Universal features of surname distribution in a subsample of a growing population

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Abstract
We examine the problem of family size statistics (the number of individuals carrying the same surname, or the same DNA sequence) in a given size subsample of an exponentially growing population. We approach the problem from two directions. In the first, we construct the family size distribution for the subsample from the stable distribution for the full population. This latter distribution is calculated for an arbitrary growth process in the limit of slow growth, and is seen to depend only on the average and variance of the number of children per individual, as well as the mutation rate. The distribution for the subsample is shifted left with respect to the original distribution, tending to eliminate the part of the original distribution reflecting the small families, and thus increasing the mean family size. From the subsample distribution, various bulk quantities such as the average family size and the percentage of singleton families are calculated. In the second approach, we study the past time development of these bulk quantities, deriving the statistics of the genealogical tree of the subsample. This approach reproduces that of the first when the current statistics of the subsample is considered. The surname distribution from the 2000 U.S. Census is examined in light of these findings, and found to misrepresent the population growth rate by a factor of 1000.

Key words: family size, growing population, coalescent, distribution

1. Introduction

There is a long history of work in the social sciences on family size distributions. The classic founding work in this field is that of Galton and Watson (GW) [4] who tried to explain the decline of the British great families, as indicated by data on surname abundance. Rejecting previous explanations based on “fitness”, e.g., that the rise of physical comfort is followed by fertility decline, they assumed that the phenomenon is purely statistical. The affiliation of an individual with certain family, expressed in his/her surname, was assumed by GW to be a neutral property. This feature is inherited to the next generation according to a well defined rule (all offsprings take the surname of their father) and is subject to the stochasticity that characterizes birth-death processes. Assuming a well-mixed population, GW claimed that all surnames undergo extinction in the long run. In fact, their conclusions were correct only for an equilibrium population, whereas for a growing population, their equations exhibit a second nontrivial solution which was found by Steffenson [13, 14] and exploited by Lotka [8, 9] using U.S. census data to deduce the offspring distribution. Subsequently the impact of surname changes (“mutations”) was considered by Manrubia and Zanette (MZ) [10]. All in all, the surname in a society undergoes a birth-death-mutation process, and the current surname abundance distribution reflects the demographic (birth-death ratio) and social (mutation rate) characteristics of the population. MZ also presented data for the distribution of surname in the populations in Argentina, Berlin, and five cities in Japan, where the statistics were obtained from phonebooks. The data exhibited the predicted $1/n^2$ behavior at large $n$ for the probability of $n$ appearances of a surname. MZ then
attempted to use the deviations for smaller \( n \) to deduce the growth rate of the population.

As already pointed out in Manrubia et al. [11], the importance of the clan statistics for a population that undergoes a birth-death-mutation process goes far beyond its applicability to surname dynamics. Any neutral genetic feature associated with a sequence that appears on certain loci and is subject to mutations undergoes exactly the same process, thus the results for surnames reflect also the amount of genetic polymorphism in the population. Another neutral process of the same kind was suggested by Hubbell [5, 6, 2] and Bell [2] as the underlying mechanism that yields the observed species abundance distribution. This heretical idea opposes the traditional "niche" theories that seeks to explain species abundance ratios in terms of interspecies interaction and fitness, and ignited an enormous contentious debate on that subject [12].

The argumentation of both sides is based on the species abundance statistics, as gathered in large-scale censuses [3] and the very same problem arises: what statistics should one expects in case of a growing or shrinking population which is subject to neutral mutation?

Before trying to compare the observed statistics with some theoretical predictions (e.g., in order to recover demographic parameters from the abundance ratio) one should address two crucial issues. The first is un\textit{versality}: to what extent should one expect the results to be independent of the "microscopic" features of the process? Fig. 1 shows the family size statistics (Pareto plot) obtained from numerical simulations of two populations with the same demographic characteristics. The dynamics assumes nonoverlapping generations, where the average number of offspring per individual is \( \lambda > 1 \) and the chance of an offspring to mutate (i.e., to differ from its originator and to start a new clan) is \( \mu \). Both populations have the same values for \( \lambda \) and \( \mu \), but they differ microscopically: in one case the chance for an individual to produce \( n \) offsprings obeys the Poisson distribution with average \( \lambda \), in the other case it satisfies the geometrical distribution with the same average. As one can clearly see, the tails of these distribution coincides, but the bulk abundance statistic is different; this implies that the theory of abundance ratio has no use for any practical purpose unless one knows the very fine details of the demographic properties of the population throughout history, an inconceivable task in almost all circumstances. A comparison between experimental data and theoretical predictions is possible if, and only if, one can show that there is a universal regime in which the statistics is independent of the microscopic details; this is one of the aims of this paper.

The second issue that should be addressed is the effect of sampling. In all cases considered above - surnames, genetic polymorphism, species abundance - the raw data is made of individuals sampled from the whole population together with their affiliation with certain surname or certain species. It is difficult to perform a complete census, given that typically one does not have access to the entire population. Thus for example, MZ only looked at the surnames beginning with “A” in the Berlin phone book. Even for the US census data, one has access to (almost) the entire population only under the assumption that the US is demographically isolated, which it is clearly not. In the application we have in mind, that of looking at genomic data to measure historic growth rates of the population, one has such data for an extremely small sample of the entire human population.

What is the effect of incomplete sampling? In Fig. 2 one can see the characteristic features of
Figure 2: Avg. number of families of a given size, for the full population of \(4 \cdot 10^6\), and subsamples of size \(2 \cdot 10^5\), \(2 \cdot 10^4\), \(2 \cdot 10^3\) and \(200\). The growth rate is \(\gamma = 0.005\), and the mutation rate is \(\mu = 5 \cdot 10^{-4}\), and the child distribution is Poisson. The circles represent averages over 100 iterations. The lines are the theory for the full population, Eq. (7) and for a “red” subsample, Eq. (8). The deviations from the power law for the largest \(m\)’s seen in the \(N_0 = 200\) and 2000 data are due to the fact that the largest family does not obey the stable distribution, but rather reflects the single individual initial conditions chosen [10].

the family statistics obtained in the two regimes: strong and weak sampling. One can see that the full statistics is characterized by a ”shoulder” in the small families region, followed by a power-law decay for large families. If the sampling is strong the distribution is shifted quite rigidly to the left, while the case of weak sampling is characterized by a peak for the singletons (families with only one member) followed by a power-law. Our second aim here is to clarify effect in both regimes.

In the following, we analyze the problem from two different angles. The first is centered on the stable distribution for the entire population. This distribution can be calculated from a Fokker-Planck equation, akin to that written down by MZ. We show this Fokker-Planck equation is in fact universally valid in the limit of small growth rate, for an arbitrary distribution of children produced by an individual, with the coefficients depending only on the average and variance of the children distribution, together with the mutation rate. From this we can calculate the distribution for a given sized subsample of the population in terms of a hypergeometric function. We then endeavor to assimilate the meaning of this result, focussing on the strong and weak sampling limits, exhibiting simpler formulae for the average family size and number of singleton families. We also show that the large-family power-law asymptotics is left unchanged by the sampling.

The second approach is based on looking at the behavior of the genealogical tree of the selected sample. We calculate the size of the tree as a function of time, as well as the number of families and singletons, all in the limit of small mutation rate. These results are seen to reproduce those of the previous approach for the current statistics of the selected sample.

The plan of the paper is as follows. In Section 2 we describe our model, explain the notation used along this work and highlight our main results. In Section III, we present our derivation of the family size distribution for the subsample, and calculate the average family size and number of singleton families. In Section IV, we present our second approach. Finally, in Section V we examine the surname distribution taken from the U.S. census data in light of our findings. We then summarize and present some concluding remarks.

2. Model, simulation technique and main results

Our basic model is that of a growing population with nonoverlapping generations, as in the original Galton-Watson work. Every member of the population simultaneously gives birth to a random number of children, drawn from a given distribution with mean \(\lambda\), and is then removed. The children are all reckoned to belong to the same family as the parent, unless they undergo a mutation at birth, which occurs with probability \(\mu\). The mutated child is considered to start a new family. We start the population with one individual, repeating the experiment until the population survives the initial stages and achieves the desired size. In principle, we could track the genealogy of every individual. In practice, for efficiency’s sake, we track only the genealogy of families, which is sufficient to determine the family identification of every individual. Thus, it is sufficient to draw the number of children of each family. In our simulations, we mostly employ a Poisson distribution for the number of children, occasionally comparing to the case of a geometrical distribution. In the former case, the distribution of children of a given family of size \(n\) is again Poisson, with mean \(\lambda\), whereas for the geometrical
distribution, it is a Pascal (or negative binomial) distribution.

As a technical point, we will be interested in Section V in the genealogical tree of subsamples of the population, so that we can track the time development of the statistics of this tree. We can do this retrospectively for the Poisson case, simply picking ancestors for each individual among the set of individuals in the family in the previous generation which gave rise to this individual (which is the same family, barring mutations). This is done to avoid having to store the genealogies of individuals, which are of course more voluminous than those of families.

**Glossary:** The growth rate $\gamma \equiv \lambda - 1$ reflects the deviation of the process from demographic equilibrium. In general, as discussed above, the distribution function depends on the details of $P(m)$, the chance of an individual to have $m$ children in the next generation. It turns out, however, that in the universal regime the family statistics depends only on three parameters: $\gamma$ (or equivalently $\lambda$), which reflects the average number of offspring per individual, $\sigma$, the standard deviation of the offspring distribution defined as

$$\sigma^2 \equiv \sum_m m^2 P(m) - \lambda^2 = \text{Var}(m),$$

and $\mu$, the mutation rate. For convenience, we define

$$\nu \equiv \frac{\mu}{\gamma - \mu}$$

as this combination appears often.

The number of families with $m$ members is defined as $n_m$. These definitions imply that the sum of $mn_m$ over all $m$’s yields the overall current size of the population, $N_c$. Except for $m$’s of order unity, one may consider the size of the family as a continuous variable, thus replacing $m$ by $x$ and $n_m$ by $n(x)$. When the sampling is incomplete we tag the sampled individuals as “red”, defining $n^R_m$ as the abundance of families with $m$ individuals in the red (sampled) population [When dealing with the whole genealogy we define a “red” subgenealogy consisting of all the individuals that have at least one descendent in the sampled population]. Sampling introduces a new parameter to the problem, $R_c$, the number of sampled (“red”) individuals. It turns out that there is a “critical” sample size which distinguishes between weak and strong sampling:

$$R_c \equiv \frac{2N_o\gamma}{\sigma^2(1 + \nu)}$$

We then measure sampling strength, $s$, through

$$s \equiv \frac{R_o}{R_c} = \frac{R_o\sigma^2(1 + \nu)}{2N_o\gamma}. \tag{4}$$

Our main results are:

1. In the large $m$ limit $n_m$ decays like a power-law, $n_m \sim m^{-\beta}$ where

$$\beta = \frac{\ln \lambda^2(1 - \mu)}{\ln(1 - \mu)} \tag{5}$$

This law is semi-universal, in that it is independent of the details of $P(m)$. It however depends on the assumption of nonoverlapping generations, and therefore differs from the power law found by MZ. It does however reduce to the MZ result, $\beta = 2 + \nu$, in the slow growth, small mutation limit $\gamma \sim \mu \ll 1$.

2. The whole distribution (except for the very smallest $m$’s) becomes universal if both $\mu$ and $\gamma$ are small. In that case $n(x)$ satisfy the Fokker-Planck equation:

$$\frac{\partial n}{\partial t} = -(\gamma - \mu)\frac{\partial}{\partial x} (xn) + \frac{\sigma^2}{2} \frac{\partial^2}{\partial x^2} (xn). \tag{6}$$

A similar equation has been obtained by MZ for their particular model; here we show that it is a universal limit of the process for small rates, and also reveal its dependence on $\sigma$.

3. Solving for the steady state distribution of $n(x)$, the abundance distribution function is:

$$n(x) \equiv \frac{\nu R_c}{x} \Gamma (2 + \nu) \left(1 + \nu, 0, \frac{2\gamma}{\sigma^2(1 + \nu)} x\right) \tag{7}$$

where $U$ is the Kummer function [1]. Thus, the abundance distribution for different microscopic processes with the same $\gamma$ and $\mu$ are related by a rescaling of the family size $m$ and the abundance $n$, $n\sigma^4$ being a universal function of $m/\sigma^4$ (since $R_c \propto 1/\sigma^4$). We see this in Fig. 3.

4. For the sampled (“red”) population, $n^R_m$ is given by the monstrous expression:

$$n^R_m \approx \nu R_c B (2 + \nu, m) s^m 2F_1 (m, m + 1; m + 2 + \nu; 1 - s)$$

where $B(a, b) \equiv \Gamma(a)\Gamma(b)/\Gamma(a + b)$ is the Beta function and $2F_1$ is the hypergeometric function [1], and $s$ is the sampling strength introduced above. To digest this, we focus on two
limits, that of strong and weak sampling. In the strong sampling limit, \( \gamma N_o \ll R_o \ll N_o \), so that \( s \gg 1 \),
\[
n^R_m \approx \nu R_o \frac{\Gamma(2 + \nu)}{m} U(1 + \nu, 0, m/s) \quad (9)
\]
Thus, since \( s \) is proportional to \( R_o \), when varying \( R_o \), \( R_o n^R_m \) is a function only of \( m/R_o \), and the dependence of \( R_o \) just amounts to rescalings of \( m \) and \( n^R_m \). This implies that in this limit the breakdown of the asymptotic power-law occurs at \( m \)'s of order \( N_o \gamma/R_o \), and in general sampling induces a rigid displacement of the family size distribution to the left and downward. For strong but partial sampling the formula applies all the way down to \( m = 1 \), whereas for the full population, the formula breaks down for the smallest \( m \)'s. For small argument, \( U \) approaches a constant, and so for \( m \ll s \), we get
\[
n^R_m \approx \frac{\nu R_o}{m} \quad (10)
\]
For large arguments, we recover the standard power-law. This is evidenced in Fig. 4. For weak sampling, \( R_o \ll \gamma N_o \), i.e., \( s \ll 1 \), the sampling strength decouples from the distribution for \( m > 1 \) except to set the overall normalization:
\[
n^R_m \approx B(2 + \nu, m - 1 - \nu)\nu R_o s^\nu \quad m > 1 \quad (11)
\]
This is demonstrated in Fig. 5. The distribution rapidly approaches the expected power-law behavior from above as \( m \) increases. Thus, the shoulder has disappeared completely. The families of family size 1, which we denote singletons, are exceptional for weak sampling, since the chance of sampling more than one member from a given family vanishes in the small \( s \) limit, except for small (scaled) mutation rate \( \nu \), where there are anomalously large numbers of large families.

5. The average red family size, \( \bar{m}^R \), is given by the equally monstrous formula
\[
\bar{m}^R = \frac{s(1 + \nu)}{\nu \frac{\nu}{\ln a s}} 2F_1 (1, 1; 3 + \nu; 1) - (1 - s) 2F_1 (1, 1; 3 + \nu; 1 - s)
\quad (12)
\]
This is shown in Fig. 6, where \( \bar{m}^R \) is shown as a function of \( R_o \), together with the results of numerical simulations. For strong sampling, there of order \( \ln \) red families, and
\[
\bar{m}^R \approx \frac{s}{\nu \ln a s} \quad (13)
\]
where \( a \) is a \( \nu \) dependent constant which approaches unity for small \( \nu \), given by Eq. (38). In particular, in the full sample, \( s = \sigma^2(1 + \nu)/2\gamma \), and the average family size is large, of order \(-1/(\gamma \nu \ln \gamma)\).

For weak enough sampling, the average family size approaches unity, since all families are
singletons. For small $\nu$ however, this again occurs only for extremely small samples, and in practice,
\[
\overline{mR} \approx \frac{1 - s}{\nu \ln s}
\]
so that for small $\nu$ the average red family size is large, and decreases logarithmically as the sampling strength decreases.

6. The distinction between weak and strong sampling is also reflected in the statistics of the genealogical tree of the subsample. For strong sampling, this is a strong coalescence at first as we must upward in the tree. For weak sampling, on the other hand, the coalescence is very small initially. Eventually, both trees narrow exponentially. For critical sampling, the narrowing of the tree is exponential for all times.

This then is the general outline of our results. In the next section, we turn to a detailed derivation of these findings.

3. General analysis of the branching-mutation process

We start with the family size distribution for the entire population. This is given by the solution to a Fokker-Planck equation generalizing that derived by MZ for their model of overlapping generations. We start with the set of difference equations for the evolution of the whole-population distribution:

\[
\begin{align*}
n_{m+1}^{t+1} &= \sum_{\ell \geq m} \ell n_\ell P(\ell \rightarrow m) \left(\frac{p}{m}\right)^{\nu-1} (1 - \mu)^m; \quad m > 1 \\
n_1^{t+1} &= \sum_{\ell} \ell n_\ell P(\ell \rightarrow 1) \left(\frac{p}{1}\right)^{\nu-1} (1 - \mu) + \mu N(t + 1)
\end{align*}
\]

The first equation represents the contribution of a family of size $\ell$ giving birth to a family of size $p$, $m - p$ are whom mutate, leaving a family of size $m$. The probability $P(\ell \rightarrow m)$ of $\ell$ individuals to give birth to $m$ children is derived in terms of the fundamental distribution of the number of children of a single individual, $P(m)$, whose mean we label $\lambda$. All mutations become new families of size one.

Before we present the differential equation, we can verify that asymptotically for large $m$ (item 1 above), the stable distribution falls like a power.
This can be accomplished by using the central limit theorem and evaluating the sums via the Laplace method. The details are this calculation are presented in Appendix A. We find that indeed \( n_m \sim m^{-\beta} \) where

\[
\beta = \frac{\ln \lambda^2 (1 - \mu)}{\ln (1 - \mu)}
\]  

(16)

This is different from the MZ result, and reflects the difference between the overlapping generations in their model versus the synchronized update of this model. Nevertheless, writing the growth factor \( \lambda \equiv 1 + \gamma \) and going to the slow growth, small mutation limit \( \gamma \sim \mu \ll 1 \), we get to leading order

\[
\beta \approx \frac{2 \gamma - \mu}{\gamma - \mu} = 2 + \nu
\]  

(17)

This is the MZ result, indicating that for overlapping generations the growth rate is always small in some sense. In any case, we conclude that when the growth and mutation are sufficiently small so that the population changes slowly, the details of the update procedure no longer matter.

By using the generating function for the child distribution, we can derive, as detailed in Appendix B the Fokker-Planck equation (item 2):

\[
\frac{\partial n}{\partial t} = -(\gamma - \mu) \frac{\partial}{\partial x}(xn) + \sigma^2 \frac{\partial^2}{\partial x^2}(xn)
\]  

(18)

This equation is approximate. In particular, the coefficients are presented, in light of the discussion above, to leading order in \( \mu \) and \( \gamma \). In addition, we have truncated the equation at terms of second order. There are additional second derivative terms, which arise from the third “spatial” derivative of \( xn(x) \), which we have as dropped, as do MZ. The first order derivative represents the drift to larger population, with an effective growth rate for the family of \( \gamma - \mu \), since mutations reduce the family size. The coefficient of the second derivative term, \( \sigma^2 \), is the variance of the given children distribution of an individual. It is eminently reasonable that the variance in the number of children is what gives rise to diffusive behavior of the family size. For the case of the geometric distribution of children, which characterizes the MZ model, with variance 2 in the small \( \gamma \) limit, the equation reduces to theirs.

Thus, up to the change of the diffusion constant, the stable distribution in this approximation is again the Kummer function \( \text{Kummer}(\beta, \alpha, x) \)

\[
n(x) \approx \frac{A}{x} U(1 + \nu, 0, 2(\gamma - \mu)/\sigma^2, x)
\]  

(19)

where \( A \) is a normalization factor. From here, we can justify the neglect of the higher derivatives. The argument of the Kummer function is proportional to the small quantity \( \gamma - \mu \), so that higher derivatives bring down additional factors of this small quantity and are thus indeed negligible. This stems from the fact that the drift term is small, and again hearkens back to the necessity of assuming slow growth and small mutation probability.

The normalization factor can be obtained from the size of the entire population, which we denote \( N_o \), since

\[
N_o = \sum_{m \geq 1} mn_m \approx \int_0^\infty xn(x)dx
\]  

(20)

This integral can be performed analytically [see Eq. 7.612.2], yielding (item 3)

\[
n(x) = \frac{\nu R_o}{x} \Gamma(2 + \nu) \ U(1 + \nu, 0, \frac{2 \gamma}{\sigma^2(1 + \nu)} x)
\]  

(21)

It should be noted that this normalization is different from that of MZ. The fact that the distribution is proportional to \( \mu \) flows from the fact that for \( \mu = 0 \) the distribution goes like \( 1/x^2 \) and so the integral diverges as \( 1/\mu \).

The form of the distribution means that \( \sigma^4 n \) is a function of the scaled family size \( m/\sigma^2 \), for all offspring distributions. This collapse is shown in Fig. 3, where the data for the Poisson and geometric offspring distributions is plotted, together with the analytic prediction. We see the analytic prediction is excellent except for the singletons, the families of size one. In any case, these are not expected to be given by the formula, since the governing equation for the singletons is exceptional.

From the full population distribution, it is straightforward to generate, at least formally, the family size distribution, \( n_m \), for the “red” subsample of size \( R_o \):

\[
n_m^R = \sum_{p \geq m} n_p \binom{p}{m} \binom{N_o - p}{N_o - R_o}
\]  

(22)

which reflects the hypergeometric probability of choosing \( m \) red members of an original family of size \( p \), when choosing \( R_o \) from \( N_o \). When \( R_o \ll N_o \), the hypergeometric distribution reduces to a Poisson distribution

\[
n_m^R \approx \sum_{p \geq m} n_p e^{-pR_o/N_o} \binom{pR_o}{m} \frac{1}{N_o^m}
\]  

(23)
We can replace the sum by an integral, yielding [see 2 Eq. 7.621.6], the result quoted in the 4th item above,

\[ n^R_m \approx \frac{1}{m!} \left( \frac{R_o}{N_o} \right)^m \int_0^\infty dx x^{m-1} e^{-xR_o/N_o} \times \]

\[ AU \left( 1 + \nu, 0, \frac{2\gamma}{\sigma^2(1 + \nu)^2} \right) \]

\[ \approx \nu R_o B(2 + \nu, m) s^m \times \]

\[ _2F_1 \left( m, m + 1; 2 + \nu + m; 1 - s \right). \]  (24)

In Fig. 2 above, we have included together with the simulational results for the family size distribution for various size subsamples the predictions of our formula Eq. (24). We see that for all but the largest families in the smallest subsample, there is excellent agreement, even for a subsample which is 1/20000 of the whole.

The first thing we can verify with our analytic formula is that the large \( m \) behavior of the distribution is unchanged, except for normalization. For large \( m \), the sum is dominated by \( p \)'s of order \( mN_o/R_o \). Expanding the summand around this maximum yields a Gaussian, and we see that power-law is preserved.

We now investigate our result in the two limits of strong and weak sampling, defined by \( s \gg 1 \) and \( s \ll 1 \), respectively. In the limit of strong sampling, we return to our fundamental expression for \( n^R_m \), Eq. (23). As we shall see momentarily, the typical scale of family size over which \( n_m \) varies is large, of order \( s \). Thus, the Poisson sum is essentially a Gaussian centered at \( \nu \) so that the whole distribution is unchanged, except for normalization. For large \( m \), the sum is dominated by \( p \)'s of order \( mN_o/R_o \). Expanding the summand around this maximum yields a Gaussian, and we see that power-law is preserved.

Since \( n_m \) itself varies over the scale \( 1/\gamma \), which is much larger, to leading order the sum over \( \ell \) reduces to a \( \delta \)-function, and we have

\[ n^R_m \approx \nu R_o \frac{\Gamma(2 + \nu)}{m} U(1 + \nu, 0, m/s) \]  (25)

Alternatively, we can obtain this expression directly from our general formula for \( n^R_m \) using the integral representation of \( _2F_1 \) and taking the large \( s \) limit. The first derivation shows that the result is valid even for \( R_o \sim N_o \), beyond the range of the Poisson sampling approximation, since the argument generalizes to the original hypergeometric sampling formula, Eq. (22). This is evident from the fact that we recover the full population distribution if we simply set \( R_o = N_o \). However, the partial sample distribution formula is reliable for all \( m \), whereas for the full population, the formula does not apply to \( m \)'s of order unity. As we noted above, this form for \( n^R_m \) implies that as we vary \( R_o \), the whole distribution moves rigidly leftward and downward. In particular, for \( m \ll s \), \( U \) approaches a constant value, 1/\( \Gamma(2 + \nu) \), and

\[ n^R_m \approx \frac{\nu R_o}{m} \]  (26)

Clearly for \( m \gg s \), we recover the MZ power-law, as expected. We refer the reader back to Fig. 3 for a graphical presentation of the strong sampling regime.

Since the shoulder of the distribution extends to \( m \)'s of order 1, we can see clearly the case \( m \gg 1 \) is exceptional, \( m \ll s \) and requires a separate treatment. We also see
that the distribution now approaches the asymptotic power-law from above. The weak sampling regime is illustrated in Fig. 5 above.

We have seen that the case of the singleton families, \( m = 1 \), requires special attention, particularly for weak sampling. Using the transformation formula above, we can verify that in the weak sampling limit, \( s \to 0 \), \( n^R_1 \) approaches \( R_o \), consistent with the vanishing of the larger size families in this limit. However, this is misleading for small \( \mu \). In this limit, the hypergeometric function can be expressed in terms of elementary functions, and

\[
n^R_1 \approx \frac{-R_o \nu}{(1-s)^2} (1 - s + \ln s) + \mathcal{O}(\nu^2) \tag{32}
\]

Thus, for dilute sampling, \( n^R_1 / R_o \) is seen to be approximately the small quantity \( -\nu \ln(s) \). The logarithmic behavior is again a reflection of the slow \( 1/x^2 \) decay of the whole population family distribution in the small \( \mu \) limit. Only for ridiculously dilute samples, with \( R_o / N_o \) of order \( \gamma e^{-\gamma/\mu} \), does the whole subsample reduce to singletons. This is what we see in the graphs of the simulations in Fig. 2 where even for the smallest subsample shown, with \( s = 0.0222 \), there are only 73 singletons in a population of 200. In the strong sampling regime, as can be seen from Eq. (26), we get \( n^R_1 \approx \nu R_o \), so it is independent of \( R_o \). This convergence is also apparent in the data. Overall, the fraction of singletons decreases with the strength of the sampling.

The last quantity of interest is the total number of red families, which we denote by \( F^R \). In principle we can calculate it as the sum over \( m \) red family size distribution \( n^m_1 \), but it is easier to go back to the definition of \( n^m_1 \) in terms of \( n_p \) and sum over \( m \) first, leaving the sum over \( p \) for last. The sum over \( m \), if it started at \( m = 0 \) would just give unity, but because it starts at \( m = 1 \), yields, in the Poisson approximation \( R_o \ll N_o \):

\[
F^R \approx \sum_{p \geq 1} n_p \left( 1 - e^{-pR_o/N_o} \right) \tag{33}
\]

Thus, all families in the full population give a family of some size in the red population, unless they are not picked at all, which occurs with probability \( exp(-pR_o/N_o) \), where \( p \) is the size of the originating family. Plugging in our expression for \( n_p \), and converting the sum to an integral yields

\[
F^R \approx \int_0^\infty \left( 1 - e^{-xR_o/N_o} \right) \times \frac{A}{x} \left( 1 + \nu, 0, \frac{2\gamma}{\sigma^2(1 + \nu)} x \right) dx \tag{34a}
\]

\[
= \frac{\nu R_o}{(2 + \nu)^s} \left[ _2F_1(1, 1; 3 + \nu; 1) - (1 - s)_2F_1(1, 1; 3 + \nu; 1 - s) \right] \tag{34b}
\]

The details of this calculation are presented in Appendix C. Again, formally, for any finite \( \nu \), we get that \( F^R \) approaches \( R_o \) as \( s \to 0 \), as all families are singletons. Nevertheless, for small, but not absurdly small, \( \nu \), the number of families reads

\[
F^R \approx -\nu R_o \frac{s \ln s}{1 - s} . \tag{35}
\]

Thus, the average family size, \( \overline{m} \equiv R_o / F^R \) is given for small \( \nu \) by

\[
\overline{m} = \frac{1 - s}{\nu \ln s} \tag{36}
\]

which is large but decreases logarithmically at small sampling, cutting off at one for extremely small samples. density \( s \). It is interesting that whereas both the number of red families and the number of red singletons behave anomalously for small samples and small \( \mu \), the fraction of red families that are singletons is smooth, approaching unity as it should.

At critical sampling, \( F^R = \nu R_o / (1 + \nu) \), and \( \overline{m} = (1 + \nu) / \nu \), which is large for small \( \nu \). For large \( s \), and general \( \nu \), the number of families is dominated by the contribution from the small families, which behaves as \( 1/m \) and so is logarithmically large:

\[
F^R \approx \nu R_o \ln \alpha \tag{37}
\]

where the \( \mathcal{O}(1) \) constant \( \alpha \) is given by

\[
\ln \alpha = \psi(1) - \psi(2 + \nu) + \frac{1}{1 + \nu} \tag{38}
\]

and \( \psi \) is the digamma function, so that \( \alpha \) approaches 1 for small \( \nu \). Thus the average family size diverges for large \( s \),

\[
\overline{m} \approx \frac{s}{\nu \ln \alpha} \tag{39}
\]

in agreement with what we found for small \( \nu \).
4. Red Statistics Through Time

4.1. Red Population

We now present an alternative derivation of these results (at least in the small $\nu$ limit), based on tracing the time development of the red genealogy. We have labeled an individual “red” if he is in the selected sample of the final population. We also label as red any ancestor of such an individual. We first address the question of the time development of the red population. The basic tool for the analysis, as with the original Galton-Watson work, is the generating function for the distribution of children, which we denote by

$$F(x) = \sum_{n=0}^{\infty} P(n)x^n$$

(40)

The key is the Galton-Watson observation that the generating function for the probability of descendants in the second generation, $F_2(x)$ is the second iterate of $F$; i.e., $F(F(x))$. We can see this noting that

$$P_2(n) = \sum_{i=0}^{\infty} P(i)P(i+n)(n)$$

so that

$$F_2(x) = \sum_{i,n} P(i)P(i+n)x^n = \sum_i P(i)(F(x))^n$$

(41)

We thus see the origin of the “critical” value of sampling we encountered in the previous section, namely the change in behavior depending on whether $R_o/N_o$ is less than or greater than $\delta \approx 2\gamma/\sigma^2$; i.e., whether $R_o$ is less than or greater than $2\gamma N_o/\sigma^2$, which is $R_e$ to leading order in $\nu$. For the remainder of this section, we will in fact rewrite $2\gamma N_o/\sigma^2 = R_e$, as we are working only to leading order in $\nu$.

As long as $x$ is close to one, $F(x)$ will similarly be close to one. Thus, for $R_o \ll N_o$, $1 - R_o/N_o$ meets this criterion and we can approximate the change in $\delta_n = R_n/N_n = 1 - Q_n$, the fraction of reds in the population.

$$\delta_{n+1} = 1 - F(1 - \delta_n) \approx \lambda \delta_n - \frac{\delta_n^2}{2} \left(\sigma^2 - \lambda(1 - \lambda)\right)$$

$$\approx (1 + \gamma)\delta_n - \frac{\delta_n^2 \sigma^2}{2}$$

(47)

so that

$$\frac{d\delta}{dn} = \gamma \delta - \frac{\sigma^2 \delta^2}{2}$$

(48)
The change in behavior as $R_o$ crosses $R_c$ is apparent. In particular, at critical sampling, $R(n)$ is a pure exponential in time. We also see that $R_n$ depends on the underlying distribution of children only through its average, i.e. $\gamma$, and its variance.

In Fig. 7 we show data for the $R(n)$ from a single simulation, for three different sampling strengths, together with our analytic formula, Eq. (50). The data exhibit the striking change in behavior from a fast initial decrease in red individuals for strong sampling, a pure exponential for critical sampling and a slow initial decrease for weak sampling. All three curves merge in the past, at the coalescence time for the entire population.

An alternate, equivalent way to arrive at our result is to consider the coalescence of branches on the red tree. If we look at the $R_n$ reds in the $n$th previous generation, these are generated by slightly less than $R_n$ parents, due to coalescence. The chances of coalescence, per potential parent, is the chance of having two surviving children, $\sum P(n)\frac{n(n-1)}{2} \delta^2 \approx \sigma^2\delta^2/2$. Thus, the decrease in the number of reds is $\sigma^2 R^2/2N$ so that the equation for $R$ is

$$\frac{dR}{dn} = -\frac{\sigma^2 R^2}{2N} \quad (51)$$

which is equivalent to our above equation for $\delta$.

4.2. Red Families

We now turn to examine the time development of the number of red families, $F^R(n)$, $n$ steps in the past. As in the absence of mutation every family survives, since it is red, the only change in families from $n$ steps in the past to $n-1$ steps in the past is the new families due to mutation. As mutations of singletons do not create new families, we have

$$F^R(n-1) = F^R(n) + \mu(R(n) - nR(n)) \quad (52)$$

In the small $\mu$ limit, the number of singletons is small, proportional to $\mu$. Thus to leading order in $\mu$, we have

$$F^R(n-1) = F^R(n) + \mu R(n) \quad (53)$$

or, in its differential equation form,

$$\frac{dF^R}{dn} = -\mu R \quad (54)$$

Using our solution, Eq. (50), for $R(n)$, we obtain

$$F^R(n) = \frac{\nu R_c R_o}{R_c - R_o} \ln \left(1 + \left(\frac{R_c - R_o}{R_c - R_o}\right) e^{-\gamma n}\right) \quad (55)$$

where we have demanded that $F^R \to 0$ as $n \to \infty$. In particular, at the sampling time,

$$F^R(0) = \frac{\nu R_c R_o}{R_c - R_o} \ln \frac{R_c}{R_o} \quad (56)$$

which of course agrees with the small $\mu$ limit result Eq. (36) we derived using the Fokker-Planck approach.

In Figure 8 we show data for the number of red families going backward in time, again for subcritical, critical and supercritical sampling. The small $\nu$ result, Eq. (55), is also shown. The small deviation is due to higher order corrections in $\nu$.
4.3. Red Singletons

We now examine the time development of the number of red singletons. The number of singletons decreases due to the fact that some singletons give birth to multiple children, who are no longer singletons. It increases due to the fact that mutations of non-singletons give rise to new singletons. As discussed above, the decreases in number of reds due to coalescence is $\sigma^2 R(n)^2/2N(n)$. Of these a fraction $n^R/R$ are singletons, so the loss of singletons due to coalescence of singletons is $\sigma^2 R n^R / 2N$. Thus the differential equation for $n^R_1$ is

$$\frac{dn^R_1}{dn} = \frac{\sigma^2 R R_1^R}{2N_0 e^{-\gamma n}} - \mu (R - n^R_1)$$

(57)

Again we can drop the $\mu n^R$ term as being higher order in $\mu$. Then the solution that vanishes as $n \to \infty$ is

$$n^R_1(t) = \frac{\nu R_c R_o}{(R_c - R_o)^2} \left[ -(R_c - R_o) + \left( R_c e^{\gamma n} + (R_c - R_o) \right) \times \ln \left( 1 + \left( \frac{R_c}{R_o} - 1 \right) e^{-\gamma n} \right) \right]$$

(58)

In particular, at the sampling time the number of red singletons is given by

$$n^R_1(0) = \frac{\nu R_c R_o}{(R_c - R_o)^2} \left[ R_c \ln \frac{R_c}{R_o} - (R_c - R_o) \right]$$

(59)

This again can be seen to reproduce our Fokker-Planck result, Eq. (32). It is interesting to note that at critical sampling $n^R_1$ approaches $\nu R_c / 2$, whereas $F^R$ approaches $\nu R_c$, so that half the families are singletons in this case. The simulation data for $n^R(n)$ is presented in Figs. 8 and 9 together with the small $\nu$ prediction, Eq. (58). The picture is qualitatively similar to that of the red families.

5. The U.S. Census Data

We now attempt to apply the theory to the surname distribution taken from the 2000 U.S. Census. We must make clear at the outset that degree of overlap between the assumptions underlying our theoretical treatment and the real dynamics of the

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Available at [http://www.census.gov/genealogy/www/freqnames2k.html](http://www.census.gov/genealogy/www/freqnames2k.html)  
This data only extends to surnames with more than 100 representatives. Data for rarer surnames is available in binned form from [http://www.census.gov/genealogy/www/surnames.pdf](http://www.census.gov/genealogy/www/surnames.pdf)

The binned data was then debinned using a smoothing procedure
The surname distribution may rightfully be questioned. Nevertheless, we will proceed and see how far we can go.

The surname data is presented in Fig. 10. We see that its overall shape is similar to that of the theory. In particular, the graph exhibits a $1/m^2$ falloff for large family sizes, in accord with our expectation. However, upon closer examination, the data presents us with a severe problem. The asymptotic power law only sets in for family sizes above $10^4$ or so. According to the theory, the onset of the power law should occur roughly at $m \sim 10/(2\gamma)$. Thus, the data would point to a value of $\gamma$ of roughly $5 \cdot 10^{-4}$. However, this is off from the true growth rate of the population by a factor of 1000!

The growth rate of the U.S. population has not been constant in time. However, it was quite constant up until the severe immigration restrictions were applied in the wake of World War I, as can be seen from Fig. 11 with a value of 0.0255/year. Assuming a generational time of 20 years, this works out to give a value of $\gamma = 0.51$. Even taking the post-WWI growth rate, the growth rate is only reduced to $\gamma = 0.3$, so we still have a factor of 500 to account for. Even if we compare the data to the theoretical curve with $\gamma = 5 \cdot 10^{-4}$, so as to reproduce the break from the power-law at $m = 10^4$, the agreement only extends down to family sizes of $m = 500$.

One might think that the problem is that the U.S. is not a demographically closed system. The large impact of immigration in driving the population growth before WWI is clear evidence of this. One might then want to claim that the U.S. population should be considered as a (biased) sample of the European (or perhaps, world) population. The growth rate of the European and world population before 1920 is roughly a factor of 5 smaller than the U.S. growth rate. However, this improvement is more than counterbalanced by the sampling effect, which is to move the distribution leftward by the sampling factor, thereby moving the onset of the asymptotic $1/m^2$ scaling to even smaller $m$. Thus, the solution cannot lie in this direction.

In fact, no clear answer presents itself at this point, and we must leave this puzzle as a challenge for the future. One possibility might be that the “mutation” of family names, at least for the U.S., is likely very different from that assumed by the model. There was a great tendency in the period of the great immigration for family name changes not to create new surnames, as we assume, but in fact the opposite. The immigration officials were (in)famous for mapping the wide spectrum of surnames they encountered onto recognizable “American” names. Further work will be needed to see what effect this might have had on the surname distribution.

In summary, then, we have calculated the family size distribution for a growing population. We have shown that the distribution is universal for slow growth rate. In addition, we have calculated the distribution for arbitrarily sized subsamples of

Figure 10: The surname distribution taken from the 2000 U.S. census. Also show is a curve outlining a $1/m^2$ power-law decay, and the theoretical curve, Eq. (7) for $N_o = 2 \cdot 10^8$, $\gamma = 5 \cdot 10^{-4}$, and $\mu = 10^{-4}$.

Figure 11: The population of the U.S. as a function of time, together with an exponential fit to the period prior to 1920.
the population. We found a distinction between strong sampling, where the shoulder at small family sizes, while truncated still exists, and weak sampling, where the small family distribution lies above the power-law. The critical sampling dividing these two regimes is \( R_c = 2\gamma N_0/\sigma^2(1 + \nu) \). In the strong sampling regime, the distribution is rigidly translated (in log space) through a rescaling of \( m \). For weak sampling, the distribution is independent of sampling, up to overall normalization. We have also seen that the distinction between weak and strong sampling holds for the properties of the genealogical tree constructed from the sampled individuals.

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A. Derivation of the Power-Law

For large \( m \), the major contribution of the sum over \( p \) is from the vicinity of \( p_* \equiv m(1 - \mu) \) and for the sum over \( \ell \) from the vicinity of \( \ell_* \equiv m_*/(1 - \mu) \). Thus, we can, invoking the central limit theorem, approximate the distribution \( P(\ell \to p) \) by

\[
P(\ell \to p) \approx \frac{1}{\sqrt{2\pi\sigma^2\ell}} e^{-\frac{(p - \lambda\ell)^2}{2\sigma^2\ell}} \tag{60}
\]

Similarly, we can approximate the binomial distribution for the number of mutations by

\[
\binom{p}{m}(1 - \mu)^m \mu^{p - m} \approx \frac{1}{\sqrt{2\pi\mu(1 - \mu)p}} e^{-\frac{(m - \mu(1 - \mu)p)^2}{2\mu(1 - \mu)p}} \tag{61}
\]

Replacing the sums over \( \ell \) and \( p \) by integrals and expanding the exponent to second order yields

\[
n^{t+1}_{m} = \int \int d\ell \, dp \, n^t_m \left[ \frac{1}{2\pi\sigma^2\ell} \frac{1}{2\mu(1 - \mu)p} \right] \exp \left( -\frac{(p - \lambda\ell)^2}{2\sigma^2\ell} - \frac{(m - \mu(1 - \mu)p)^2}{2\mu(1 - \mu)p} \right)
\]

\[
\approx \sqrt{\frac{\lambda(1 - \mu)}{2\pi\sigma^2\ell}} n^t_m \int \int d\ell \, dp \, e^{\mathcal{F}(\ell, p)} \tag{62}
\]

where

\[
\mathcal{F}(\ell, p) = -\frac{\lambda^2(1 - \mu)(\ell - m_*)^2}{2\sigma^2 m} - \frac{(1 - \mu)(\lambda\mu + \sigma^2(1 - \mu))(p - p_*)^2}{2\mu m} - \frac{\lambda^2(1 - \mu)(\ell - \ell_*)(p - p_*)}{\sigma^2 m} \tag{63}
\]

Substituting \( n^t_m = Am^{-\beta}\lambda^t \) and doing the Gaussian integrals gives

\[
\lambda m^{-\beta} = \frac{1}{\lambda(1 - \mu)} \left( \frac{m}{\lambda(1 - \mu)} \right)^{-\beta} \tag{64}
\]

Equivalently, we could simply replace the Gaussians by the delta-functions \( \delta(p - m\ell)\delta(m(1 - \mu)p) \) and integrate over \( \ell \) and \( p \) successively. Taking the logarithm gives us our equation for \( \beta \), Eq. (16).

B. The Fokker-Planck Equation

We start with Eq. (15a), and write \( P(\ell \to p) \) in terms of the generating function, \( F \):

\[
P(\ell \to p) = \int \frac{dz}{z^{p+1}} F^\ell(z) \tag{65}
\]

giving

\[
n^{t+1}_m = \sum_{p \geq m} \int \int \frac{dz}{z^{p+1}} F^\ell(z) n^t_m \left( \frac{p}{m} \right) \mu^{p-m}(1 - \mu)^m \tag{66}
\]

We next expand \( n^t_m \) in a Taylor series around \( n^t_m \):

\[
n^t_m = n^t_m + \frac{\partial n}{\partial m}(\ell - m) + \frac{1}{2} \frac{\partial^2 n}{\partial m^2}(\ell - m)^2 + \ldots \tag{67}
\]

We can now do the geometric sums over \( \ell \) and the sum over \( p \) using

\[
\sum_{p \geq m} \left( \frac{p}{m} \right) x^p = \frac{x^m}{(1 - x)^{m+1}} \tag{68}
\]

to get

\[
n^{t+1}_m = \int \int \frac{dz}{2\pi i} \left( z - \frac{1}{(z - F(\ell))^{m+1}} \right) \left[ -n' n'' + \frac{n'^2}{2} \left( 1 - F(\ell) \right) \right]
\]

\[
\left. + \frac{n''/2}{\lambda^2(1 - \mu)^2} \left[ \lambda^2(1 - \mu)^2 f_3 + 12(1 - \mu)^2 f_2 - 6\lambda(1 - \mu)^2 \lambda f_2 + 6\lambda(1 - \mu)(m + 1)f_2 + \lambda^2(1 - \mu)^2 \right. \right.
\]

\[
\left. - 3\lambda^2(1 - \mu)(m + 1) + (m + 2)(m + 1)\lambda^2 \right] \tag{69}
\]
where we have set the contour between the pole at $z = \mu$ and that at $z = 1$ and picked up the residue at the outer pole at $z = 1$, and the expansion of $F(z)$ near $z = 1$ is

$$F(z) \approx 1 + \lambda(z-1) + f_2(z-1)^2 + f_3(z-1)^3 + \ldots \tag{70}$$

It is possible to translate this difference equation to a differential equation only in the limit $\gamma \sim \mu \ll 1$. Then, expanding the coefficients of the various derivatives of $n$ to leading order, things simplify to

$$\dot{n}(m) = - (\gamma - \mu)n + [-(\gamma - \mu)m + 2f_2]n' + 2f_2mn'' \tag{71}$$

Using that, to leading order in $\gamma$, $f_2 = \sigma^2/2$, gives us Eq. [18].

C. The Number of Red Families

The number of red families is, from Eq. [34a]

$$F_R^\gamma = \int_0^\infty dx \left( 1 - e^{-\nu R_o/N_a} \right) \times \frac{A}{x} \left( 1 + \nu, 0, \frac{2\gamma}{\sigma^2(1 + \nu)} x \right) \tag{72}$$

We cannot break up the term in the parenthesis as is, since both integrals would then diverge. We therefore regularize the problem:

$$F_R^\gamma = A \lim_{\epsilon \to 0} \int_0^\infty \left( 1 - e^{-x^\epsilon} \right) x^{\epsilon-1} U(1 + \nu, 0, x) \, dx \tag{14}$$

$$= A \lim_{\epsilon \to 0} \sum_{n=1}^\infty \frac{\Gamma(\epsilon + n)}{n! \Gamma(2 + \nu + \epsilon + n)} \frac{1}{1 - (1 - s)^n}$$

$$= A \sum_{n=0}^\infty \frac{\Gamma(n + 1)}{(n + 1)! \Gamma(3 + \nu + n)} \frac{1}{1 - (1 - s)^{n+1}}$$

$$= A \frac{1}{\Gamma(3 + \nu)} \left[ _2F_1(1, 1; 3 + \nu; 1) - (1 - s) _2F_1(1, 1; 3 + \nu; 1) \right]$$

$$= \frac{\nu \sigma^2}{(2 + \nu)s} \left[ _2F_1(1, 1; 3 + \nu; 1) - (1 - s) _2F_1(1, 1; 3 + \nu; 1) \right] \tag{73}$$

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