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BAYESIAN ANALYSIS OF RESPONDENT-DRIVEN SURVEYS WITH OUTCOME UNCERTAINTY

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Contents

Todo list

List of sections to revise

1. Respondent-driven sampling;
2. Add Hierarchical modelling chapter;
3. Should I add a subsection in Bayesian Statistics revising Prevalence estimation models using Bayesian paradigm?

What to do after?

1. Notes about Bivariate Beta;
2. Study case about CAR models in bernoulli aspect.

1 Introduction

Hidden or hard-to-reach populations have two main features: no sampling frame exists, given that their size and boundaries are unknown, and there are privacy concerns because the subjects are stigmatized or have illegal behavior (HECKATHORN, 1997). Fear of exposition or prosecution complicates the enumeration of the populations and the learning about them. Moreover, if the occurrence frequency of the condition is low, there are high logistic costs involved. Some examples are heavy drug users, sex workers, homeless people, and men who have sex with men.

Research has been carried out with the development of some methods to reach these populations, such as, for example, snowball sampling (GOODMAN, 1961), key important sampling (DEAUX; CALLAGHAN, 1985), and targeted sampling (WATTERS; BIERNACKI, 1989). (HECKATHORN) introduced the Respondent-Driven Sampling (RDS) to fill some gaps from other methods he depicted in his work. In his proposed approach, the researchers select a handful of individuals from the target population and give them coupons to recruit their peers. The individuals receive a reward for being recruited and for recruiting, which creates a dual incentive system. After (HECKATHORN, 1997), several papers studied this topic more deeply.

Following the sampling from the target population, a questionnaire or a disease test is conducted. This work considers binary outcomes. For instance, asking about smoking status or testing for HIV infections. However, the diagnoses are subject to measure error, and regard their accuracy is a vital step (REITSMA et al., 2005). One common way to do this is to measure jointly *sensitivity* and *specificity*. The former is the ability to detect the condition, while the latter to identify the absence of it.

Nevertheless, because of our lack of knowledge about Nature itself, it is necessary to model the uncertainty of this process, and Bayesian Statistics is the indicated area of study. In the Bayesian paradigm, the parameters are random variables, and the beliefs about them are updated given new data. The idea is to propagate uncertainty about the outcome through the network of contacts, which has its probability distribution.

This work proposes to study the survey method Respondent-Driven Sampling (RDS), a chain-referral method with the objective of sampling from hard-to-reach

populations when necessary to estimate the prevalence of some binary condition from this population. The modeling also accounts for sensibility and sensitivity since the imperfection of the detection tests. We also intend to apply this framework efficiently, comparing Monte Carlo algorithms and Laplace approximations.

2 Theoretical background

In this chapter, we shall describe the theoretical background taken under consideration for the developed models and analysis, including Bayesian statistics (Section 2.4), the prevalence estimation problem (Section 2.1), Respondent-driven sampling (Section 2.2), and computational methods (Section 2.5) used in our research.

Fix order after.

2.1 Prevalence estimation problem

The study of how health-related conditions are distributed among populations is known as *Epidemiology* (ROTHMAN; GREENLAND; LASH, et al., 2008, p. 32), which aims to derive valid estimates for potential causes from diseases that affect people. It is a fundamental research area in policy formulation, implementation of prevention programs, and development of laws. In order to accomplish these goals, the epidemiologists use some *measures of disease frequency*, including *incidence* and *prevalence*. The former is related to the proportion of new cases of a disease given a period of time, while the latter is the proportion of individuals exposed at time t and it is the object of study of this section. An interesting point is the following:

> Diseases with high incidence rates may have low prevalence if they are rapidly fatal or quickly cured. Conversely, diseases with very low incidence rates may have substantial prevalence if they are nonfatal but incurable. (ROTHMAN; GREENLAND; LASH, et al., 2008, p. 46).

As a result, prevalence represents both incidence and the duration of disease. Noordzij et al. (2010, p. c18) highlights that prevalence reveals the burden of a disease in respect to its effects on society, such as, monetary costs, quality of live, and morbidity. They also comment that when measured periodically, its evolution can identify potential causes of the infection and prevention and care methods. We remark that when it is impossible to test every individual at the same time, we assume that all individuals remain exposed to the disease at time of the last tested individual.

Should I mention more reasons to study prevalence?

Consider a population of interest and a known condition, such as, for instance, a disease or a binary behavior. A diagnostic test is done in the individuals to measure the presence or the absence of this condition, such as serological tests.

It might be nice to add examples.

Mathematically, we denote $\theta \in (0,1)$ the prevalence of the condition, which is the parameter of interest. Let I be a index set for the individuals. We also denote Y_i^{true} the indicator function of the presence of the condition in the ith individual, that is,

$$Y_i^{\text{true}} = \begin{cases} 1, & \text{if individual } i \text{ has the condition.} \\ 0, & \text{otherwise.} \end{cases}$$

Assume for simplicity that all tests are performed at time t. Assume that Y_i indicates the result of the test, then

$$Y_i = \begin{cases} 1, & \text{if test was positive in individual } i. \\ 0, & \text{otherwise.} \end{cases}$$

Since it is not usually feasible to test everyone in the population, it is necessary to random select individuals from the population. On that point, other sampling approaches may be better options, such as stratified random sampling, systematic sampling, and two-stage cluster sampling. From that experiment, we get a sample $\{y_1, ..., y_n\}$. Based on that outcomes the Maximum Likelihood Estimator is the following expression

$$\hat{p} = \frac{1}{n} \sum_{i=1}^{n} y_i, \tag{2.1}$$

which is an estimator for the *apparent prevalence*, that is, the probability of a positive outcome.

However, this estimator assumes that the diagnostic test used is perfect, which is often incorrect . It is also not interesting when the samples are not randomly selected (See Section 2.2). From that point, it is crucial to regard the evaluation of the diagnostic procedure by some measurement. Šimundić (2009, p. 2) presents several options with different aspects, such as the *likelihood ratio*, *sensitivity and specificity*, and *the area under the ROC curve*. In this work, we consider the sensitivity and specificity of the test.

Provide some reference

It may be good to justify this choice.

A perfect test would discriminate every sick individual from the non-sick ones. Given that there is not such thing, we suppose having a *gold standard test* that is the best available test (VERSI, 1992) to diagnose a particular disease. Its result is a proxy for the real Y_i^{true} and

Should I cite when this not true? Cite Rutjes (2007)

> In the context of infectious diseases, a gold standard can be a very precise molecular test that detects the presence of the pathogen's genetic material, polymerase chain reaction (PCR) for instance. (BASTOS; CARVALHO; GOMES, 2021, p. 125).

From the gold standard, we can evaluate a second test, typically faster or cheaper. The possible results upon comparing these tests are presented in table 1. The definitions for each initials in the table are the following:

a) True positive (TP): when both tests agree that the individual has the disease.

b) True negative (TN): when both tests agree that the individual does not have the disease.

c) False positive (FP): when the test under evaluation has a positive diagnose, despite the golden standard being negative.

d) False negative (FN): when the test under evaluation has a negative diagnose, despite the golden standard being positive.

	$Y = 0$	$Y = 1$
$Y^{\text{true}} = 0$	TN	FP
$Y^{\text{true}} = 1$	FN	TP

Table 1 – Two-by-two table that compares the result from the gold standard to the test under evaluation.

For now, we drop the index i in the random variables Y_i and Y_i^{true}. Let $p = \Pr(Y = 1)$ be the probability of a positive test. We call p the *apparent prevalence* since it is what the researchers observe. Equation (2.1) is an estimator for it. We also have that $\Pr(Y^{\text{true}} = 1) = \theta$. Notice that p depends on the used test, while θ does not. In prevalence estimates, we will only have $\theta = p$ if the test is perfect or the test is the gold standard itself. Define the following:

Definition 2.1.1 (Sensitivity)**.** Probability of a positive test correctly identified. In mathematical terms, conditioned on $Y^{\text{true}} = 1$, the *sensitivity* γ_s is the probability of $Y = 1$:

$$\gamma_s = \Pr(Y = 1 | Y^{\text{true}} = 1). \tag{2.2}$$

Definition 2.1.2 (Specificity)**.** Probability of a negative test correctly identified. In mathematical terms, conditioned on $Y^{\text{true}} = 0$, the *specificity* γ_e is the probability of $Y = 0$:

$$\gamma_e = \Pr(Y = 0 | Y^{\text{true}} = 0). \tag{2.3}$$

Theorem 1 (Relation between prevalence and apparent prevalence)**.** *These quantities are related by the following equation:*

$$p = \gamma_s \theta + (1 - \gamma_e)(1 - \theta). \tag{2.4}$$

Proof. This is a direct application of the definition of conditional probability and the countable additivity axiom of Probability:

$$\begin{aligned}
p &= \Pr(Y=1) = \Pr(Y=1, Y^{\text{true}}=1) + \Pr(Y=1, Y^{\text{true}}=0) \\
&= \Pr(Y=1|Y^{\text{true}}=1)\Pr(Y^{\text{true}}=1) + \Pr(Y=1|Y^{\text{true}}=0)\Pr(Y^{\text{true}}=0) \\
&= \Pr(Y=1|Y^{\text{true}}=1)\Pr(Y^{\text{true}}=1) \\
&+ (1-\Pr(Y=0|Y^{\text{true}}=0))(1-\Pr(Y^{\text{true}}=1)) \\
&= \gamma_s\theta + (1-\gamma_e)(1-\theta).
\end{aligned}$$

□

The intuition behind this equation is pretty simple: the proportion of positive test counts the correct identified exposed individuals and the incorrect identified not exposed. Equation (2.4) also reveals that if $\gamma_s = \gamma_e = 1$, we have the trivial case $p = \theta$. Moreover, if $\gamma_s = \gamma_e = 0.5$, we have that $p = 0.5$ and there is no information about θ.

A frequentist approach assumes that θ is fixed and unknown. Its inference is based on the point estimate for the apparent prevalence $\hat{p}$ given in Equation (2.1), along with a Confidence Interval, such as the Wald Confidence Interval built with a normal approximation. In order to provide a point estimate for $\hat{\theta}$, Rogan and Gladen (1978, p. 73) propose

$$\hat{\theta}^{RG} = \frac{\hat{p} - (1-\gamma_e)}{\gamma_s + \gamma_e - 1}.$$

Suppose a disease with prevalence $\theta = 0.01$. In this case, we would have that $p \approx 1 - \gamma_e$ by equation (2.4). Given the randomness, it is possible to have $\hat{p} < 1 - \gamma_e$, which would define a useless estimative for θ. Besides that, Confidence Intervals for that expression does not include uncertainty about γ_e and γ_s. On the other side, a Bayesian approach let θ be a random variable, allowing the researcher to incorporate their uncertainty on the prior distribution, which is explained in Section 2.4. It also allows to include uncertainty in sensitivity and specificity of the test. According to Branscum, Gardner, and Johnson (2005):

> Diagnostic-test evaluation is particularly suited to the Bayesian framework because prior scientific information about the sensitivities and specificities of the tests and prior information about the prevalences of the sampled populations can be incorporated. (BRANSCUM; GARDNER; JOHNSON, 2005, p. 1).

Therefore, this work focus on the Bayesian paradigm.

2.2 Respondent-driven sampling

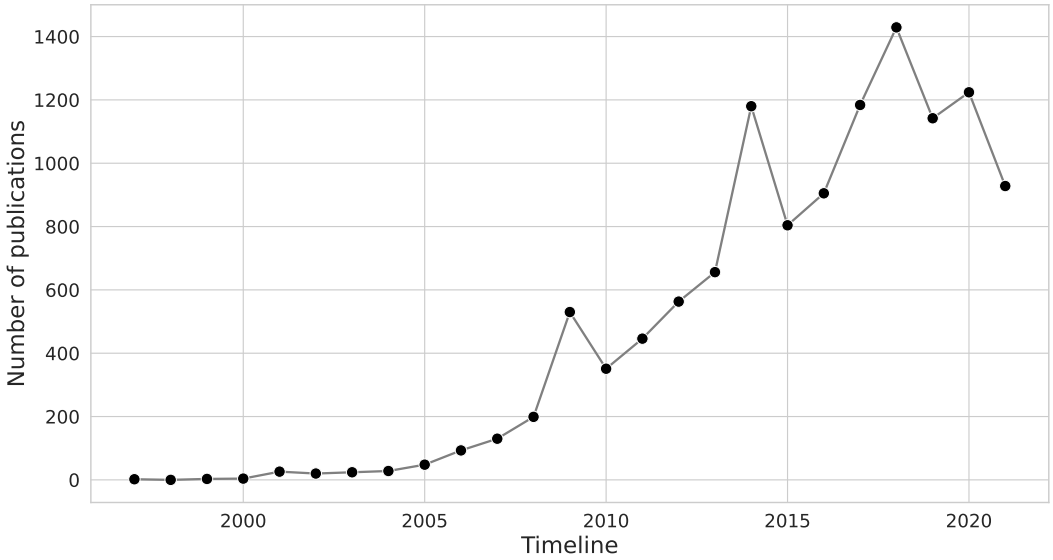
Respondent-driven sampling (RDS) is a procedure developed by Heckathorn (HECKATHORN, 1997) to survey *hidden* or *hard-to-reach populations*, whose main characteristic is the absence of a sampling frame, i.e.,it is not possible to enumerate its individuals since size and boundaries are unknown. The second characteristic of these populations is the confidentiality concerns, given that membership is stigmatized or illegal. With that aspect, traditional sampling methods which produce probability samples are infeasible. To overcome this, Snowball Sampling (GOODMAN, 1961) is the most common method, and it relies on the respondents to nominate more subjects within the population as a snowball. Examples of studied groups include people who inject drugs (PWID), men who have sex with men (MSM), and female sex workers (FSW) (GILE et al., 2018, p. 66).

Heckathorn's proposal (1997) was to specialize this method without the need of nominating peers. In this approach, the researchers select some individuals, called *seeds* from the target population, and give them a fixed amount of *recruitment coupons* to recruit their peers. Each recipient of the coupons reclaims it in the study site, is interviewed, and receives more coupons to continue the recruitment. This process occurs until it reaches some stopping criteria, such as the sample size achieving some desired number. The sampling is without replacement, so the participants cannot be recruited more than once. Moreover, the respondents inform how many subjects from the population they know. Other less usual methods include Key Important Sampling (DEAUX; CALLAGHAN, 1985), and Targeted Sampling (WATTERS; BIERNACKI, 1989), both are convenience sampling methods.

According to Gile et al. (2018, p. 66), there are two main advantages of RDS over other snowball samplings. First, the fixed number of recruitment coupons enforces the network gets deeper and distant from the seeds, which reduces the dependence of the final sample from the initial chosen by researchers. Second, since the recruited subjects do not have to name their peers, confidentiality is maintained until the recruitment is completed. Other problems cited by Heckathorn (1997, p. 175) include biases towards individuals who are more cooperative, biases by masking when the participants do not name friends for the next wave to protect them, and individuals with more links may be oversampled. RDS offers a solution with a *dual incentive system*, explained in Subsection 2.2.1.

Since the creation of the method by Heckathorn, several papers have been published, as Figure 1 presents. The figure was produced searching publications with the term "Respondent-driven sampling." These works generally aim to give basis

Figure 1 – Publications by year with the term "Respondent driven sampling" from 1997 to 2021.



Source: htpps://app.dimensions.ai. Exported on October 31, 2021.

to public health policies. Good examples in Brazil are (DAMACENA et al., 2019), (MOTA, 2012), and (BASTOS; BASTOS, et al., 2018). Damacena et al. (2019) apply the RDS method to carry out biological and behavioral surveillance in FSW populations from twelve cities in Brazil. Mota (2012) proposes the RDS method in MSM populations from ten cities in Brazil. Bastos, Bastos, et al. (2018) study several sexually transmitted infections among transgender women from twelve Brazilian cities.

2.2.1 Details about the sampling procedure

The RDS method was expanded by Heckathorn (2002). It detailed two aspects: introducing a way to correct *homophily* biases that is the tendency for individuals to connect to others similar to them, and *personal network size* or *degree* that is the number of connections of an individual within the target population. It also presented a bootstrapping procedure to quantify uncertainty about inferences. Salganik and Heckathorn (2004) slightly modified the RDS procedure and introduced proof that under some regularity conditions, RDS estimators were asymptotically unbiased. World Health Organization (2013) is a reference to know how to execute an RDS survey. According to it

> Seeds are non-randomly selected members of the survey population who initiate the RDS recruitment process. From each seed, a

> recruitment chain is expected to grow. Seeds play an extremely important role in conducting an RDS survey. (WORLD HEALTH ORGANIZATION, 2013, p. 70).

No rule was established on the number of seeds to start the sampling. It typically varies from 2 to 32, with the mean being 10 (WORLD HEALTH ORGANIZATION, 2013, p. 70). The number can not be small since unsuccessful recruitments are common. A diverse choice among the target population may accelerate the convergence to equilibrium. It also allows the access to isolate and subpopulations. After this selection, three coupons are distributed to each participant. The coupons musta have information about survey site location, an unique identification code, telephone number, and opening hours. Gile et al. (2018, p. 67) highlights that "this number is chosen to strike a balance between the inferencial desire [...] and the practical necessity of guarding against early termination of the sample trees." Figure XXX illustrates an RDS process in XXX.

Subjects receive a reward for being interviewed and recruiting their peers within the target population, which establishes a dual incentive system. The *primary incentive* is the *individual-sanction-based control*, so there is a reward for participating in the survey. The second one is the *group-mediated social control* that influences the participants to induce others to comply to get the remuneration for the recruitment. When social approval is relevant for the members, recruitment can be more efficient and cheaper. It happens because material incentives are converted into peer-based symbolic since there is social influence involved. In conclusion, consenting to be recruited provide material and symbolic motivation to both recruiter and participant.

2.2.2 Assumptions and statistical properties

Clustering and homophily: extent to which people have connections with 'like' people (27); Hx = 2pi(1 - pi)R - 1

2.2.3 Models for the RDS Process

Although regression analysis of RDS data is frequently undertaken, the best method for accommodating correlation between participants (clustering) and the non-random sampling of recruits remains unknown."

2.2.3.1 Markov process

Describe as a Markov process (HECKATHORN, 1997)

2.2.3.2 Random walk

(HECKATHORN, 2002) Actually if was 2004.

2.2.3.3 Graphical model

Let $G = (V, E)$ be an undirected graph representing the hidden population. The *recruitment graph* $G_R = (V_R, E_R)$ represents the recruited individuals and the recruitment edges, that is, $(i, j) \in E_R$ if, and only if, i recruited j. Given that each individual can be sampled only once, it is not possible to observe the *recruitment-induced subgraph*, that is the induced subgraph generated by V_R. Moreover, the *coupon matrix* C defined by $C_{ij} = 1$ if the ith subject has at least one coupon before the jth recruitment event, is also observed with the recruitment times. Assuming an exponential and independent distribution of the times, the likelihood can be written explicitly, and the distribution interpreted as an exponential random graph model (CRAWFORD, 2016).

These models allowed several applications in social sciences, epidemiology, and statistics, including hidden populations size estimation (CRAWFORD; WU; HEIMER, 2018), regression (BASTOS; PINHO, et al., 2012), communicable disease prevalence estimation (ALBUQUERQUE et al., 2009), among others.

2.2.3.4 New model

Present the non-read-yet model.

2.2.4 Prevalence estimators

Review of RDS prevalence estimators: naive estimator; RDS-I estimator; RDS-II; Gile SS; HCG estimator;

weighting of those with more connections;

GLM procedures cited!

2.2.5 Regression methods

"A search of PubMed for the terms ‘respondent driven sampling’ and ‘regression’"

Regression techniques that already exist: weighted regression (33-37);

2.3 Generalized linear models

Generalized linear models are an extension of classical linear models. Let $y \in \mathbb{R}^n$ be a realization of a random variable $Y : \Omega \to \mathbb{R}^n$ associated with a phenomena such that each component Y_i is independent of the others. The systematic process in modelling is the specification of the vector $\mu = \mathbb{E}[Y]$ through a small number of parameters $\beta_1, \ldots, \beta_p$. The classical linear model assumes that $Y_i \overset{iid}{\sim} \text{Normal}(\mu_i, \sigma^2)$ and $\mu = X\beta$, where $X \in \mathbb{R}^{n \times p}$ is the data, where X_{ij} is the measure of the j-th covariate in the i-th individual.

The main generalization of this aspect is the introduction of the *link function*. This is a monotonic differentiable function g such that $\eta_i = g(\mu_i)$ and $\eta = X\beta$. Therefore the link function relates the linear predictor η to the expected value μ. The distribution of Y may also come from another exponential family distribution

Maybe explain or cite what is this?

Classical link functions when Y_i has Binomial distribution with parameter $0 < \mu < 1$ are

1. *logit*: $\eta = \log(\mu/(1-\mu))$ that represents the log odds of $Y_i = 1$.
2. *probit*: $\eta = \Phi^{-1}(\mu)$ where the $\Phi(\cdot)$ is the Normal cumulative distribution function;
3. *complementary log-log*: $\eta = \log(-\log(1-\mu))$.

2.4 Bayesian statistics

We can represent our beliefs and information about unknown quantities through probabilities. There are two more common interpretations: frequentist and Bayesian. While the frequentists define probability as the limit of a frequency in a large number of trials, the Bayesians represent an individual's degree of belief in a statement that is updated given new information. This philosophy allows assigning probabilities to any event, even if a random process is not defined (STATISTICAT, 2016).

In 1761, Reverend Thomas Bayes wrote for the first time the Bayes' formula relating the probability of a parameter after observing the data with the evidence (written through a likelihood function) and previous information about the parameter. Pierre Simon Laplace rediscovered this formula in 1773 (ROBERT, 2007), and this theory became more common in the 19th century. After some criticisms, a modern treatment considering Kolmogorov's axiomatization of the theory of probabilities

started after Jeffreys in 1939. The recent development of new computational tools brought these ideas again.

Therefore, Bayesian inference is the process of inductive learning using Bayes' rule, where inductive means that characteristics of a population are learned from a subset of it. We generally express numerical characteristics of the population as a parameter θ which is indirectly observed through numerical descriptions y of the population. Both are uncertain until the observation of a sample, when its information can decrease our uncertainty about the population characteristics (HOFF, 2009, p. 1-2).

The set of all possible outcomes y forms the *sample space* $\mathcal{Y}$, while the set of all possible parameters forms the *parameter space* Θ. Bayesian inference is composed by the following:

(a) *Prior distribution:* A probability distribution defined over Θ that quantifies our beliefs about θ before observing the data;

(b) *Sampling model:* A probability distribution of the data generation process that express our belief that $y \in \mathcal{Y}$ is the outcome when $\theta \in \Theta$ is true. When it is seen as function of the parameter, it is called *likelihood function*;

(c) *Loss function:* Only in a decision theory framework, it measures the error of a estimative $\delta \in \Theta$ in comparison to θ.

(d) *Posterior distribution:* Once we get the data y, it represents our updated beliefs out the parameter conditioned All inferences are based on this probability distribution.

Bayes' theorem establishes that when the sampling model is absolutely continuous with respect to some measure ν with conditional density $f_{Y|\theta}(y \mid \theta)$ and the prior distribution is a well defined probability measure μ_θ, the posterior distribution $\mu_{\theta|Y}(\cdot \mid y)$ is absolutely continuous with respect to μ_θ almost surely and its Radon-Nikodym derivative is (SCHERVISH, 2012, p. 16)

$$\frac{d\mu_{\theta|Y}}{d\mu_\theta}(\theta|y) = \frac{f_{Y|\theta}(y \mid \theta)}{\int_\Theta f_{Y|\theta}(y|t)d\mu_\theta(t)}. \tag{2.5}$$

When the prior distribution is absolutely continuous with respect to the Lebesgue measure, equation (2.5) resumes to

$$p(y|\theta) = \frac{f(y \mid \theta)\pi(\theta)}{\int_\Theta f(y \mid t)\pi(t)\,dt}. \tag{2.6}$$

2.5 Computational methods

2.5.1 Hamiltonian Monte Carlo

We follow (BETANCOURT, 2017). This method was developed in the late 1980s as Hybrid Monte Carlo to tackle calculations in Lattice Quantum Chromodynamics. Instead of moving in the parameter space randomly with uninformed jumps, the direction from the vector field given by the gradients are used to trace out a trajectory through the *typical set*, the region which has significant contribution to the expectations. However, if only the gradient was used, the trajectory would pull towards the mode of the distribution, so more geometric constraints are needed. In order to a satellite rotate around the Earth, we have to endow ir with enough momentum to counteract the gravitational field, turning the system into a conservative one.

First, we introduce auxiliary momentum parameters p_n (lift) of the same dimension from the parameter space $\Omega \subseteq \mathbb{R}^D$. Then q_n turns to (q_n, p_n), with the use the joint probability distribution $\pi(q, p) = \pi(p \mid q)\pi(q)$. Particularly, we use

$$\pi(q, p) = e^{-H(q,p)},$$

such that H is the *Hamiltonian*. Note that $H(q, p) = -\log \pi(p \mid q) - \log \pi(q) =: K(p, q) + V(q)$. We call K the kinetic energy, and V the potential energy. The vector field is generated by Hamilton's equations,

$$\frac{dq}{dt} = \frac{\partial H}{\partial p} = \frac{\partial K}{\partial p}$$
$$\frac{dp}{dt} = -\frac{\partial H}{\partial q} = -\frac{\partial K}{\partial q} - \frac{dV}{dq}.$$

Therefore, we are able to define the Hamiltonian flows $\phi_t : (p, q) \to (p, q), \forall t \in \mathbb{R}$.

2.5.1.1 Diagnostics

The importance of diagnosing. The potential problems that it can show.

- Divergent transitions;
- Transitions that hit the maximum tree depth;
- Low E-BFMI values;

- Low effective samples sizes;
- $\hat{R} \notin (0.95, 1.05)$.

3 Statistical modelling

Fisher (1922, p. 311) stated that the objective of statistics is to reduce the data since its volume is impossible to comprehend by the researchers. In that sense, few parameters should represent the whole phenomenon catching the most relevant information. Years later, Newman studied the theory of modelling which can be divided in three aspects (LEHMANN, 2012, p. 161):

1. Models of complex phenomena are created by putting together simple building elements that the researcher is familiar with and can handle;
2. There are two types of models: the *explanatory models*, which will be focused on this work, and the *interpolatory formulae*.
3. An explanatory theory necessitates a thorough understanding of the problem's scientific context. In this regard, we investigated this kind of problem involving Respondent-driven sampling and prevalence estimation as introduced in Chapter 2.

In this chapter, we develop models that enclose these ideas building each block separately. For a Bayesian modelling, we assume that each parameter of the model has a probability distribution that incorporates the researcher's uncertainty about it. For each individual, we observe k covariates that are possible risk factors represented by the vector $\boldsymbol{x}_i \in \mathbb{R}^k$ of the i^{th} individual. We denote θ_i the probability of the i-th individual have been exposed to the disease that depends on the prevalence θ and $\boldsymbol{x}_i$. We also consider when it depends on a spatial random effect caused by the connections analysed by the RDS. The probability of positive test in the i^{th} individual is denoted by p_i.

Another important feature of the model is that sensitivity and specificity have the same distribution for all individuals and it only depends on the test used to diagnose. This is an assumption that must be analysed for the studied disease. For instance, COVID-19 tests have different sensibilities and specificities for symptomatic and asymptomatic individuals.

From above, we develop three different models: the first considers perfect tests, that is, $\gamma_s = \gamma_e = 1$ and no spatial random effect; the second considers imperfect tests, regarding γ_s and γ_e, but ignoring the RDS structure; and the third one has imperfect tests and RDS structure.

3.1 Perfect tests

The first model supposes the samples are independent and the test is perfect, which means that $\theta_i = p_i$ for all i. Therefore it only considers the risk factors $\boldsymbol{x}_i$.

$$\begin{aligned} T_i &\sim \text{Bernoulli}(\theta_i), \\ g(\theta_i) &= g(\theta) + \boldsymbol{x}_i^T \beta, \end{aligned} \tag{3.1}$$

where v^T denotes the transpose of v, and $g(\cdot)$ is a link function. The parameter $\beta \in \mathbb{R}^k$ is the risk effects. For Bayesian inference, priors on β and θ must be included. We use $\beta \sim \text{Normal}(\mu, \Sigma)$ and $\theta \sim \text{Beta}(a^p, b^p)$, where $\mu \in \mathbb{R}^k$, $\Sigma \in \mathbb{R}^{k \times k}$ symmetric positive-definite matrix, $a^p \in \mathbb{R}_{++}$, and $b^p \in \mathbb{R}_{++}$ are fixed hyperparameters.

Remark. If the risk factors are zero, i.e $\boldsymbol{x}_i = 0$, the probability of the i^{th} having been exposed is the prevalence θ, which means that in a population with no risk effects, the probability of a person has the disease is exactly the proportion in this population.

3.1.1 Identifiability

3.1.2 Toy example

3.2 Sensitivity and specificity

In this section, we describe a model for estimating the sensitivity and specificity of a diagnostic test separately from the final model for estimating prevalence. This model is relevant to analyze and experiment with different prior specification approaches. Suppose having a *gold standard test* that is the best available test (VERSI, 1992), and also another test (for instance a simpler, a faster, or a less invasive one) which we want to estimate the accuracy by the sensitivity and specificity. In this scenario, a true positive / negative individual means they have been tested positive by the gold standard. Therefore, in a population with n_{γ_e} true positives and n_{γ_s} true negatives, we denote

$$\begin{aligned} y_{negative} &\sim \text{Binomial}(n_{\gamma_e}, \gamma_e), \\ y_{positive} &\sim \text{Binomial}(n_{\gamma_s}, \gamma_s), \end{aligned}$$

such that $y_{negative}$ are negative tests on known negative subjects and $y_{positive}$ are positive tests on known positive. In a classic Bayesian analysis, we have to define a prior distribution for the parameters $(\gamma_e, \gamma_s) \sim \pi(\gamma_e, \gamma_s)$.

For this, we consider three different approaches:

1. The parameters are independent a priori and each one has a beta distribution with pre-specified hyperparameters.

2. Hierarchical partial pooling, when dealing with more studies.

3. A Bivariate Beta (see Appendix A) distribution.

3.2.1 Independent beta distribution priors

When considering separated experiments for specificity and sensitivity, there is no information about their correlation, which is the case for our model. Then we define the the prior distributions

$$\begin{aligned}\gamma_e &\sim \text{Beta}(a_e, b_e),\\ \gamma_s &\sim \text{Beta}(a_s, b_s),\\ \theta &\sim \text{Beta}(a_\theta, b_\theta).\end{aligned}$$

Using data from (BENNETT; STEYVERS, 2020) about COVIDPrior information of these quantities lead to a bivariate analysis cite:t‘guo2017bayesian‘. As we have already mentioned, the definitions of *sensitivity* and *specificity* can be expressed as below: -19 seroprevalence in Santa Clara:

$$\begin{aligned}y/n &= 50/3330,\\ y_{negative}/n_{\gamma_e} &= 399/401,\\ y_{positive}/n_{\gamma_s} &= 103/122,\end{aligned}$$

we fit the model and obtain the results showed in Figure 2. All the codes were done in *Stan* and *PyStan*.

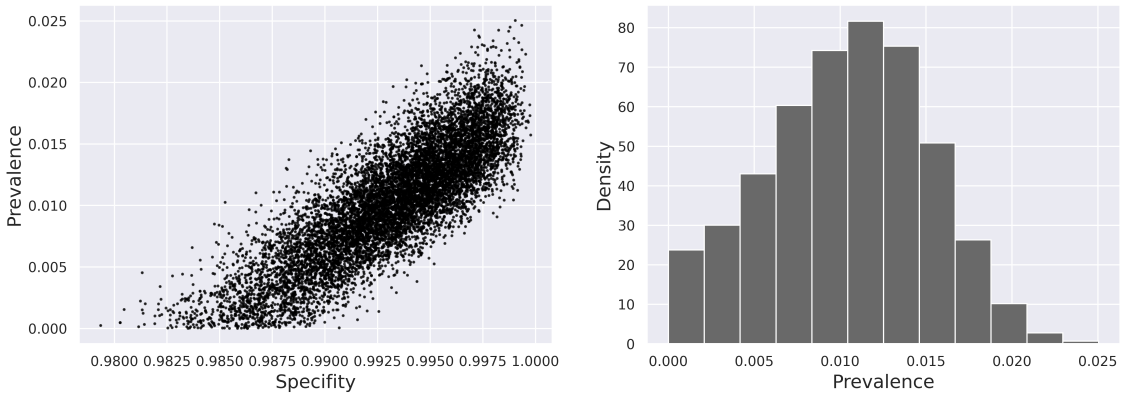


Figure 2 – Scatter plot of posterior simulations of prevalence against specificity and histogram of posterior simulations of the prevalence.

3.2.2 Hierarchical partial pooling prior

Other approach considers more than one study about specificity and sensitivity. A *hierarchical partial pooling* model for these studies can be done in the following way:

$$\begin{aligned}\text{logit}(\gamma_s^j) &\sim \text{Normal}(\mu_{\gamma_s}, \sigma_{\gamma_s}),\\ \text{logit}(\gamma_e^j) &\sim \text{Normal}(\mu_{\gamma_e}, \sigma_{\gamma_e}),\end{aligned}$$

for $1 \leq j \leq K$ studies, such that the first study is the considered one. Partial pooling because the parameters can be sampled from the same distribution. Hierarchical because the parameters of this distribution have its one prior distributions. For instance,

$$\begin{aligned}\mu_{\gamma_s} &\sim N(0, 10),\\ \mu_{\gamma_e} &\sim N(0, 10),\\ \sigma_{\gamma_s} &\sim N^+(0, 1), \text{ and}\\ \sigma_{\gamma_e} &\sim N^+(0, 1),\end{aligned}$$

where $N^+(a, b)$ is the truncated normal distribution in $[0, +\infty)$.

3.2.3 Bivariate Beta prior

Finally, we studied a joint distribution for specificity and sensitivity, a possible bivariate beta distribution built in (OLKIN; TRIKALINOS, 2015). This distribution is derived from a Dirichlet distribution of order four. Let $U = (U[1], ..., U[4]) \sim \text{Dirichlet}(\boldsymbol{\alpha})$, where $\boldsymbol{\alpha} \in \mathbb{R}_+^4$. Therefore, defining $X = U[1]+U[2]$ and $Y = U[1]+U[3]$, we will have that (X, Y) has a well-defined probability distribution in $[0, 1] \times [0, 1]$ such that X and Y have marginally beta distributions, and they have correlation in all space. Depending on the definition of $\boldsymbol{\alpha}$, the correlation between the variables range from -1 and 1. Figure 3 shows some examples of this construction.

In this section, we shall describe how to use the Bivariate Beta (see Appendix A) to model the correlation between specificity and sensitivity.

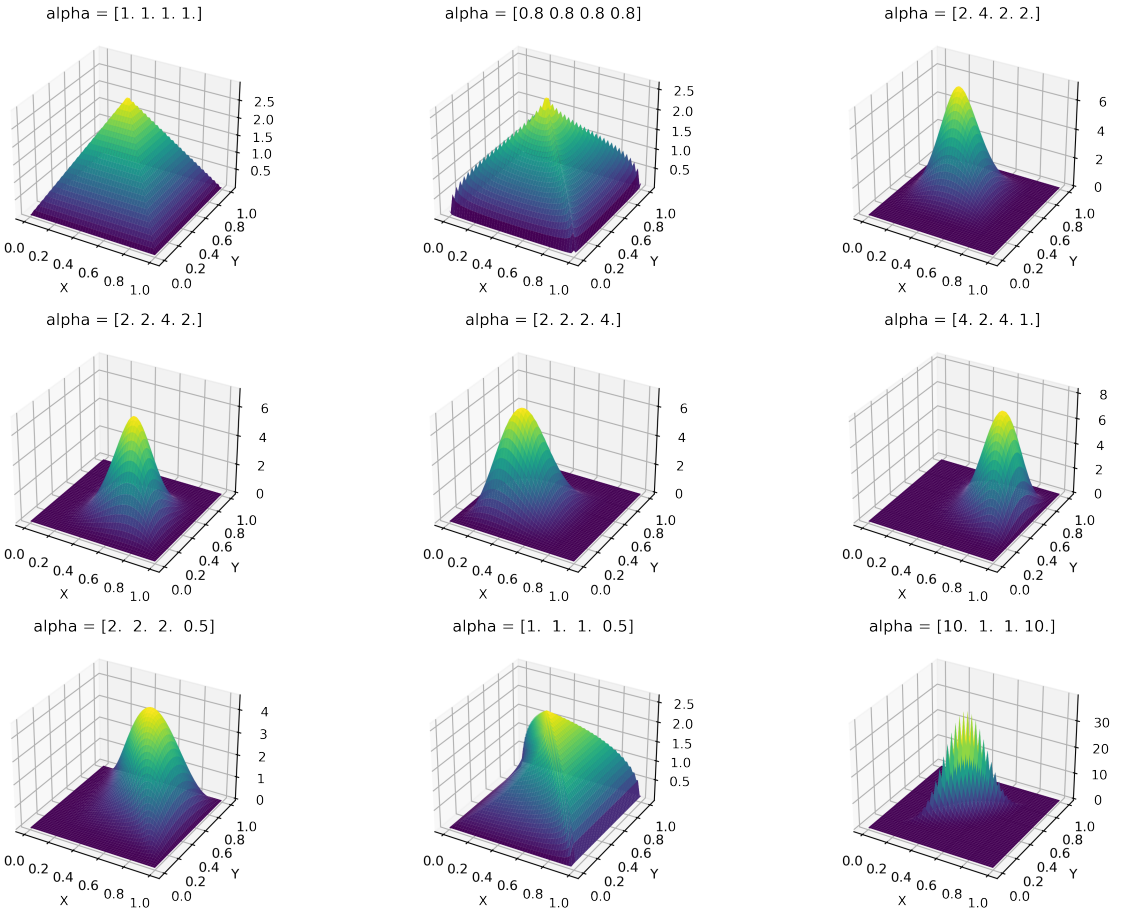


Figure 3 – Different choices of α and the joint distribution of the variables X and Y.

3.3 Imperfect tests

This model includes the sensitivity and specificity of the diagnostic test.

$$\begin{aligned} T_i &\sim \text{Bernoulli}(p_i) \\ p_i &= \gamma_s \theta_i + (1 - \gamma_e)(1 - \theta_i), \\ g(\theta_i) &= g(\theta) + \boldsymbol{x}_i^T \beta, \\ \beta &\sim \text{Normal}(\mu, \Sigma), \\ \theta &\sim \text{Beta}(a^p, b^p) \\ \gamma_s &\sim \text{Beta}(a^s, b^s), \\ \gamma_e &\sim \text{Beta}(a^e, b^e), \end{aligned} \tag{3.2}$$

where $a^p, a^s, a^e, b^p, b^s, b^e \in \mathbb{R}_{++}$ are fixed hyperparameters. This model does not include prior knowledge about the correlation between specificity and sensitivity.

3.3.1 Toy example

Consider the following model (GELMAN; CARPENTER, 2020):

$$\begin{aligned} y &\sim \text{Binomial}(n, p), \\ p &= \theta\gamma_s + (1-\theta)(1-\gamma_e), \end{aligned}$$

such that y is the number of positive tests in a population of size n. In a Bayesian paradigm, a prior $\pi(\theta, \gamma_e, \gamma_s)$ must be specified. For instance, $\pi(\theta, \gamma_e, \gamma_s) = \pi(\theta)\pi(\gamma_e, \gamma_s)$ and $\theta \sim \text{Beta}(\alpha_\theta, \beta_\theta)$, in which α_θ and β_θ are positive hyperparameters. Since the three parameters θ, γ_e, and γ_s are not jointly identifiable only from y, prior information on γ_e and γ_s need be added.

3.4 Imperfect tests and respondent-driven sampling

For now, we consider the network dependence induced by the RDS with no associated model. Therefore, we treat it as a random effect for each individual. Conditionally autoregressive (CAR) models in the Gaussian case are used. Let $[\tilde{Q}]_{ij} = \tilde{q}_{ij}$ be a fixed matrix which measures the distance between i and j, and $\tilde{q}_{i+} = \sum_j \tilde{q}_{ij}$. In general, we use

$$\tilde{q}_{ij} = \begin{cases} 1, & \text{if } i \text{ recruited } j \text{ or the contrary} \\ 0, & \text{otherwise.} \end{cases}$$

Next we define the scaled adjacency matrix $Q = D^{-1}\tilde{Q}$, such that D is a diagonal matrix with $D_{ii} = \tilde{q}_{i+}$. Finally let $|\rho| < 1$ be a parameter to controls the dependence between neighbors. Hence, we specify the model as follows:

$$\begin{aligned} T_i &\sim \text{Bernoulli}(p_i) \\ p_i &= \gamma_s\theta_i + (1-\gamma_e)(1-\theta_i), \\ g(\theta_i) &= g(\theta) + \boldsymbol{x}_i^T\beta + \omega_i, \\ \omega_i|\{\omega_j\}_{j\neq i}, \tau &\sim \text{Normal}\left(\rho\sum_j q_{ij}\omega_j, \tau^{-1}/\tilde{q}_{i+}\right) \\ \beta &\sim \text{Normal}(\mu, \Sigma), \\ \theta &\sim \text{Beta}(a^p, b^p) \\ \gamma_s &\sim \text{Beta}(a^s, b^s), \\ \gamma_e &\sim \text{Beta}(a^e, b^e), \\ \tau &\sim \text{Gamma}(a^\tau, b^\tau). \end{aligned} \tag{3.3}$$

By Brook's Lemma (BROOK, 1964), the joint distribution of ω can be specified as

$$\omega \sim \text{Normal}\left(0, \left[\tau(D - \rho\tilde{Q})\right]^{-1}\right).$$

3.4.1 Toy example

1. Between the model with the log odds of prevalence having a Gaussian prior distribution and the other with the prevalence having a Beta prior distribution, the latter was usually faster and without divergences. Therefore the preferable model is with the prevalence.

2. Non-centered distributions are really worst.

3. Comparison between parametrization of sigma and tau showed that they are similar in sight of time of execution, energy and divergences, among others diagnostics. However, the mean estimate of sigma is more controlled. The median estimate is very similar. This happens because there are a few very high samples for τ that will have high weight in the final result. Small samples for σ have less impact, despite having some.

4. More sparse matrices (RDS data is very sparse) is generating the funil we do not want to see. This is not connected to the number of connected components. In order to see that, a simple example with the Erdos-Renyi Random Graph can answer to us. In the sparse case, the number of edges is $O(n)$ with $p = 1/n$. If $p = 1$, the number of edges is $O(n^2)$ and the funil disappears. This problem does not appear in the poisson model.

3.4.2 Exponential Random Graph Model (ERGM)

RDS has the constraint of being without replacement. For that reason, we do not observe all links among the samples (CRAWFORD, 2016). Considering the model developed by Crawford, we can model the matrix Q as *Exponential Random Graph Model* (ERGM). Define the following

1. $\boldsymbol{s} = \text{tril}(QC)^T\mathbf{1} + C^T u$, such that Q is the adjacency matrix of the recruited subjects, C is the *Coupon Matrix*, u the vector of the number of edge ends belonging to each vertex (in the order of recruitment) that are not connected to any other sampled vertex, and $\text{tril}(M)$ the lower triangle of M.

2. $T(Q) = -\lambda\boldsymbol{s}$, such that λ is the rate of the recruitment time.

3. $V(Q) = \sum_{k \text{ is not seed}} \log(\lambda \boldsymbol{s}_k)$

4. $w = (0, t_2 - t_1, ..., t_n - t_{n-1})$ is the vector of the waiting times between recruitments.

Therefore $\Pr(Q|w) \propto \exp[T(Q)^T w + V(Q)]$. With that, the model becomes

$$\begin{aligned}
T_i &\sim \text{Bernoulli}(p_i) \\
p_i &= \gamma_s \theta_i + (1 - \gamma_e)(1 - \theta_i), \\
g(\theta_i) &= g(\theta) + \boldsymbol{x}_i^T \beta + \omega_i, \\
\omega_i | \{\omega_j\}_{j \neq i}, \tau &\sim \text{Normal}\left(\rho \sum_j q_{ij} \omega_j / q_{i+}, \tau^2 / q_{i+}\right) \\
Q|w &\propto \exp[T(Q)^T w + V(Q)] \\
\lambda &\sim \Gamma(a^\lambda, b^\lambda), \\
\beta &\sim \text{Normal}(\mu, \Sigma), \\
\theta &\sim \text{Beta}(a^p, b^p) \\
\gamma_s &\sim \text{Beta}(a^s, b^s), \\
\gamma_e &\sim \text{Beta}(a^e, b^e), \\
\tau &\sim \text{Normal}^+(0, \sigma_\tau^2).
\end{aligned} \tag{3.4}$$

The problem with this model is that we are assigning a posterior distribution for Q.

4 Discussion about prior distributions and sensitivity analysis

4.1 Prior analysis of sensitivity and specificity

4.2 Prior analysis on the parameter tau

4.3 Prior analysis on theta

5 Real data applications

6 Conclusion

Parte final do trabalho, apresenta as conclusões correspondentes aos objetivos ou hipóteses.

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Appendix

APPENDIX A – Bivariate Beta distribution

Let $U = (U_1, U_2, U_3, U_4) \sim \text{Dirichlet}(\boldsymbol{\alpha})$, where $\boldsymbol{\alpha} = (\alpha_1, \alpha_2, \alpha_3, \alpha_4)$ with $\alpha_i > 0, i = 1, \ldots, 4$ and $U_4 = 1 - U_1 + U_2 + U_3$. The joint density of U with respect to the Lebesgue measure is given by

$$f_U(u_1, u_2, u_3) = \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} u_3^{\alpha_3 - 1} (1 - u_1 - u_2 - u_3)^{\alpha_4 - 1}, \tag{A.1}$$

when $u_i \in [0, 1], i = 1, 2, 3$, $u_1 + u_2 + u_3 \leq 1$, and 0 otherwise. The normalizing constant is, for $v \in \mathbb{R}^n$,

$$B(v) = \frac{\prod_{i=1}^n \Gamma(v_i)}{\Gamma\left(\sum_{i=1}^n v_i\right)}.$$

Definition A.0.1. Let

$$X = U_1 + U_2 \text{ and } Y = U_1 + U_3. \tag{A.2}$$

The distribution of (X, Y) is *Bivariate Beta* with parameters $\boldsymbol{\alpha}$.

Proposition 1. *The marginal distribution of X is Beta with parameters $\alpha_1 + \alpha_2$ and $\alpha_3 + \alpha_4$. Similarly, the marginal distribution of Y is Beta with parameters $\alpha_1 + \alpha_3$ and $\alpha_2 + \alpha_4$.*

Proof. First we derive the probability density of (U_1, U_2) with respect to the Lebesgue measure.

$$\begin{aligned} f_{U_1,U_2}(u_1, u_2) &= \int_{-\infty}^{\infty} f_U(u_1, u_2, u_3)\, du_3 \\ &= \frac{1}{B(\boldsymbol{\alpha})} \int_0^1 u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} u_3^{\alpha_3 - 1} (1 - u_1 - u_2 - u_3)^{\alpha_4 - 1}\, du_3 \\ &= \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} \int_0^1 u_3^{\alpha_3 - 1} (1 - u_1 - u_2 - u_3)^{\alpha_4 - 1}\, du_3. \end{aligned} \tag{A.3}$$

Let $u_3 = (1 - u_1 - u_2)z$. Then,

$$\begin{aligned} f_{U_1,U_2}(u_1, u_2) &= \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} \int_0^1 (1 - u_1 - u_2)^{\alpha_3 - 1} z^{\alpha_3 - 1} (1 - u_1 - u_2)^{\alpha_4} (1 - z)^{\alpha_4 - 1}\, dz. \\ &= \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} (1 - u_1 - u_2)^{\alpha_3 + \alpha_4 - 1} \int_0^1 z^{\alpha_3 - 1} (1 - z)^{\alpha_4 - 1}\, dz. \\ &= \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} (1 - u_1 - u_2)^{\alpha_3 + \alpha_4 - 1} \frac{\Gamma(\alpha_3)\Gamma(\alpha_4)}{\Gamma(\alpha_3 + \alpha_4)} \\ &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} u_1^{\alpha_1 - 1} u_2^{\alpha_2 - 1} (1 - u_1 - u_2)^{\alpha_3 + \alpha_4 - 1}. \end{aligned} \tag{A.4}$$

We conclude that

$$(U_1, U_2, 1 - U_1 - U_2) \sim \text{Dirichlet}(\alpha_1, \alpha_2, \alpha_3 + \alpha_4).$$

Define

$$H(v) = \begin{bmatrix} 1 & 0 \\ 1 & 1 \end{bmatrix} v, \text{ for } v \in \mathbb{R}^2.$$

Then $(U_1, X) = H(U_1, U_2)$ and $H(\cdot)$ is bijective and differentiable function. By the Change of Variable Formula,

$$\begin{aligned} f_{U_1,X}(u_1, x) &= f(H^{-1}(u_1, x)) \left| \det \left[\frac{dH^{-1}(v)}{dv} \Big|_{v=(u_1,x)} \right] \right| \\ &= f(u_1, x - u_1) = \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} u_1^{\alpha_1 - 1} (x - u_1)^{\alpha_2 - 1} (1 - x)^{\alpha_3 + \alpha_4 - 1}, \end{aligned} \tag{A.5}$$

where (u_1, x) belongs to the triangle defined by the points (0,0), (0,1), and (1,1). The distribution of X for $x \in [0, 1]$ is

$$\begin{aligned} f_X(x) &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} \int_0^x u_1^{\alpha_1 - 1} (x - u_1)^{\alpha_2 - 1} (1 - x)^{\alpha_3 + \alpha_4 - 1} \, du_1 \\ &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} (1 - x)^{\alpha_3 + \alpha_4 - 1} \int_0^x u_1^{\alpha_1 - 1} (x - u_1)^{\alpha_2 - 1} \, du_1. \\ &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} (1 - x)^{\alpha_3 + \alpha_4 - 1} \int_0^x x^{\alpha_1 - 1} \left(\frac{u_1}{x} \right)^{\alpha_1 - 1} x^{\alpha_2 - 1} \left(1 - \frac{u_1}{x} \right)^{\alpha_2 - 1} \, du_1. \end{aligned} \tag{A.6}$$

Setting $u = u_1/x$ (if $x = 0, f_X(x) = 0$, then suppose $x > 0$), we have,

$$\begin{aligned} f_X(x) &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} (1 - x)^{\alpha_3 + \alpha_4 - 1} x^{\alpha_1 + \alpha_2 - 1} \int_0^1 u^{\alpha_1 - 1} (1 - u)^{\alpha_2 - 1} \, du. \\ &= \frac{1}{B(\alpha_1, \alpha_2, \alpha_3 + \alpha_4)} (1 - x)^{\alpha_3 + \alpha_4 - 1} x^{\alpha_1 + \alpha_2 - 1} B(\alpha_1, \alpha_2) \\ &= \frac{1}{B(\alpha_1 + \alpha_2, \alpha_3 + \alpha_4)} (1 - x)^{\alpha_3 + \alpha_4 - 1} x^{\alpha_1 + \alpha_2 - 1} \end{aligned} \tag{A.7}$$

Therefore $X \sim \text{Beta}(\alpha_1 + \alpha_2, \alpha_3 + \alpha_4)$. Similarly $Y \sim \text{Beta}(\alpha_1 + \alpha_3, \alpha_2 + \alpha_4)$.

□

Proposition 2. *The joint density of (X, Y) with respect to the Lebesgue measure is given by*

$$f_{X,Y}(x, y) = \frac{1}{B(\boldsymbol{\alpha})} \int_\Omega u_1^{\alpha_1 - 1} (x - u_1)^{\alpha_2 - 1} (y - u_1)^{\alpha_3 - 1} (1 - x - y + u_1)^{\alpha_4 - 1} \, du_1, \tag{A.8}$$

where

$$\Omega = (\max(0, x + y - 1), \min(x, y)).$$

Proof. Note that

$$\begin{bmatrix} U_1 \\ X \\ Y \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 \\ 1 & 1 & 0 \\ 1 & 0 & 1 \end{bmatrix} \begin{bmatrix} U_1 \\ U_2 \\ U_3 \end{bmatrix},$$

where the linear function is bijective and differentiable function, such that the determinant of the derivative is 1. By the Change of Variable Formula,

$$\begin{aligned} f_{U_1,X,Y}(u_1,x,y) &= f_{U_1,U_2,U_3}(u_1,x-u_1,y-u_2) \\ &= \frac{1}{B(\boldsymbol{\alpha})} u_1^{\alpha_1-1}(x-u_1)^{\alpha_2-1}(y-u_1)^{\alpha_3-1}(1-x-y+u_1)^{\alpha_4-1}, \end{aligned} \tag{A.9}$$

where $0 \leq u_1 \leq x, u_1 \leq y$, and $0 \leq 1-x-y+u_1$. Hence,

$$f_{X,Y}(x,y) = \frac{1}{B(\boldsymbol{\alpha})} \int_\Omega u_1^{\alpha_1-1}(x-u_1)^{\alpha_2-1}(y-u_1)^{\alpha_3-1}(1-x-y+u_1)^{\alpha_4-1}\, du_1, \tag{A.10}$$

such that $\Omega = \{u_1 : \max(0, x+y-1) < u_1 < \min(x,y)\}$. □

Proposition 3. *The covariance between X and Y is*

$$\mathrm{Cov}(X,Y) = \frac{1}{\tilde{\alpha}^2(\tilde{\alpha}+1)}(\alpha_1\alpha_4 - \alpha_2\alpha_3).$$

Proof. Let $\tilde{a} = \sum_i \alpha_i$. The covariance between U_i and U_j is (LIN, 2016)

$$\mathrm{Cov}(U_i,U_j) = -\frac{\alpha_i\alpha_j}{\tilde{\alpha}^2(\tilde{\alpha}+1)}, i,j = 1,...,4, i \neq j \tag{A.11}$$

and the variance of U_i is

$$\mathrm{Var}(U_i) = \frac{\alpha_i(\tilde{\alpha}-\alpha_i)}{\tilde{\alpha}^2(\tilde{\alpha}+1)}, \tag{A.12}$$

since $U_i \sim \mathrm{Beta}(\alpha_i, \tilde{\alpha}-\alpha_i)$. Therefore

$$\mathrm{Cov}(X,Y) = \mathrm{Cov}(U_1+U_2, U_1+U_3) = \frac{1}{\tilde{\alpha}^2(\tilde{\alpha}+1)}(\alpha_1\alpha_4 - \alpha_2\alpha_3) \tag{A.13}$$

□

The main moments of X and Y are the following

$$\begin{aligned} \mathbb{E}(X) &= \mathbb{E}(U_1+U_2) = \frac{\alpha_1+\alpha_2}{\alpha_1+\alpha_2+\alpha_3+\alpha_4} \\ \mathbb{E}(Y) &= \mathbb{E}(U_1+U_3) = \frac{\alpha_1+\alpha_3}{\alpha_1+\alpha_2+\alpha_3+\alpha_4} \\ \mathrm{Var}(X) &= \mathrm{Cov}(U_1+U_2, U_1+U_2) = \frac{1}{\tilde{\alpha}^2(\tilde{\alpha}+1)}(\alpha_1+\alpha_2)(\alpha_3+\alpha_4) \\ \mathrm{Var}(Y) &= \mathrm{Cov}(U_1+U_3, U_1+U_3) = \frac{1}{\tilde{\alpha}^2(\tilde{\alpha}+1)}(\alpha_1+\alpha_3)(\alpha_2+\alpha_4) \\ \mathrm{Cor}(X,Y) &= \frac{\mathrm{Cov}(X,Y)}{\sqrt{\mathrm{Var}(X)\,\mathrm{Var}(Y)}} = \frac{\alpha_1\alpha_4-\alpha_2\alpha_3}{\sqrt{(\alpha_1+\alpha_2)(\alpha_3+\alpha_4)(\alpha_1+\alpha_3)(\alpha_2+\alpha_4)}} \end{aligned}$$

Remark. The original paper has a mistake at page 6.

A.1 Comments about integration

The density of (X, Y) with respect to the Lebesgue measure is $f_{X,Y}(x, y)$ as in equation (A.10). Therefore it can be undefined in sets of null Lebesgue measure in $\mathbb{R}^2$. This section aims to find them to help writing the function properly. If $\alpha_i \geq 1$, $i = 1, ..., 4$, the integral is clearly well defined for every $x, y \in [0, 1]$. Let $0 < \alpha_2 = \alpha_3 = a \leq 0.5$ and $x = y < 0.5$. Then

$$\begin{aligned} f_{X,Y}(x,y) &= \frac{1}{B(\boldsymbol{\alpha})} \int_0^x u_1^{\alpha_1-1}(x-u_1)^{a-1}(x-u_1)^{a-1}(1-2x+u_1)^{\alpha_4-1}\,du_1 \\ &= \frac{1}{B(\boldsymbol{\alpha})} \int_0^{x/2} u_1^{\alpha_1-1}(x-u_1)^{2a-2}(1-2x+u_1)^{\alpha_4-1}\,du_1 + \\ &\quad + \frac{1}{B(\boldsymbol{\alpha})} \int_{x/2}^x u_1^{\alpha_1-1}(x-u_1)^{2a-2}(1-2x+u_1)^{\alpha_4-1}\,du_1 \end{aligned}$$

Note that the first integral is well defined and non-negative. If $\alpha_1 \geq 1$,

$$\begin{aligned} &\int_0^{x/2} u_1^{\alpha_1-1}(x-u_1)^{2a-2}(1-2x+u_1)^{\alpha_4-1}\,du_1 \\ &\qquad \leq \int_0^{x/2} \frac{x}{2}^{\alpha_1-1} \left(\frac{x}{2}\right)^{2a-2} \max\left(\left(1-\frac{3}{2}x\right)^{\alpha_4-1}, (1-2x)^{\alpha_4-1}\right) du_1 < +\infty. \end{aligned}$$

If $0 < \alpha_1 < 1$,

$$\begin{aligned} &\int_0^{x/2} u_1^{\alpha_1-1}(x-u_1)^{2a-2}(1-2x+u_1)^{\alpha_4-1}\,du_1 \\ &\qquad = \lim_{t\to 0^+} \int_t^{x/2} u_1^{\alpha_1-1} \left(\frac{x}{2}\right)^{2a-2} \max\left(\left(1-\frac{3}{2}x\right)^{\alpha_4-1}, (1-2x)^{\alpha_4-1}\right) du_1 \\ &\qquad = K(x) \lim_{t\to 0^+} \int_t^{x/2} u_1^{\alpha_1-1}\,du_1 \\ &\qquad = \frac{K(x)}{\alpha_1} \lim_{t\to 0^+} \left[\left(\frac{x}{2}\right)^{\alpha_1} - t^{\alpha_1}\right] < +\infty. \end{aligned}$$

where $K(x)$ is a function of x. Moreover, since the integrand is non-negative, so is the integral. On the other hand, the second integral is not defined:

$$\begin{aligned} &\int_{x/2}^x u_1^{\alpha_1-1}(x-u_1)^{2a-2}(1-2x+u_1)^{\alpha_4-1}\,du_1 \\ &\qquad \geq \int_{x/2}^x \min\left(\left(\frac{x}{2}\right)^{\alpha_1-1}, x^{\alpha_1-1}\right)(x-u_1)^{2a-2} \min\left(\left(1-\frac{3}{2}x\right)^{\alpha_4-1}, (1-x)^{\alpha_4-1}\right) du_1 \\ &\qquad = K'(x) \int_0^{x/2} v^{2a-2}\,dv \\ &\qquad = \begin{cases} \dfrac{K'(x)}{2a-1} \lim_{t\to 0^+} \left[(x/2)^{2a-1} - t^{2a-1}\right] & \text{if } a < 0.5 \\ K'(x) \lim_{t\to 0^+} \left[\log(x/2) - \log(t)\right] & \text{if } a = 0.5 \end{cases} \\ &\qquad \to +\infty. \end{aligned}$$

Based on this divergence, we conclude that if $0 < \alpha_2 = \alpha_3 \leq 0.5$ and $x = y < 0.5$, $f_{X,Y}(x, y)$ is not defined. Note that if $x = y \geq 0.5$, divergence problems still happens, since the problems appear when u_1 converges to x. Similar calculations show that if $x + y = 1$ and $0 < \alpha_1 = \alpha_4 \leq 0.5$, the density is also not defined. More generally, $f_{X,Y}(x, y)$ is not defined if

1. $\alpha_1 + \alpha_4 \leq 1$ and $x + y = 1$.
2. $\alpha_2 + \alpha_3 \leq 1$ and $x = y$.

A.2 Specifying parameters $\boldsymbol{\alpha}$

Suppose that the researcher has knowledge about the main moments of X and Y, such that $\mathbb{E}(X) = m_1 \in (0, 1), \mathbb{E}(Y) = m_2 \in (0, 1), \text{Var}(X) = v_1 \in (0, 1)$, and $\text{Var}(Y) = v_2 \in (0, 1)$. Notice that $v_1 + m_1^2 = \text{Var}(X_1) + \mathbb{E}[X_1]^2 = \mathbb{E}[X_1^2]$ and

$$\mathbb{E}[X_1^2] - \mathbb{E}[X_1] = \frac{(\alpha_1 + \alpha_2 + 1)(\alpha_1 + \alpha_2)}{(\tilde{\alpha} + 1)\tilde{\alpha}} - \frac{\alpha_1 + \alpha_2}{\tilde{\alpha}} = -\frac{(\alpha_1 + \alpha_2)(\alpha_3 + \alpha_4)}{\tilde{\alpha}(\tilde{\alpha} + 1)} < 0,$$

that is, $v_1 + m_1^2 - m_1 < 0 \implies v_1 < m_1 - m_1^2$ and similarly, $v_2 < m_2 - m_2^2$. After fixing these quantities, we will have a non-linear system with four equations and four unknown variables. Hence, we want to solve the following

$$\begin{cases} m_1 = \dfrac{\alpha_1 + \alpha_2}{\tilde{\alpha}} \\ m_2 = \dfrac{\alpha_1 + \alpha_3}{\tilde{\alpha}} \\ v_1 = \dfrac{(\alpha_1 + \alpha_2)(\alpha_3 + \alpha_4)}{\tilde{\alpha}^2(\tilde{\alpha} + 1)} = m_1 \dfrac{\alpha_3 + \alpha_4}{\tilde{\alpha}(\tilde{\alpha} + 1)} \\ v_2 = \dfrac{(\alpha_1 + \alpha_3)(\alpha_2 + \alpha_4)}{\tilde{\alpha}^2(\tilde{\alpha} + 1)} = m_2 \dfrac{\alpha_2 + \alpha_4}{\tilde{\alpha}(\tilde{\alpha} + 1)}. \end{cases} \tag{A.14}$$

Proposition 4. *System (A.14) has a solution if, and only if, the relation*

$$v_2 = \frac{(1 - m_2)\tilde{\alpha}}{\tilde{\alpha}(\tilde{\alpha} + 1)} = \frac{1 - m_2}{\frac{m_1 - m_1^2}{v_1}} = \frac{v_1(1 - m_2)}{m_1(1 - m_1)}, \tag{A.15}$$

is satisfied. When there is a solution, there will be infinitely many and they all lay in the ray

$$\mathcal{L} = \{(1, -1, -1, 1)\alpha_4 + k : \alpha_4 > 0\},$$

such that $k = ((m_1 + m_2 - 1)\tilde{\alpha}, (1 - m_2)\tilde{\alpha}, (1 - m_1)\tilde{\alpha}, 0)$.

Proof. The first two equations of the system (A.14) can be rewritten as a linear system:

$$\begin{aligned}(m_1-1)\alpha_1+(m_1-1)\alpha_2+m_1\alpha_3+m_1\alpha_4=0\\(m_2-1)\alpha_1+m_2\alpha_2+(m_2-1)\alpha_3+m_2\alpha_4=0,\end{aligned}$$

which is equivalent to

$$\begin{aligned}\alpha_1+\alpha_2+\frac{m_1}{m_1-1}\alpha_3+\frac{m_1}{m_1-1}\alpha_4=0\\\alpha_2+\frac{1-m_2}{m_1-1}\alpha_3+\frac{m_1-m_2}{m_1-1}\alpha_4=0.\end{aligned}$$

Then, we can write α_1 and α_2 as functions of α_3 and α_4:

$$\alpha_1=\frac{m_1+m_2-1}{1-m_1}\alpha_3+\frac{m_2}{1-m_1}\alpha_4 \tag{A.16}$$

$$\alpha_2=\frac{1-m_2}{1-m_1}\alpha_3+\frac{m_1-m_2}{1-m_1}\alpha_4. \tag{A.17}$$

With that expression, let $\alpha_1=a_3\alpha_3+a_4\alpha_4$ and $\alpha_2=b_3\alpha_3+b_4\alpha_4$. Denote $c_3=a_3+b_3+1$ and $c_4=a_4+b_4+1$. Then, consider the third equation of the system (A.14),

$$\begin{aligned}&\frac{v_1}{m_1}=\frac{\alpha_3+\alpha_4}{\tilde{\alpha}(\tilde{\alpha}+1)}=\frac{\alpha_3+\alpha_4}{(\alpha_1+\alpha_2+\alpha_3+\alpha_4)^2+(\alpha_1+\alpha_2+\alpha_3+\alpha_4)}\\
&\implies \frac{v_1}{m_1}(\alpha_1+\alpha_2+\alpha_3+\alpha_4)^2=\alpha_3+\alpha_4-\frac{v_1}{m_1}(\alpha_1+\alpha_2+\alpha_3+\alpha_4)\\
&\implies \frac{v_1}{m_1}(c_3\alpha_3+c_4\alpha_4)^2=\left(1-\frac{v_1}{m_1}c_3\right)\alpha_3+\left(1-\frac{v_1}{m_1}c_4\right)\alpha_4\\
&\implies \frac{v_1c_3^2}{m_1}\alpha_3^2+\left(\frac{2v_1c_3c_4\alpha_4+v_1c_3}{m_1}-1\right)\alpha_3+\left(\frac{v_1c_4^2\alpha_4^2+v_1c_4\alpha_4}{m_1}-\alpha_4\right)=0\\
&\implies v_1c_3^2\alpha_3^2+(2v_1c_3c_4\alpha_4+v_1c_3-m_1)\alpha_3+(v_1c_4^2\alpha_4^2+v_1c_4\alpha_4-m_1\alpha_4)=0.\end{aligned}$$

Using a Computer Algebra System (CAS) with the Python library SymPy, the above expression can be simplified as follows:

$$v_1\alpha_3^2+\left(v_1(1-m_1)+2v_1\alpha_4-m_1(1-m_1)^2\right)\alpha_3-\alpha_4m_1(1-m_1)^2+\alpha_4v_1(1-m_1)+v_1\alpha_4^2=0.$$

This way, the solutions of the above equation are function of α_4. Therefore, after solving the equations, we can use the last equation of the system (A.14) as a function on of α_4. Let,

$$\Lambda=\left(v_1(1-m_1)+v_1\alpha_4-m_1(1-m_1)^2\right).$$

Then,

$$\begin{aligned}\Delta &= \Big(v_1(1-m_1) + 2v_1\alpha_4 - m_1(1-m_1)^2\Big)^2 - 4v_1(\alpha_4 v_1(1-m_1) - \alpha_4 m_1(1-m_1)^2 + v_1\alpha_4^2),\\ &= (\Lambda + v_1\alpha_4)^2 - 4v_1\alpha_4\Lambda\\ &= \Lambda^2 - 2\Lambda v_1\alpha_4 + (v_1\alpha_4)^2\\ &= (\Lambda - v_1\alpha_4)^2\\ &= \Big(v_1(1-m_1) - m_1(1-m_1)^2\Big)^2\\ &= (1-m_1)^2(v_1 + m_1^2 - m_1)^2.\end{aligned}$$

Note that $v_1 + m_1^2 = \mathrm{Var}(X_1) + \mathbb{E}[X_1]^2 = \mathbb{E}[X_1^2]$ and

$$\mathbb{E}[X_1^2] - \mathbb{E}[X_1] = \frac{(\alpha_1+\alpha_2+1)(\alpha_1+\alpha_2)}{(\tilde{\alpha}+1)\tilde{\alpha}} - \frac{\alpha_1+\alpha_2}{\tilde{\alpha}} = -\frac{(\alpha_1+\alpha_2)(\alpha_3+\alpha_4)}{\tilde{\alpha}(\tilde{\alpha}+1)} < 0.$$

Therefore,

$$\sqrt{\Delta} = (1-m_1)(m_1 - v_1 - m_1^2)$$

and

$$\begin{aligned}\alpha_3 &= \frac{1}{2v_1}\Big(\Big(m_1(1-m_1)^2 - v_1(1-m_1) - 2v_1\alpha_4\Big) \pm (1-m_1)(m_1 - v_1 - m_1^2)\Big)\\ &= -\alpha_4 + \frac{(1-m_1)(m_1 - m_1^2 - v_1) \pm (1-m_1)(m_1 - v_1 - m_1^2)}{2v_1}.\end{aligned}$$

When the sign is negative, we have that $\alpha_3 = -\alpha_4$, an impossible solution. Then,

$$\alpha_3 = \frac{(1-m_1)(m_1 - m_1^2 - v_1)}{v_1} - \alpha_4.$$

We summarize the expressions in function of α_4:

$$\begin{aligned}\alpha_3 &= \frac{(1-m_1)(m_1 - m_1^2 - v_1)}{v_1} - \alpha_4\\ \alpha_1 &= \frac{m_1+m_2-1}{1-m_1}\alpha_3 + \frac{m_2}{1-m_1}\alpha_4 = \frac{(m_1+m_2-1)(m_1 - m_1^2 - v_1)}{v_1} + \alpha_4\\ \alpha_2 &= \frac{1-m_2}{1-m_1}\alpha_3 + \frac{m_1-m_2}{1-m_1}\alpha_4 = \frac{(1-m_2)(m_1 - m_1^2 - v_1)}{v_1} - \alpha_4.\end{aligned}$$

From here, one can calculate that

$$\tilde{\alpha} = \frac{m_1 - m_1^2 - v_1}{v_1}.$$

Since $\alpha_2 + \alpha_4 = (1-m_2)\tilde{\alpha}$, we have that the last equation of the system (A.14) is given by (A.15), that is, the system (A.14) has a solution if and only if, equation (A.15) is satisfied. If it is, the solution is the ray

$$\mathcal{L} = \{(1,-1,-1,1)\alpha_4 + k : \alpha_4 > 0\},$$

such that $k = ((m_1 + m_2 - 1)\tilde{\alpha}, (1 - m_2)\tilde{\alpha}, (1 - m_1)\tilde{\alpha}, 0)$.

□

Now change the fourth equation of (A.14) by:

$$\mathrm{Cor}(X, Y) = \frac{\alpha_1\alpha_4 - \alpha_2\alpha_3}{\sqrt{(\alpha_1 + \alpha_2)(\alpha_3 + \alpha_4)(\alpha_1 + \alpha_3)(\alpha_2 + \alpha_4)}} = \frac{\alpha_1\alpha_4 - \alpha_2\alpha_3}{\tilde{\alpha}^2\sqrt{m_1m_2(1 - m_1)(1 - m_2)}}$$

Supposing the expression for α_1, α_2 and α_3, that is, m_1, m_2 and v_1 are fixed, and supposing we fix $\rho = \mathrm{Cor}(X, Y)$, we can simplify the above expression (using a software) as follows:

$$\rho = \frac{1}{\tilde{\alpha}\sqrt{m_1m_2(1 - m_1)(1 - m_2)}}\alpha_4 - \sqrt{\frac{(1 - m_1)(1 - m_2)}{m_1m_2}},$$

which is linear on α_4, that is, for fixed values of m_1, m_2, v_1 and ρ, there is an unique α_4, and hence, α_1, α_2 and α_3 that satisfies system (A.14) with the fourth equation changed by the correlation.