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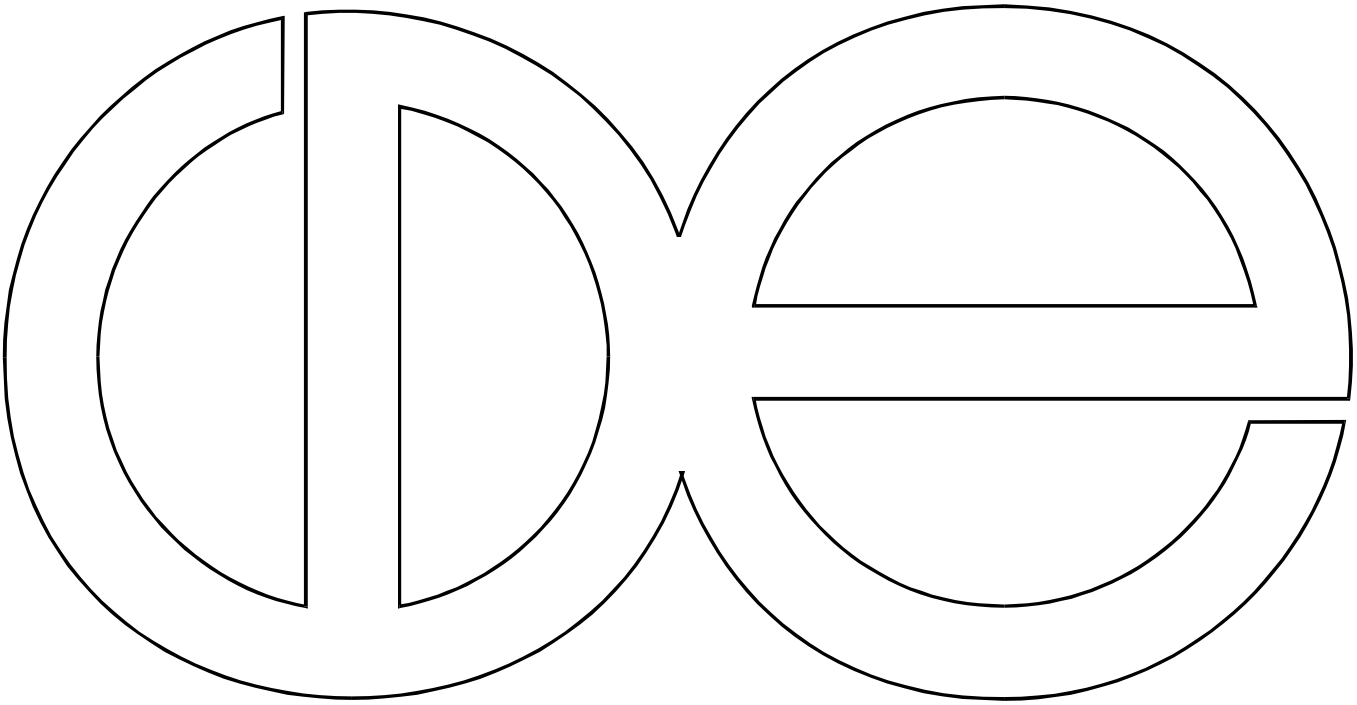

EXPLORING SOCIAL INTERACTION AND DIFFERENTIATION EFFECTS IN LATIN AMERICA'S MORTALITY TRANSITION

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Problem statement

> "The processes in interpersonal relations and networks shape our social experiences and roles. These social interactions are circumscribed by macro-structural conditions, which do not rob individuals of choice but do limit the alternatives available to them." (Peter Blau, 1964:144).

A nagging problem in demography is how to account for the peculiarly uneven paths of contemporary mortality transitions. Classic demographic transition theory held that economic development would increase health preferences and induce people to adopt healthier behaviors. By the mid-1970s demographic transition theory had been discredited for incorporating the functionalist assumptions of modernization theory. Its demise signaled the triumph of methodological individualism over holistic development theories (Kontopoulos 1993). Among demographers, the proximate determinants and related household health production paradigms remain the mainstays of mortality research (DaVanzo and Gertler 1990; Schumann and Mosley 1994). Yet empirical support for these utilitarian theories has been inconsistent (Preston 1985a,1985b; Szreter 1988). Researchers are currently pursuing a variety of strategies to identify sociological transition determinants. In a recent review, Bongaarts and Watkins (1996) argued that increasing social interaction at all levels explains why the latest transitions have begun in unexpected places and proceeded faster than earlier transitions. Their causal argument is that, as heterogenous actors increasingly interact, their thresholds for behavioral change fall (Bongaarts and Watkins 1996:46). Rising educational levels, urbanization, the development of global mass media and communications networks, increasing international travel and foreign investment are some of the structural changes they see reconstituting reference groups and enabling these new kinds of interpersonal interactions. From this perspective, the proliferation of "weak ties" across

previously independent social structures is a cause of behavioral change over and above household and local constraints. These structural factors indirectly affect individual outcomes by increasing or decreasing the potential for new practices and attitudes to diffuse.

In this paper I elaborate an event history model designed to measure the interaction of structural and social effects on one demographically important behavior, child immunization. The model incorporates some recent work on social interactions and a macrosocological theory of social exchange. I then apply the model using data from Colombia and Paraguay. The data come from Demographic and Health Surveys carried out in both countries in 1990, and information from recent censuses and ministry of health records.

Conceptualizing mortality transitions

Increased demand on the part of parents for healthier children is presumably the mechanism propelling mortality transitions. Changing tastes brought on by socioeconomic development causes parents to accord higher preference to health, obtain necessary knowledge and skills and adopt more health-producing behaviors. Among these behaviors is the choice to utilize health services offered by states and markets. As these services are extended and improved the marginal costs of health production decrease. Health is just one of a number of production functions in a household, all of which are subject to income and time constraints. Yet household health production often increases in the absence of economic growth, suggesting that the link between economic conditions and child mortality is, in fact, tenuous (Palloni 1981). Examining the World Fertility Surveys, Preston (1985b) found that child mortality hardly varied by urban-rural residence but did vary by ethnicity, even with household wealth controlled. The importance of

cultural variables led Preston to conclude that mortality differentials reflect gaps in health knowledge and the persistence of certain domestic child care practices rather than differences in income or access to health services (Preston 1985b). Similar results were reported in a twelve-country study that compared WFS and Demographic and Health Survey data (Cleland, Bicego and Fegan 1992). Between 1965 and 1985, the study showed, underfive mortality fell 50 percent. Rates of child mortality decline were twice those of infant mortality. Despite economic shocks and prolonged worldwide recession, the declines in the eighties were greater than those in the seventies. Only a modest part of the declines, less than 20 percent, could be attributed to compositional changes in socioeconomic levels. In fact, socioeconomic differentials remained constant or slightly increased over the period (Cleland, Bicego and Fegan 1992). Multilevel research has uncovered evidence that social forces are at work in mortality transitions. The evidence is in the form of significant community-level random effects on child mortality and health behaviors. This clustering generally persists even when socioeconomic and geographical factors are controlled (Sastry 1995; Steele, Diamond and Amin 1995; Pebley, Goldman and Rodriguez 1996; Matthews and Diamond 1997). These and other empirical findings suggest that economic transformation and consequent individuation are not the fundamental mechanisms underlying demographic change. Rather, they support alternative theories, championed by historical and contemporary researchers alike, that view health practices as relatively labile and socially constructed (Szreter 1988; Noack 1988; Hammel 1990; Nathanson 1996). In this view, the characteristically uneven pattern of mortality transitions reflects the differential diffusion of health knowledge, beliefs and practices across time and social space (Strang and Meyer 1993). These diffusion processes are imperfect due to population heterogeneity and the ability of agents to act collectively- factors which formal diffusion models cannot easily incorporate. This

inadequacy has led to a number of new demographic models, which reconceptualize diffusion in network-analytic terms (Entwisle et al., 1996; Montgomery and Casterline 1996; Kohler 1997). Though intuitively appealing, these models assume that sets of influential others can be enumerated and bounded for each individual in the sample. Relational data of this sort are hard to come by. Another approach, which this paper pursues, is to conceptualize diffusion in stochastic, macrostructural terms.

Analytical framework

Social interactions

A social interaction effect occurs whenever the proportion of actors engaging in a specific behavior affects the likelihood any one actor will perform that behavior (Erbring and Young 1979; Brock and Durlauf 2000). A given actor's behaviors are conditional on her perception of how influential others act. In diffusion terms, the presence of social interaction indicates an internal diffusion or contagion process. Interaction and contagion effects are distinct from contextual effects, a mainstay of early sociological theories (Lazarsfeld and Menzel 1969; Blalock 1985), in that they are dynamic and entail behavioral feedback. Actors observe others' outcomes over time and continually reassess the costs and benefits of choosing the behavior (Palloni 1998). Recently, Brock and Durlauf (2000) proposed a series of nonlinear models that generate consistent, unbiased estimates of social interaction effects. In these models the utility an agent i assigns to a given behavioral choice w_i is partly determined by her perceptions of the choices made by other agents j in her neighborhood, $m^e_i(w_{-i})$. Assuming that the agents in a particular neighborhood $n(i)$ equally influence one another's behaviors, the proportion of agents n choosing behavior w measures the social utility S to agent i of choosing w_i. With the further

assumptions that agents are rational and self-consistent and subject to observable constraints Z_i, social utility can be expressed as the expected mean behavioral choice ϖ:

$$S(\omega_i, Z_i, \mu^e{}_i(\omega_{-i})) = \varpi = J_{i,j}\ \omega_{-i}\ \Sigma_{\ j \in Ni}\ E_i\ (\omega_j)$$

where J is a weight assigned to all pairs of members of community *n(i)* such that $J_{i,j} = 0$ if $j \notin n(i)$. This contextual variable is identified, the authors argue, provided the model includes at least one additional, exogenous neighborhood-level variable (Brock and Durlauf 2000:33). This formulation finesses a number of statistical problems that have impeded the modeling of what Erbring and Young (1979) first referred to as endogenous social effects. Following Brock and Durlauf, I compute a social interaction measure consisting of the summed waiting times to full immunization divided by the summed exposure durations of all children older than index child *i.* I weight this measure, assigning full weight to children in cluster *j* and smaller weights to children in the same *municipio k* and *departmento l.* The lower this ratio, the faster children in that area complete their immunizations. Because I consider only the observed behaviors of older children this measure is exogenous in a statistical sense to the index child's behavioral choice.

Structural differentiation

The second component of the model is social structural differentiation. I conceptualize social structure based on the macrosociological theory of Peter Blau (1964, 1977a, 1977b, 1988; Blau and Schwartz 1984). Blau maintained that the *distributions* of attributes of individuals occupying positions in social structures affect the probability actors in different groups will associate, experience social mobility or exchange goods and services (Blau 1977a:4-5). He postulated that

these distributions form macrostructural parameters of three kinds. The first is *heterogeneity*, the extent to which members of a social system are divided into nominal groups (e.g., race, ethnicity, religion). Heterogeneity is high when actors are evenly distributed across many groups. The second parameter is *inequality*, the extent to which members are differentiated in terms of ranked status attributes (e.g., income, educational attainment). Inequality captures the degree to which resources are concentrated in certain strata (Blau 1977b:31). Because social associations are more likely among similar actors in adjacent positions (Blau 1977b:41), both heterogeneity and inequality tend to reduce inter-group relations. The degree to which heterogeneity and inequality dimensions are correlated (*consolidation*) comprises Blau's third structural parameter. If heterogeneity and inequality are consolidated, group and status differences are conflated and inter-group relations are further diminished. The pure effects of Blau's structural parameters, however, are seldom evident. They vary according to size and level of aggregation. On the neighborhood level differentiation favors social integration because the physical propinquity of actors makes relations across nominal and status groups more frequent (Blau 1977b:42). Actors in such localities are relatively sophistocated. They occupy many positions (multiform heterogeneity), so that relationships established on the basis of one similarity necessarily exposes them to a panoply of new influences (Blau 1977a:1; Blau and Schwartz 1984). Structural differentiation at the micro level thus facilitates the transmission of new ideas and practices. At higher levels of aggregation the ameliorating effects of propinquity lessen so that differentiation reduces inter-group relations. In contrast to heterogeneity and inequality, the effects of consolidation are everywhere negative. High consolidation at the macro level begets segregation. The diffusion of new health ideas and practices then would be fastest in social systems where collectivities are highly differentiated and the higher-level structures in which they are embedded

are relatively homogenous and egalitarian. Where contagion is present, a social interaction measure ought to interact with these differentiation measures to affect the probability an individual will perform a specific behavior.

To operationalize Blau's differentiation parameters I use census data to compute a series of inequality, heterogeneity and consolidation measures at both cluster and *municipio* levels. I then categorize these into quartiles, and model the interactions of an indicator variable for the mean behavioral choice with the highest and lowest quartiles at each level of aggregation. This procedure yields a total of 48 interactive models.

Local constraints

While Blau emphasized the constraints that macro-level structural properties place on social interaction he maintained the assumption of rational choice, as do Brock and Durlauf (2000). Parents undoubtedly choose health behaviors given their income, endowments, time and the performance of local health services (Da Vanzo and Gertler 1990), over and above other effects that may be operating. To control for household constraints I include in the model the index child's birth order, gender of the household head, maternal education, spousal occupation and, in Paraguay, language spoken in the home. I also add to each child's record the ministry of health's vaccination coverage estimate for that *municipio* the year that that child reached nine months of age. Without this immunization supply-side control, social interaction and exogenous program effects on immunization behaviors might be confounded. To complete the model I include dummies for annual birth cohorts, which I expect will absorb unmeasured period effects. Descriptive statistics for all these variables are shown in Tables 1 and 2.

Hypotheses

I use this model to test four specific hypotheses that support a diffusion model of immunization behavioral change:

1. The hazard of full immunization for child *i* increases as the mean waiting time to full immunization decreases among older children in and around that collectivity (social interaction effect).

2. The effect of social interactions on the hazard of full immunization is conditional on the degree of differentiation in a collectivity and the social structures in which it is embedded (interaction of differentiation and social interaction effects).

3. The interaction of inequality and heterogeneity with social interaction should increase the hazard at the cluster level but decrease the hazard at the *municipio* level (level effects).

4. The interaction of consolidation measures with social interaction should everywhere decrease the hazard of full immunization (consolidation effect).

Data and methods

Hierarchical datasets

To construct the Colombian dataset I generated 3751 individual records for all surviving children below age 5, subsetted the 1263 children ages 9-68 months at the time of the survey who had immunization cards, and computed exposure and waiting times to full immunization for each. I generated event histories for 1214 Paraguayan cardholding children in similar fashion. The DHS surveys utilize standard multistage cluster sampling procedures (Institute for Resource Development/Westinghouse 1987). Colombia's 1990 DHSII sampling frame was a subsample of the Departamento Nacional de Estadistica y Censo (DANE) national master sample. The latter is based on the 1985 census and had been last updated in late 1989. The sampling frame was

constrained to include the country's three largest cities: Bogota, Medellin and Cali. A total of 713 DHS clusters (segments) were surveyed, consisting of the smallest censal units- census tracts- drawn from 120 randomly selected *municipios*. The Paraguayan DHS sample was drawn from 232 primary sampling units. The units were randomly sampled from lists of enumeration areas used in the country's 1982 census. In each unit an average of 25 households were interviewed. Thus, the Paraguayan DHS clusters were roughly twice as large as the Colombian clusters. The fact that households were systematically selected within neighborhoods makes inferences on social interaction effects tenable.

During visits to Bogota and Asunción I obtained vaccine coverage data from the ministries of health as well as the lists of the particular census tracts and sectors from which the DHS sample cluster were selected. I added the 1982 (Paraguay) and 1985 (Colombia) census identifiers (segment number) to each child record. With these I was able to link the DHS, ministry of health and census data. There were no links to 13 Colombian and 14 Paraguayan DHS clusters; these I discarded from the datasets. The local vaccine supply measure for Colombia is the annual proportion of children under age three in a *municipio* who have received their third dose of oral polio vaccine (OPV3). For Paraguay it is the proportion of children under two who have received their third dose of diphtheria-pertussis-tetanus vaccine (DPT3)[1]. Ministry data were missing for at least one year in 26 Colombian *municipios*, representing 17% of sample children, and in 39/210 Paraguayan *distritos* in 1985. In each case I imputed the value from the corresponding

[1] During this period Paraguay delivered almost all of its polio vaccinations through periodic mass campaigns. The proportions of children receiving DPT3 would thus better measure routine EPI performance.

departmento level. I then recoded the coverage data into quartiles, and added an indicator for *municipios* with missing data.

Social Interaction Measures

I used a series of N x (N-1) matrices to create unique social interaction measures for each child. I operationalize adjacency in a geographic sense by assigning different weights to actors within clusters, *municipios* and *departmentos*. A child *i+1* living in the same cluster *k* as index child *i* is assigned a weight of 1; child *i+2* living in another cluster within the same *municipio k* is assigned a weight of 0.5, and child *i+3* in another *municipio* within the same *departmento l* is assigned a weight of 0.1. All other children's weights are set to zero. This is a spatial effects weighting scheme, which uses hierarchical geographic identifiers to delimit ongoing social processes (Doreian 1980). Tolnay, Deane and Beck (1996) used this approach to represent the potential for lynching in a given county. Next, I compute two N x (N-1) matrices with the immunization duration data. One contains waiting times to immunization or censoring, the other contains months of exposure to the risk of full immunization. I use another N x (N-1) matrix to generate a lag operator for each cell of the matrix to select only information on children older than index child *i*. I then compute the cross products of these matrices and sum the values for each child (row). To compute the actual social interaction measure I divide the sum of lagged, weighted waiting times by the sum of lagged, weighted exposure times, yielding a proportion between 0 and 1. I categorize and reverse code this continuous variable and use the dummies as regressors in the hazard models.

Differentiation measures

Using 5% samples of Colombia's 1985 and Paraguay's 1982 national population censuses I computed the means of a series of cluster- and *municipio*-level contextual variables, including migrational patterns, marital status, occupations, quality of housing and the educational attainment of household heads. I used the first four of these to compute social heterogeneity measures. I used Theil's (1967) entropy index, expressed as: $H_{rel}= (-\Sigma P_i log2P_i)/log2K$, where K equals the number possible categories. The index is maximized when all nominal groups are equally represented. To measure inequality, I used educational attainment levels and a housing quality index to compute Gini indexes for each level of aggregation. I use the following formula (Theil 1972): $G= 1/2 \Sigma\Sigma |(1/k)Pi - (1/k)Pj|$ where Pi and Pj are the weighted housing and educational scores for each of k=5 strata. The housing index, a proxy for income, consisted of a score based on four key housing characteristics: wall construction, floors, location of the kitchen and electricity. To generate Blau's third class of structural parameters, consolidation, I computed simple Pearson correlations for each pair of inequality and heterogeneity measures in each census tract and *municipio/distrito.* I then merged these census indicators with the combined DHS and ministry of health files. The census file, however, failed to match 61 of the 713 Colombian DHS clusters. I concluded that these represented newer settlements that did not exist in 1985 but which DANE had subsequently added to the national master sample. Unable to impute any census-based values for any of these clusters, I dropped them from the dataset.

Hazard models

I use continuous-time proportional hazards models of the following form:

$$\mu_{ijkl}(t)= \mu_{0p}(t)*\exp[X_i\beta_1 + M_{jkl}(t-1)\beta_2 + P_k(t-1)\beta_3 + H_{jk}(t-1)\beta_4 + I_{jk}(t-1)\beta_5 + C_{kl}(t-1)\beta_6 + Q_{jkl}(t-1)\beta_7] + \varepsilon_{ijk}$$

where $\mu_{ijk}(t)$ is the hazard of full immunization for child *i* in cluster *j*, *municipio k*, during the interval (*t, t+1*) and $\mu_{0p}(t)$ is the baseline hazard at time *t* in time interval *p*. A child enters the risk set at age nine months and her exposure to the hazard of full immunization is measured in months. I specify an exponential model, imposing the assumption that the hazard is constant within each *p*. The cutpoints for the segments are determined empirically. The β_1-β_7 terms are vectors of fixed parameters; X_i is a vector of household characteristics; $M_{jkl}(t-1)$ is an indicator coded 1 if the ratio of waiting times over exposure times among older children in cluster j, *municipio k* and *distrito l* is below the sample mean, 0 otherwise; $P_k(t-1)$ is the ministry's annual estimate of program effort for *municipio k* for the year the index child enters the risk set; (I,H,C) are inequality, heterogeneity and consolidation measures computed at cluster and *municipio* levels using census data; and (Q_j,Q_k) are the interactions of these structural variables with $M_k(t-1)$. I interpret a significant interaction as evidence of a diffusion effect.

Results

Table 1 reveals several important differences in the two samples. Compared to their Colombian counterparts, children in the Paraguayan risk set are relatively older, have less educated mothers, and are more likely to live in larger, agrarian households that are located in *municipios* with low immunization program effort. The continuous social interaction measure is lower in Colombia, indicating that waiting times to full immunization are shorter there than in Paraguay.

Table 2 shows that social structures in the two countries also vary. At both levels of aggregation, the housing quality Ginis are comparable but the educational attainment Gini is noticeably higher in Paraguay, indicating greater educational inequality. The three social heterogeneity measures are uniformly higher in Colombia. The consolidation terms reveal more complex structural characteristics. Four of the six cluster-level consolidation measures are comparable in direction and magnitude. In Paraguay, high housing inequality is associated with less variation in household head nativity while the opposite is true in Colombia. A second difference is the relationship between educational inequality and occupational diversity. In Colombia this correlation is positive but in Paraguay it is negative and considerably larger. Overall, housing inequality in Colombia and occupational heterogeneity in Paraguay are the two most consolidated cluster-level parameters. At the *municipio* level the structural patterns are more consistent. Four of five inequality and heterogeneity measures are higher in Colombia as are three of the consolidation measures. In Colombia, the three educational correlations are strongly negative at the macro level. This is a marked departure from the cluster level. In Paraguayan *municipios* there are comparable, strongly negative correlations of occupational heterogeneity with both inequality measures. Overall, the most important *municipio*-level consolidation terms involve educational inequality in Colombia and occupational heterogeneity in Paraguay. I now consider how the various parameters behave in multivariate models.

Additive models

Figures 1 and 2 show the nonparametric hazard rates for each country conditional only on the social interaction quartile indicators. In both cases the hazard rises monotonically with the intensity of the social effect. In Colombia, the hazards are concentrated below nine months' exposure; in Paraguay they range across the duration structure. This pattern is compatible with the smaller social interaction mean value in Colombia.

Tables 3 and 4 show the baseline piecewise logistic models for the two countries. In Model 2 of both tables, I add a single dummy variable indicating a *municipio* with social interaction effects above the sample mean. This term is positive and significant, as predicted. Comparisons of the log-likelihoods of Models 1 and 2 show that adding the indicator improves model fit in both cases. In these proportional hazard models, each coeffecient can be exponentiated to show its relative contribution to the log-odds of full immunization. As the tables show, the relative risk of full immunization is nearly ten times higher for a high-risk Colombian child in a high social interaction *municipio* versus one with little or no social interaction. For a similar Paraguayan child it is 24 times higher.

Figures 3 and 4 show the piecewise hazards by social interaction quartiles, arbitrarily, for a child age 24-35 months living in a household with all risk factors, in a *municipio* with program coverage between 80-90 percent the previous two years. As in the unconstrained models, the hazards are highest where the social interaction effects are strongest. Notably, an interaction effect in the 25^{th} to 75^{th} percentile range substantially increases the hazard in Colombia but not in

Paraguay. The results so far support the first hypothesis: The immunization behaviors of adjacent others influence each child's individual immunization outcome.

How do these social interaction effects compare in magnitude to the other effects? Figures 5 and 6 show the relative effects of risk status and local program effort with no social interaction terms in the models. As one would expect, the hazards are highest for low-risk households in *municipios* with over 90% program coverage in both countries. The hazards of high-risk households in the two countries differ according to program effort. The hazard of a high-risk Colombian child is considerably higher if she lives in a high-coverage *municipio*; this is true in Paraguay as well but the difference is quite small. High-risk Paraguayan children have substantially lower hazards regardless of the level of local program effort. Conversely, in Colombia, children in high-effort *municipios* have the highest hazards regardless of their household risk status. Colombia's routine immunization program essentially offsets the negative risk effects. However, in neither country do program effort or risk status affect the baseline hazards as dramatically as do the social interactions.

Table 5 shows how the macrostructural variables directly affect the hazard models. To simplify exposition, I show only the log-likelihoods of the 48 additive models. The baseline models refer to Model 2 of Table 3 and Model 2 of 4. The "low" structural effects models show how model fit is affected by simultaneously adding the lowest quartile dummies for each set of inequality, heterogeneity and consolidation variables.[2] At the cluster-level, four of the twelve low-

differentiation combinations significantly improve model fit. In Paraguay, these significant combinations include low occupational entropy with both low Gini measures and their respective consolidation terms. In Colombia, only the combinations of the low union status entropy indicator with the low inequality indicators and their respective consolidation terms improve fit. Five of the high-differentiation combinations significantly improve model fit at the cluster level. At the *municipio* level, most of the combinations improve model fit. The heterogeneity measures tend to have the same significant effects with either Gini coefficient. Similarly, there is agreement among the three heterogeneity measures with each Gini. These are reassuring signs that the structural variables are consistently measuring the same constructs. These additive models serve as baselines for the more instructive interactive models.

Interactive models

The essential research question is how the structural variables interact with the social interaction indicator to affect the hazards. To gauge this I procede by inspection, comparing the direction of the interactive effects of the social interaction dummy with low and high differentiation measures. Figures 7 through 12 illustrate my method for a particular cluster-level structural location in Colombia. In Figures 7 and 8, I compare the hazards from additive models involving the nativity entropy and both Ginis. The solid lines in the graphs represent models with no structural effects (corresponding to Model 1 of Table 3). The dashed and dotted lines represent

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[2] The models are nested. To assess goodness-of-fit I interpret twice the difference in negative log-likelihoods as a chi square statistic with three degrees of freedom. The critical values are 6.25 at p=.10 and 7.81 at p=.05. I report both in Table 5.

low and high additive models in which the low and high quartile nativity entropy indicators are combined with the corresponding quartile indicators for each Gini and their respective consolidation terms. They correspond to the additive models described in Table 5. Figure 7 shows that the main effects of low heterogeneity, as measured by household head nativity, low inequality as measured by the housing quality Gini, and low consolidation of the two, considerably reduces the hazard even though, as shown in Table 5, this particular set of variables does not improve model fit. Figure 8 shows the same effect of the structural variables on the hazard, but the effect is more modest when inequality is measured by the educational Gini. Figures 9 and 10 show how the curves shift when the lowest quartile interaction terms for inequality and social interaction are added to each model, while Figures 11 and 12 show the effects of adding the high quartile interaction terms. When differentiation is low the interactions shift the hazards upward in both models. When it is high the interaction reduces the hazard in the household Gini model but increases it in the educational Gini model. These findings only partially support Blau's postulate that high differentiation at the micro level favors social interaction.

The results of all such comparisons are summarized in Table 6. The first panel of the table shows no consistent evidence that low differentiation at the cluster level depresses social interaction. The second panel, however, does show that social interaction effects are higher in eleven of twelve highly heterogenous clusters. Looking at inequality, only Colombia's high differentiation models, with inequality measured by the educational Gini, show the expected positive interaction

with the social interaction measure. The interactions of the social interaction dummy with the consolidation term dummies are negative as expected in only 9 of 24 cases.

The lower two panels of Table 6 show the *municipio*-level interactions. On this macro level of aggregation, low inequality has the expected positive effects in nine of twelve low-differentiation models. The exceptions are the educational models in Paraguay, where the interactions are negative. The heterogeneity measures are more consistent: nine of twelve are positive. Looking at the high-differentiation *municipio*-level models, inequality has the expected negative effects in five of the six Colombian models and none of the Paraguayan models. Examining heterogeneity, the expected relationships appear in five of six Colombian models and in four of six Paraguayan models. The interactive consolidation effects are ambiguous in both low- and high-differentiation panels.

Discussion and conclusions

Social interaction effects on immunization are evident in both Colombia and Paraguay, and their magnitudes are comparable to, or exceed, household and program effects. It is in highly differentiated clusters and less-differentiated *municipios* where I find the strongest graphic evidence that social structure conditions social interaction effects. Indeed, Blau's theory states that diffusion effects ought to be maximal in these kinds of structures. The second hypothesis is thus supported. But there is only partial support for the third hypothesis. Of the three differentiation measures, only heterogeneity consistently has the expected positive cluster-level effects, only in highly differentiated clusters. Yet there are unmistakable level effects. All three parameters tend to be oppositely signed in low- and high-differentiation clusters. The supporting

evidence at the *municipio* level is somewhat indirect. Instead of the expected negative effects when differentiation is high, the results show positive effects where differentiation is low. Perhaps structural effects are too subtle to be precisely measured in highly differentiated *municipios*. The measures may also be picking up different dimensions of differentiation. In Colombia, inequality and heterogeneity measures are internally consistent in less differentiated clusters and *municipios* while, in Paraguay, they are consistent in the more differentiated structures. The fourth hypothesis- that consolidation effects are everywhere negative- is only partly supported. The consolidation terms may not absorb all the consolidation effects because they are inadequately measured by the simple Pearson correlations.

The differing interactive effects, it must be remembered, reflect both structural differences and varying social interaction effects in the two countries. The latter were more pervasive in Colombia, affecting outcomes even where they were least intense. I conclude that contagion affected immunization behaviors in both countries over the 1985-90 period, and that the effects were comparatively stronger in Colombia. It is plausible that contagion processes affect other demographically important health behaviors in similar fashion. Examples include home use of oral rehydration therapy, infant feeding practices, treatment of acute respiratory infections, contraceptive use and prenatal and delivery care. These behaviors alone can prevent most of the child deaths in the developing world (WHO 1978).

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Table 1. Descriptive statistics for hazard risk sets and observed covariates, Colombia and Paraguay DHSII, 1990

	Colombia			Paraguay		
Variable	**N**	**Mean**	**Std. Dev**	**N**	**Mean**	**Std. Dev**
children ages 9-68m	1263			1214		
failed	945			674		
censored (%)	318 (25.2)			540 (44.5)		
months exposure	10593			19325		
duration structure						
age 9 m		0.238	0.426		0.126	0.332
age 10 m		0.114	0.317		0.061	0.240
age 11 m		0.098	0.298		0.058	0.234
age 12 m		0.068	0.251		0.050	0.217
age 13 m		0.055	0.228		0.045	0.207
age 14 m		0.046	0.209		0.042	0.200
age 15-17 m		0.099	0.299		0.107	0.310
age 17-19 m		0.073	0.260		0.090	0.286
age 19-21 m		0.054	0.227		0.077	0.266
age 21-23 m		0.041	0.198		0.066	0.248
age 24+ m		0.113	0.317		0.279	0.449
OPV3/DPT3 coverage[1]						
0-60%		0.062	0.242		0.327	0.469
60-80%		0.215	0.411		0.207	0.405
80-90%		0.159	0.366		0.082	0.274
> 90%		0.478	0.500		0.093	0.291
not reported		0.086	0.280		0.290	0.454
birth cohorts						
age 8-11 m		0.032	0.175		0.011	0.105
age 12-23 m		0.194	0.396		0.098	0.298
age 24-35 m		0.219	0.414		0.167	0.373
age 36-68 m		0.555	0.497		0.724	0.447
household risk factors						
female hh head		0.124	0.329		0.069	0.254
second or higher birth		0.675	0.469		0.882	0.322
mother primary or no ed		0.546	0.498		0.830	0.376
no Spanish					0.542	0.498
spouse farmer		0.067	0.249		0.496	0.562
social interaction		0.281	0.148		0.361	0.217

[1] Colombia: OPV3 among ages 12-23m; Paraguay: DPT3 among ages 12-23m.

Table 2. Descriptive statistics for social interaction and social structural variables, Colombia and Paraguay, 1990

	Colombia			Paraguay		
Variable	**median**	**Mean**	**std. dev.**	**median**	**mean**	**std. dev.**
Cluster level						
inequality (Ginis)						
housing	0.715	0.656	0.165	0.668	0.620	0.189
educational	0.616	0.621	0.185	0.779	0.740	0.142
heterogeneity (entropies)						
nativity	0.904	0.836	0.203	0.698	0.691	0.258
Occupations	0.551	0.535	0.126	0.535	0.506	0.173
union status	0.842	0.809	0.152	0.783	0.779	0.123
consolidation						
housing*nativity	0.103	0.047	0.578	-0.091	-0.085	0.262
housing*occupations	-0.259	-0.168	0.533	-0.131	-0.188	0.315
housing*union status	-0.273	-0.177	0.483	-0.167	-0.106	0.339
education*nativity	-0.191	-0.089	0.605	-0.101	-0.050	0.328
education*occupations	0.026	0.057	0.585	-0.241	-0.203	0.347
education*union status	0.138	0.043	0.509	0.050	0.034	0.290
Municipio level						
inequality (Ginis)						
housing	0.574	0.573	0.154	0.431	0.471	0.187
educational	0.431	0.482	0.153	0.726	0.631	0.198
heterogeneity (entropies)						
nativity	0.937	0.897	0.131	0.791	0.724	0.253
occupations	0.632	0.628	0.066	0.557	0.543	0.110
union status	0.870	0.846	0.116	0.818	0.805	0.092
consolidation						
housing*nativity	0.338	0.185	0.419	0.116	0.120	0.391
housing*occupations	-0.107	-0.055	0.513	-0.537	-0.502	0.238
housing*union status	0.158	0.053	0.468	-0.096	-0.092	0.418
education*nativity	-0.395	-0.396	0.400	-0.147	-0.100	0.396
education*occupations	-0.297	-0.298	0.590	-0.629	-0.531	0.249
education*union status	-0.546	-0.442	0.310	0.056	-0.060	0.406

Table 3. Baseline piecewise hazard models for full immunization, Colombia

	Model 1			Model 2		
Variable	beta	se(beta)	exp(B)	beta	se(beta)	exp(B)
intercept	1.817	0.212		0.508	0.256	
age 10 m	1.423*	0.114	4.148	1.549*	0.129	4.706
age 11 m	0.915*	0.137	2.498	1.498*	0.159	4.471
age 12 m	0.834*	0.153	2.302	2.082*	0.173	8.021
age 13 m	1.093*	0.154	2.982	2.528*	0.191	12.528
age 14 m	0.582*	0.195	1.790	2.059*	0.237	7.839
age 15-17 m	0.271‡	0.158	1.311	1.781*	0.202	5.933
age 17-19 m	0.186	0.183	1.205	1.674*	0.221	5.331
age 19-21 m	0.148	0.213	1.159	1.604*	0.246	4.974
age 21-23 m	0.404*	0.220	1.498	1.836*	0.253	6.273
age 24+ in	0.018	0.176	1.019	1.428*	0.214	4.169
OPV3 60-80%	0.459*	0.202	1.582	0.286	0.220	1.332
OPV3 80-90%	0.642*	0.207	1.899	0.463*	0.224	1.589
OPV3 > 90%	0.739*	0.200	2.094	0-653*	0.217	1.921
OPV3 not reported	0.594*	0.235	1.810	0.426‡	0.254	1.531
age 8-11 m	0.197 ‡	0.101	1.218	0.105	0.106	1.111
age 12-23 m	0.503*	0.104	1.653	0.142	0.108	1.152
age 24-35 m	0.651*	0.182	1.918	-0.332‡	0.171	0.717
female hh head	-0.049	0.113	0.952	-0.063	0.113	0.939
second or higher birth	-0.207*	0.076	0.813	-0.183*	0.078	0.833
mother primary or no ed	-0.027	0.075	0.973	-0.010	0.077	0.990
spouse farmer	-0.181	0.161	0.835	-0.264	0.178	0.768
social interaction effect				2.263*	0.125	9.615
log-likelihood		-1419.6			-1684.8	

* $p < .05$, ‡ $p < .10$ (two-tailed test)

Table 4. Baseline piecewise hazard models for full immunization, Paraguay

	Model 1			Model 2		
Variable	beta	se(beta)	exp(B)	Beta	se(beta)	exp(B)
intercept	1.195	0.195		-0.858	0.259	
age 10 m	1.753*	0.184	5.769	1.784*	0.190	5.955
age 11 m	1.346*	0.201	3.843	1.505*	0.208	4.504
age 12 m	1.065*	0.222	2.901	1.377*	0.229	3.961
age 13 m	0.971*	0.235	2.641	1.412*	0.248	4.104
age 14 m	0.883*	0.247	2.419	1.515*	0.264	4.550
age 15-17 m	0.730*	0.203	2.075	1.923*	0.228	6.844
age 17-19 m	0.739*	0.214	2.093	2.672*	0.250	14.471
age 19-21 m	0.751*	0.228	2.119	2.843*	0.288	17.168
age 21-23 m	0.776*	0.238	2.173	2.832*	0.301	16.987
age 24+ m	0.885*	0.185	2.423	2.843*	0.255	17.165
DPT3 60-80%	-0.101	0.123	0.904	-0.116	0.127	0.891
DPT3 80-90%	0.442*	0.146	1.556	0.481*	0.141	1.617
DPT3 > 90%	0.177	0.172	1.193	0.159	0.167	1.172
DPT3 not reported	-0.053	0.109	0.948	-0.054	0.115	0.947
age 8-11 m	0.559*	0.111	1.748	0.011	0.122	1.011
age 12-23 m	1.035*	0.129	2.816	-0.121	0.134	0.886
age 24-35 m	1.315*	0.271	3.723	0.044	0.273	1.045
female hh head	-0.091	0.156	0.913	-0.088	0.172	0.916
second or higher birth	-0.260*	0.107	0.771	-0.227*	0.104	0.797
mother primary or no ed	-0.339*	0.100	0.713	-0.306*	0.104	0.736
no Spanish	-0.529*	0.107	0.589	-0.437*	0.104	0.646
spouse farmer	-0.347*	0.106	0.707	-0.302*	0.105	0.739
social interaction effect				3.171*	0.183	23.826
log-likelihood		-434.2			-697.8	

* $p < .05$, ‡ $p < .10$ (two-tailed test)

Table 5. Negative log-likelihoods and goodness-of-fit for additive structural immunization hazard models, cluster level, Colombia and Paraguay 1990

	Colombia		Paraguay	
Variable and model	housing	education	housing	education
Cluster level				
nativity				
Baseline	-1684.8		-697.8	
Low structural effects	-1687.0	-1686.4	-698.5	-698.5
high structural effects	-1685.8	-1684.9	-700.1	-702.7‡
occupations				
Baseline	-1684.8		-697.8	
Low structural effects	-1685.2	-1684.8	-701.1‡	-701.9*
high structural effects	-1685.0	-1688.5‡	-699.8	-699.6
union status				
Baseline	-1684.8		-697.8	
Low structural effects	-1697.0*	-1700.3*	-698.9	-698.5
high structural effects	-1689.2*	-1690.3*	-700.3	-702.7*
Municipio level				
nativity				
baseline	-1684.8		-697.8	-713.3
Low structural effects	-1688.6‡	-1689.4*	-700.2*	-719.1*
high structural effects	-1689.1*	-1689.5*	-709.1*	-718.5*
occupations				
baseline	-1684.8		-697.8	-713.3
low structural effects	-1690.1*	-1688.6‡	-709.9*	-716.6‡
high structural effects	-1689.0*	-1688.0‡	-700.8	-715.9
union status				
baseline	-1684.8		-697.8	
low structural effects	-1695.5*	-1695.1*	-702.6*	-704.8*
high structural effects	-1688.8*	-1693.3*	-702.8*	-700.7

* $p < .05$, ‡ $p < .10$ by log-likelihood ratio test
NOTE: all structural models add 3 degrees of freedom

Table 6. Interactive effects of high social interaction with social differentiation parameters on the hazard of full immunization, cluster and municipio levels, Colombia and Paraguay 1990

	Colombia						Paraguay					
	housing			education			housing			education		
	I	H	C	I	H	C	I	H	C	I	H	C
Cluster level												
low differentiation												
nativity	+‡	-	+	+	-	+‡	+	+	+	+	-	+
occupations	+‡	+	+‡	+	+	+	+	-	-	+	+	+
Union status	+‡	-‡	+	+	-‡	+	+	+*	-	+	-	+
high differentiation												
nativity	-	+	+	+‡	+*	-*	-*	+	-*	-*	+	-*
occupations	-‡	+	+	+	+	-	-	+	-	-	+	-
union status	-‡	+	+	+	+	+	-	-	-	-	+	-
Municipio level												
low differentiation												
nativity	+‡	+	+*	+	+	+	+	+	+	-	-	-
occupations	+‡	-	-	+	-	+	+	+	-	-	+	-*
union status	+*	+	-	+	+	+	+	+‡	-	-	+‡	-
high differentiation												
nativity	-*	+	+	-	+	+*	+	+	-	+*	+	-
occupations	+*	+	-	-	-	-	+	+	-	+	+	-
union status	-*	+‡	-	-	+	-	+	-‡	-	+*	-	-

* p < .05, ‡ p < . 10 by log-likelihood ratio test

Fig. 1. Hazard of full immunization, by social interaction effect quartiles, COL DHSII 1990

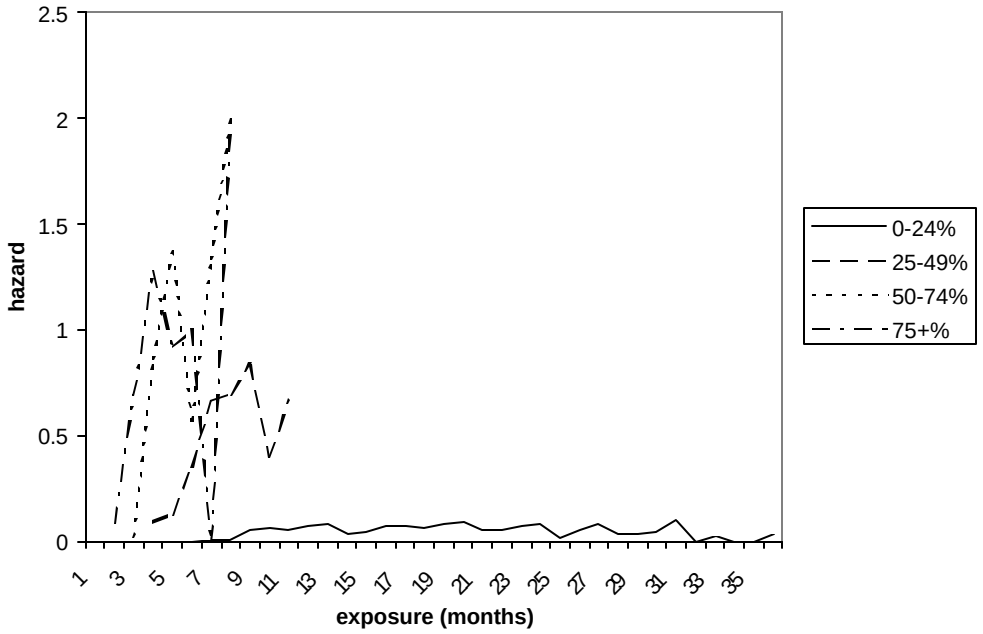


Fig. 2. Hazard of full immunization, by social interaction effect quartiles, PAR DHSII 1990

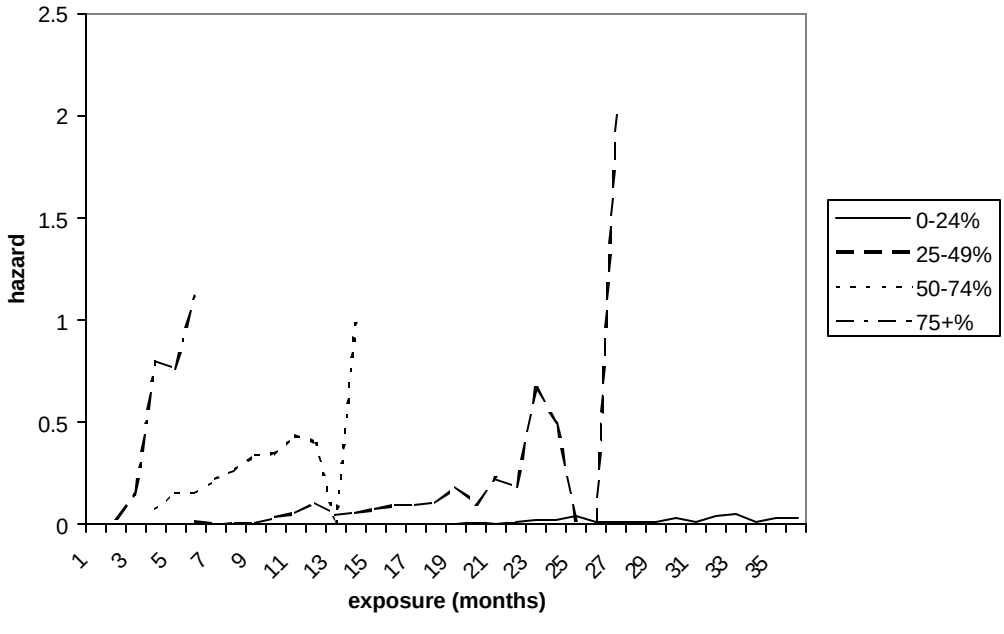


Fig. 3. Hazard of full immunization conditional on household factors, municipal coverage 80-90% and social interaction effects, by score quartiles, COL DHSII 1990

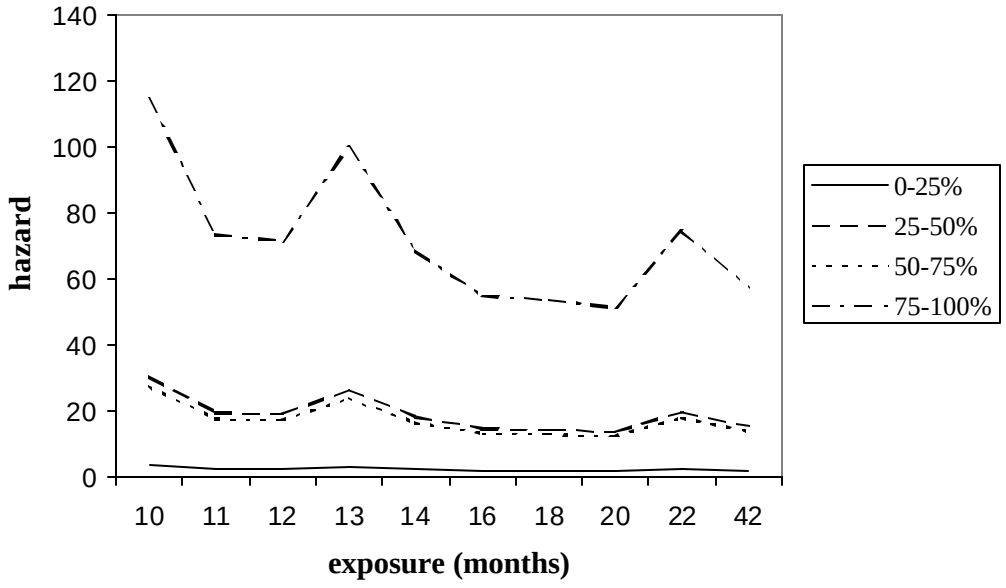


Fig. 4. Hazard of full immunization conditional on household risk factors, municipal coverage 80-90% and social interaction effects, by score quartiles, Paraguay DHSII, 1990

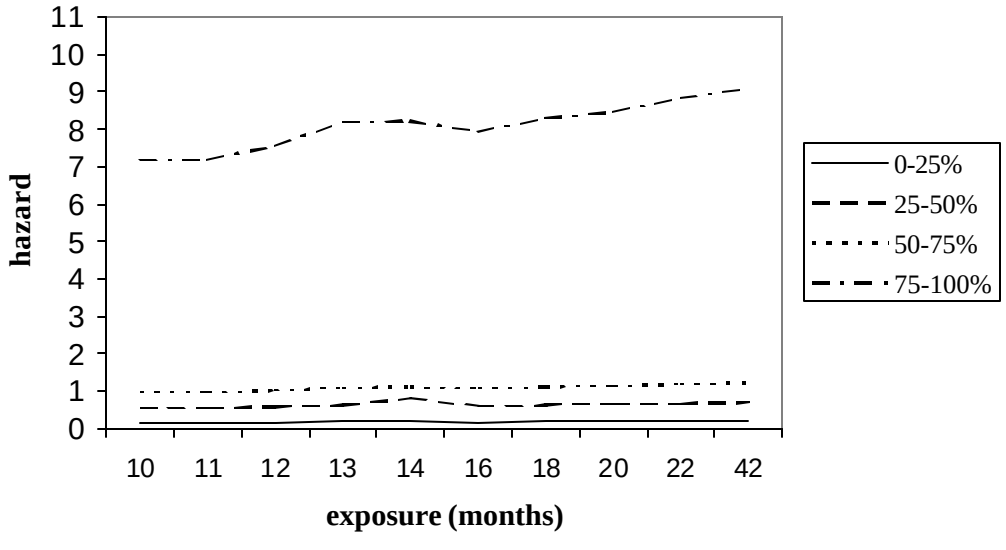


Fig. 5. Effects of 90%+ municipal vaccine coverage on hazard of full immunization, by risk group, COL DHSII, 1990

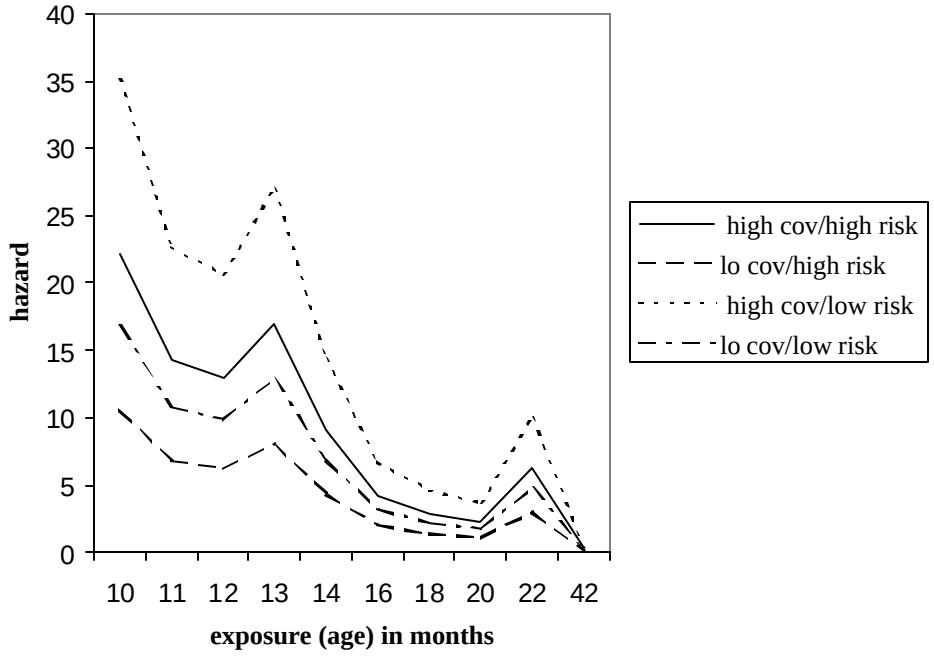


Fig. 6. Effects of 90%+ municipal vaccine coverage on hazard of full immunization, by risk group, PAR DHSII 1990

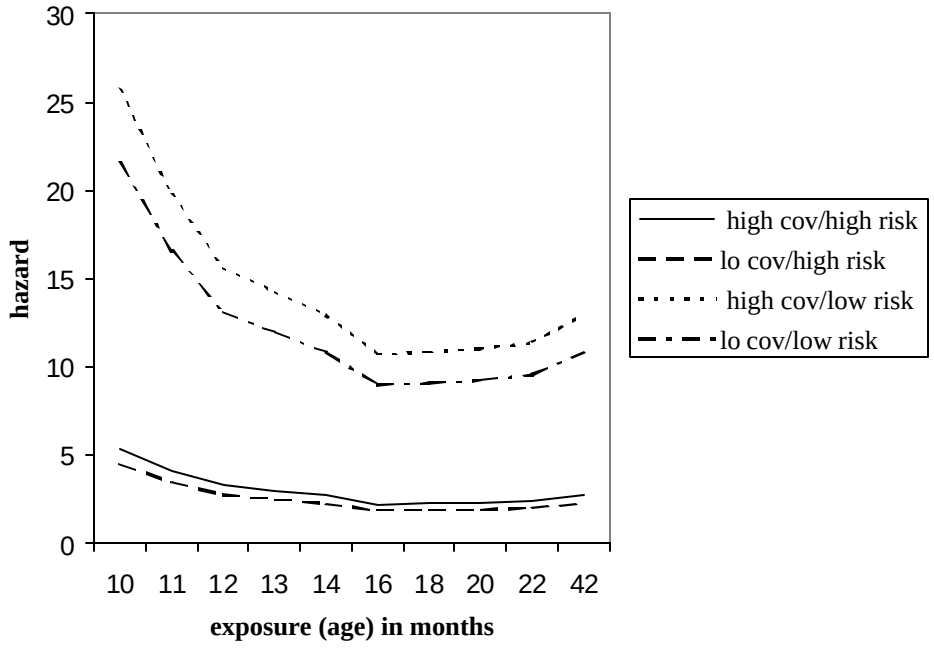


Fig. 7. Hazard of full immunization conditional on household factors, municipal coverage,high social interaction and structural differentiation (housing gini+nativity+corr(hgini,nativity), COL DHSII, 1990

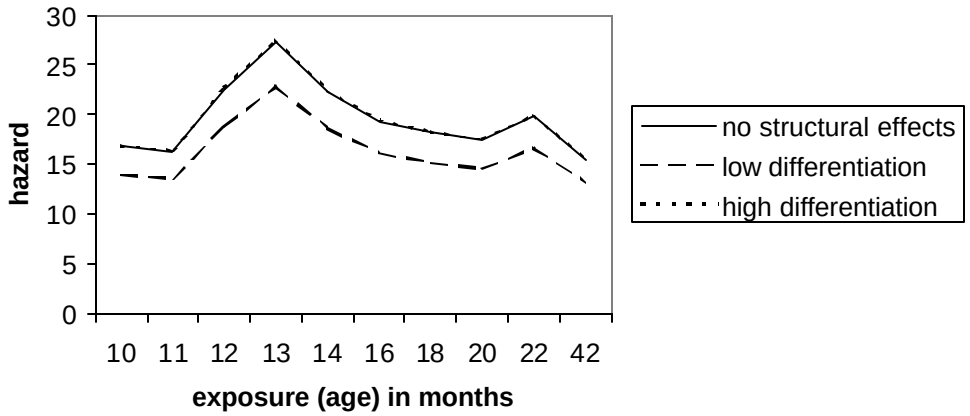


Fig. 8. Hazard of full immunization conditional on household factors, municipal coverage, high social interaction and structural differentiation (education gini+nativity+corr(edgini,nativity), COL DHSII, 1990

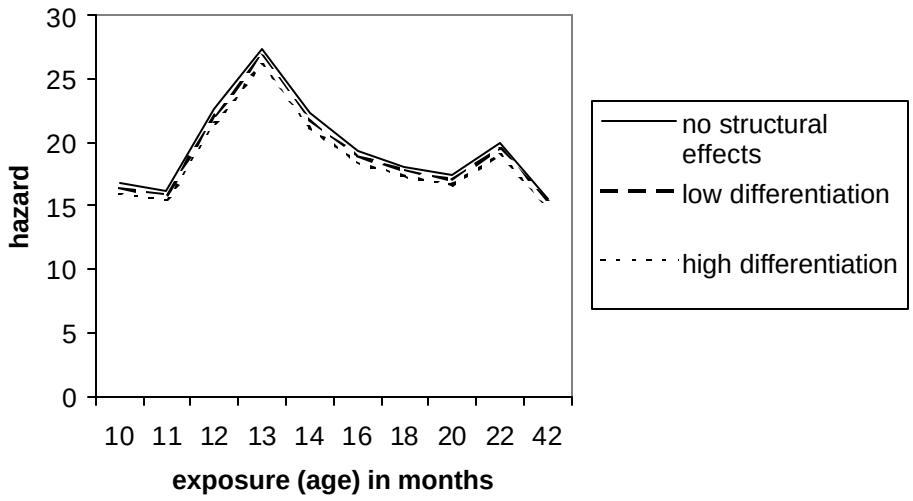


Fig.9. Hazard of full immunization conditional on household factors, municipal coverage, high social interaction and "low" interactive structural effects (housing gini+ nativity+corr(hgini,nativity)+si*hgini), COL DHSII, 1990

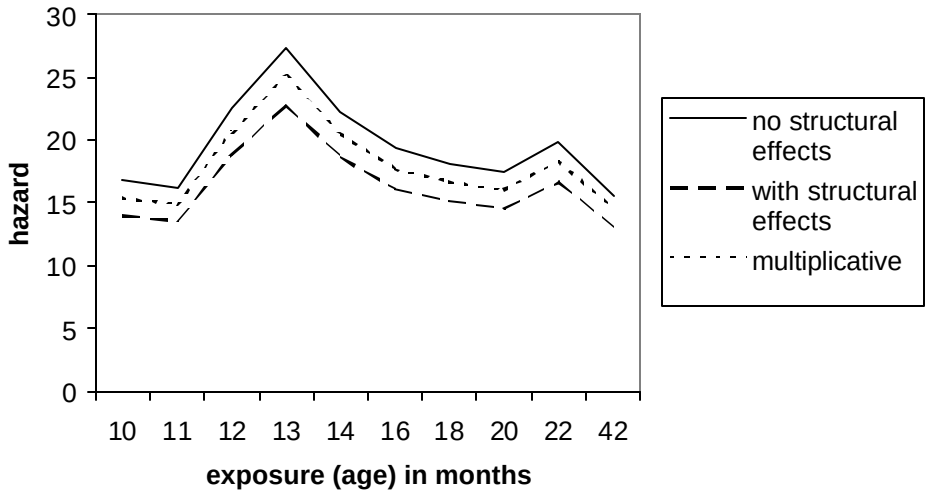


Fig. 10. Hazard of full immunization conditional on household factors, municipal coverage, high social interaction and "low" interactive structural effects (education gini+ nativity+corr(edgini,nativity)+si*edgini), COL DHSII, 1990

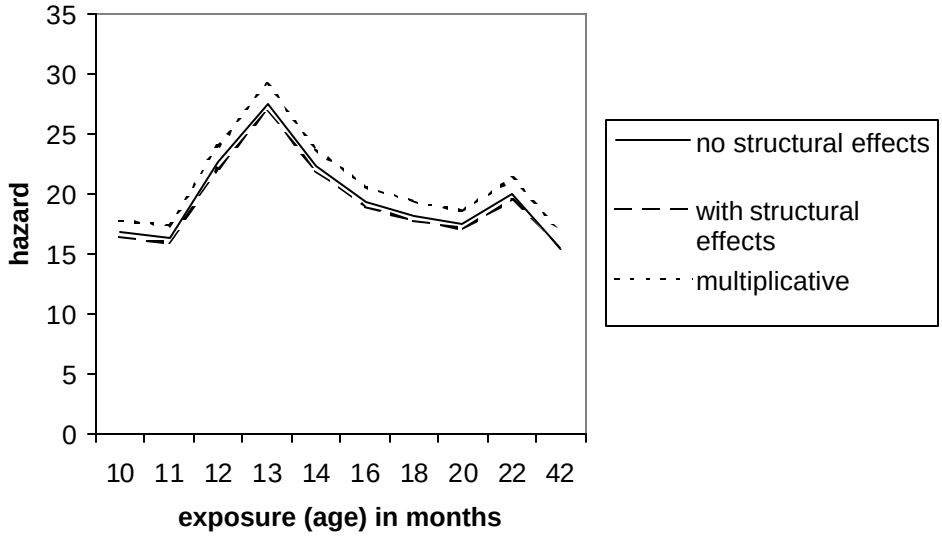


Fig. 11. Hazard of full immunization conditional on household factors, municipal coverage, high social interaction and "high" interactive structural effects (housing gini+ nativity+corr(hgini,nativity)+si*hgini), COL DHSII, 1990

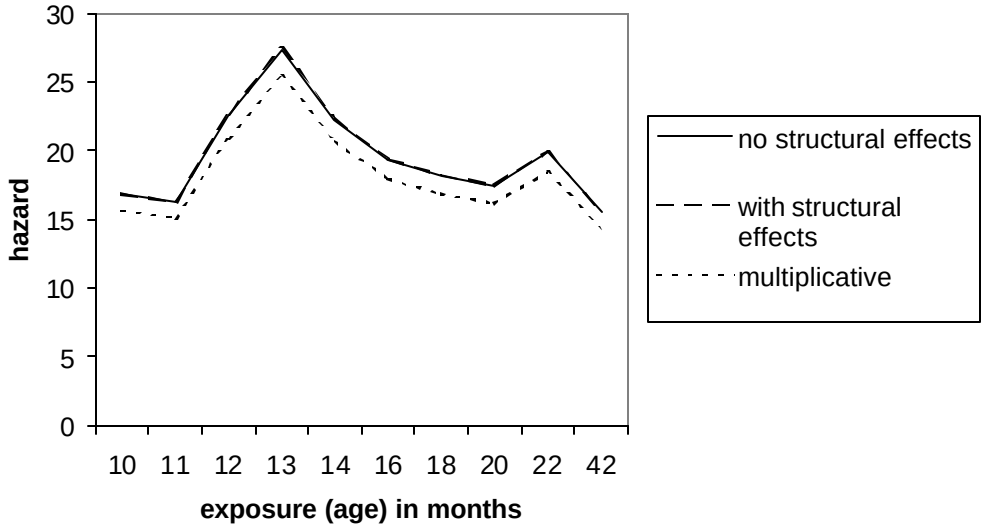
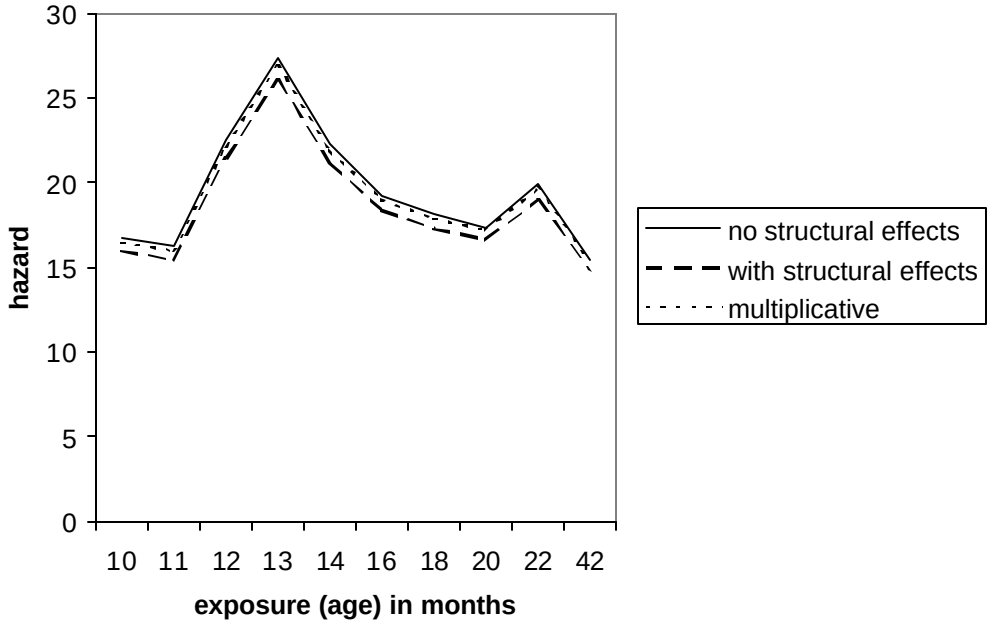


Fig. 12. Hazard of full immunization conditional on household factors, municipal coverage, high social interaction and "high" interactive structural effects (education gini+nativity+corr(edgini,nativity)+si*edgini), COL DHSII, 1990



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