-0.89
	(0.43)	(0.46)
Patients Managed	0.0004	0.0002
	(0.001)	(0.001)
Past Drug 1	0.004	0.003
	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*
	(0.004)	(0.005)
LL	-227.93	-223.79

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. Both models include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-4: Alternatives to the Volume Contagion Model

Variable	Share of Prescriptions (1)	Indegree Weighted Adoption (2)	Indegree Weighted Use (3)	Prelaunch Categ. Vol. Weighted Adoption (4)	Prelaunch Categ. Vol. Weighted Use (5)
Intercept	-3.89**	-3.25**	-3.32**	-3.31**	-3.36**
	(0.73)	(0.66)	(0.66)	(0.68)	(0.69)
Indegree	0.28	0.35*	0.37*	0.31*	0.31*
	(0.15)	(0.16)	(0.16)	(0.14)	(0.14)
Outdegree	0.07	0.10	0.09	0.11	0.09
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Self-rep. Leadership	0.44*	0.18	0.16	0.37*	0.36
	(0.19)	(0.19)	(0.19)	(0.19)	(0.19)
Contagion	0.52	-0.00	-0.00	0.08	0.56
	(0.29)	(0.001)	(0.001)	(2.19)	(2.29)
Detailing Stock	0.39**	0.38**	0.37**	0.37**	0.37**
	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)
Carry Over Effect	0.45*	0.41	0.42*	0.45*	0.45*
	(0.21)	(0.21)	(0.21)	(0.20)	(0.20)
Indegree × Contagion	0.11	-0.0001	-0.0002	0.59	0.53
	(0.18)	(0.0005)	(0.0005)	(1.08)	(1.09)
Indegree × Detailing Stock	-0.04	-0.06	-0.06	-0.06	-0.02
	(0.04)	(0.05)	(0.05)	(0.04)	(0.07)
Self-rep. Leadership × Contagion	-0.52*	0.0003	0.0004	-2.79	-2.67
	(0.24)	(0.0007)	(0.0007)	(1.84)	(1.87)
Self-rep. Leadership × Detailing Stock	-0.05	-0.01	-0.01	-0.02	-0.02
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
LA Dummy	0.03	-0.15	-0.12	-0.22	-0.19
	(0.40)	(0.38)	(0.38)	(0.41)	(0.42)
NYC Dummy	-0.38	-0.49	-0.47	-0.58	-0.55
	(0.41)	(0.39)	(0.39)	(0.42)	(0.42)
Solo Practice	0.11	0.07	0.06	-0.02	-0.01
	(0.35)	(0.35)	(0.35)	(0.35)	(0.35)
University / Teaching Hospital	0.65	0.64	0.63	0.55	0.57
	(0.41)	(0.40)	(0.40)	(0.41)	(0.41)
Primary Care	-0.41	-0.55	-0.57	-0.64	-0.63
	(0.77)	(0.77)	(0.76)	(0.77)	(0.77)
Early Referral	-0.80	-0.69	-0.67	-0.71	-0.71
	(0.45)	(0.44)	(0.43)	(0.43)	(0.43)
Patients Managed	0.0008	0.0003	0.0002	0.0001	0.0001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.003	0.004	0.004	0.003	0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*	0.01*	0.01**	0.01*
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
LL	-226.79	-229.75	-229.27	-228.88	-228.91

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All models include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-4 (continued): Alternatives to the Volume Contagion Model

Variables	Current Categ. Vol. Weighted Adoption (6)	Current Categ. Vol Weighted Use (7)	Detailing Weighted Adoption (8)	Detailing Weighted Use (9)	Referral Volume Weighted Adoption (10)	Referral Volume Weighted Use (11)
Intercept	-3.32**	-3.37**	-3.43**	-3.54**	-3.36**	-3.39**
	(0.66)	(0.69)	(0.67)	(0.67)	(0.67)	(0.68)
Indegree	0.31*	0.31*	0.25	0.24	0.32*	0.32*
	(0.15)	(0.14)	(0.14)	(0.14)	(0.14)	(0.14)
Outdegree	0.10	0.09	0.09	0.07	0.09	0.09
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Self-rep. Leadership	0.33	0.33	0.33	0.37*	0.28	0.28
	(0.18)	(0.18)	(0.18)	(0.18)	(0.17)	(0.17)
Contagion	0.00	0.00	0.03	0.06	0.00	0.00
	(0.002)	(0.002)	(0.03)	(0.04)	(0.004)	(0.004)
Detailing Stock	0.37**	0.37**	0.37**	0.36**	0.36**	0.36**
	(0.13)	(0.13)	(0.13)	(0.14)	(0.13)	(0.13)
Carry Over Effect	0.45*	0.45*	0.48*	0.47*	0.44*	0.44*
	(0.20)	(0.21)	(0.21)	(0.21)	(0.21)	(0.21)
Indegree × Contagion	0.0006	0.0005	0.04	0.05	0.001	0.001
	(0.001)	(0.001)	(0.02)	(0.03)	(0.002)	(0.002)
Indegree × Detailing Stock	-0.06	-0.06	-0.05	-0.04	-0.06	-0.06
	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)
Self-rep. Leadership × Contagion	-0.002	-0.002	-0.03	-0.04	-0.002	-0.002
	(0.002)	(0.002)	(0.02)	(0.03)	(0.003)	(0.003)
Self-rep. Leadership × Det. Stock	-0.02	-0.02	-0.02	-0.03	-0.02	-0.02
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
LA Dummy	-0.21	-0.18	-0.16	-0.09	-0.15	-0.13
	(0.41)	(0.42)	(0.39)	(0.39)	(0.39)	(0.40)
NYC Dummy	-0.58	-0.56	-0.56	-0.51	-0.52	-0.51
	(0.42)	(0.42)	(0.42)	(0.42)	(0.41)	(0.41)
Solo Practice	0.003	0.006	0.06	0.07	0.02	0.01
	(0.35)	(0.36)	(0.35)	(0.35)	(0.35)	(0.35)
University / Teaching Hospital	0.58	0.59	0.61	0.61	0.62	0.62
	(0.41)	(0.41)	(0.41)	(0.41)	(0.41)	(0.41)
Primary Care	-0.65	-0.64	-0.68	-0.71	-0.63	-0.61
	(0.77)	(0.77)	(0.77)	(0.77)	(0.77)	(0.77)
Early Referral	-0.71	-0.70	-0.67	-0.66	-0.74	-0.75
	(0.43)	(0.43)	(0.43)	(0.43)	(0.44)	(0.44)
Patients Managed	0.0001	0.0001	0.0004	0.0003	0.0001	-0.0001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.004	0.004	0.004	0.004	0.004	0.004
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*	0.01*	0.01*	0.01*	0.01*
	(0.004)	(0.005)	(0.004)	(0.004)	(0.004)	(0.005)
LL	-229.26	-229.26	-228.12	-227.39	-229.53	-229.46

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All models include 16 monthly time dummies and so have a flexible baseline hazard rate.