ON RICHARD III, THE LIVINGSTONS OF CALLENDAR, AND THE CONFLICT BETWEEN GENEALOGY AND GENETICS 1.0.0

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A skeleton excavated at the presumed site of the Grey Friars friary in Leicester in 2012 is almost certainly that of the English king, Richard III (1452 -1485), and mtDNA (which is passed from mother to child) extracted from the skeleton matches mtDNA taken from descendants of Richard's sister Anne of York. However *Y-DNA* (which is passed from father to son) extracted from the skeleton apparently *doesn't match* Y-DNA taken from descendants of Henry Somerset the 5th Duke of Beaufort, who according to history descended from Richard's 2nd great grand father Edward III (1312 - 1377). The implication according to geneticists, and the media, is that there is a 'false paternity event' somewhere between Edward and the Somersets. Also, the false paternity events don't end there, for only 4 of these 5 Somerset descendants match each other. And it may be worse even than this: the patrilineal line of a Frenchman named Patrice de Warren apparently traces back to Richard III through the illegitimate son of Edward III's 4th great grandfather, Geoffrey Plantagenet, Count of Anjou (1113 - 1151), but de Warren's Y-DNA doesn't match that of either Richard III *or* any of the Somersets. In this note, a formula for calculating the time of the most recent common ancestor is introduced, and some of its consequences outlined. This formula arises from a mathematical framework within which it is possible that the traditional genealogy is correct, and that Geoffrey Plantaganet was the father of a male line incorporating Richard III, all 5 Somersets, and Patrice de Warren.

PART I

DNA COPYING ERRORS

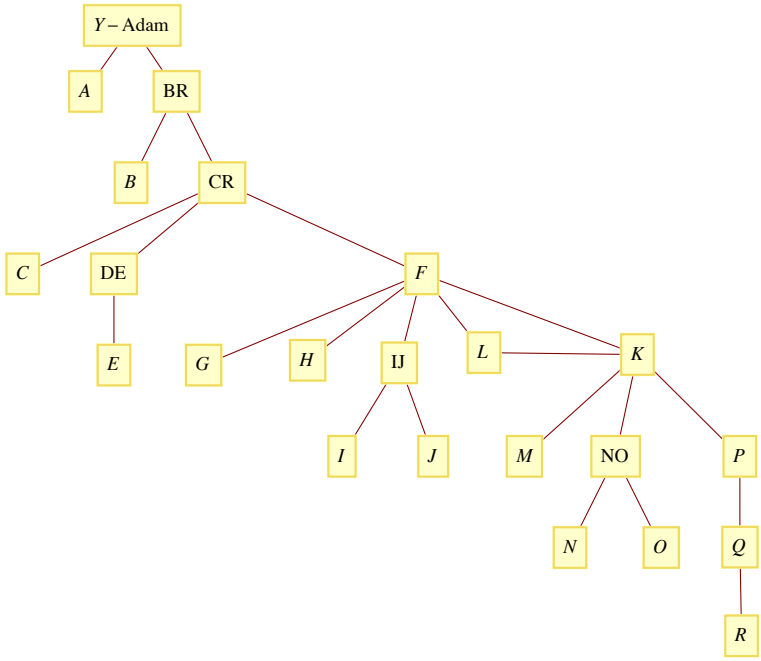
Deoxyribonucleic acid -the molecule that encodes the genetic information- can be visualized as a staircase whose steps are formed by pairs of molecules called 'bases' (there are four types of bases - Adenine, Cytosine, Guanine, Thymine (or A, C, G, and T)). This structure is copied from parent to child, and in the process is the subject yo copying errors called 'mutations'. These errors are of two types, Unique Event Polymorphisms (UEPs), and Short Tandem Repeats (STRs). The first, as the name suggests, occur very rarely, and involve either the deletion of a base, the replacement of one base by another, or the insertion of a base. The replacement of one base by another -simply the swapping of a pair of letters- is known as a Single Nucleotide Polymorphism (SNP), and this is by far the most common kind of UEP. It is so common that 'SNP' is often used in place of 'UEP'. STRs occur much more frequently than SNPs, and they involve repeated motifs of 2 - 6 bases. The regions on the DNA where these repetitions occur are known as markers, and they have names like DYS393. In the Y-DNA configurations displayed below, there is a repetition of the bases AGAT of 13 times in 5 cases, and 14 times in the other, at the location DYS393.

Supplementary Table 3: Results of Y-chromosome typing. R1b-U152* = R1b-U152 (xM160, M126, L2, Z192, Z36, Z56).

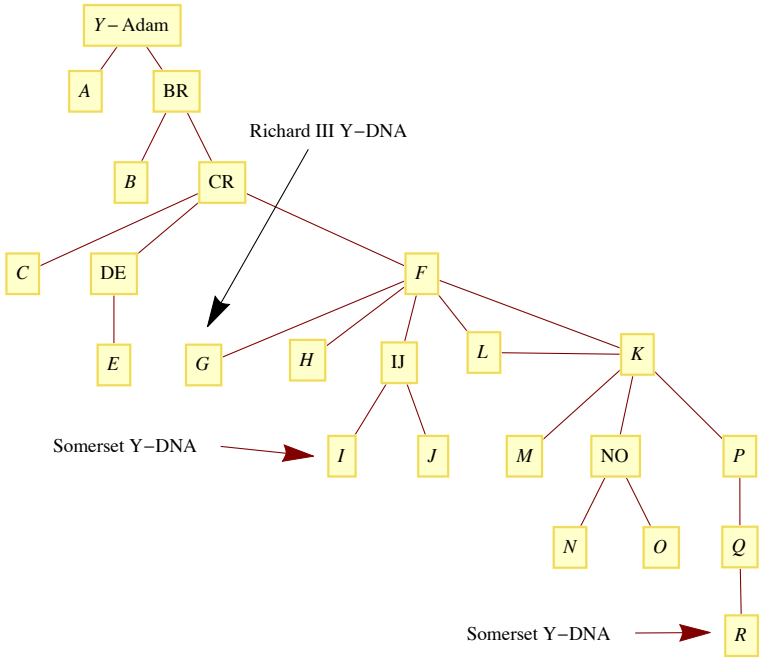
Sample Name	DYS19	DYS385	DYS385II	DYS389 I	DYS389 II	DYS390	DYS391	DYS392	DYS393	DYS437	DYS438	DYS439	DYS448	DYS456	DYS458	DYS635	YGATAH4	DYS481	DYS533	DYS549	DYS570	DYS576	DYS643	Haplogroup
Som1	14	11	14	13	29	23	11	13	13	14	12	12	20	15	18	23	12	22	12	13	19	19	10	R1b-U152*
Som2	14	11	14	13	29	23	11	13	13	14	12	12	20	15	18	23	12	22	12	13	19	18	10	R1b-U152*
Som4	14	11	14	13	29	23	11	13	13	14	12	12	20	15	18	23	12	22	12	13	18	19	10	R1b-U152*
Som5	14	11	14	13	29	23	11	13	13	14	12	12	20	15	18	23	12	22	12	13	18	19	10	R1b-U152*
Som3	15	12	15	14	30	23	10	11	13	14	10	11	18	14	20	21	10	26	13	12	17	17	12	I-M170 (xM223, M253)
Sk1	15	13	14	13	30	22	10	11	14	16	10	12	22	15	18	21	11	21	10	12	16	15	12?	G2-P287

SNPs are supposed in general not to change or to cease to exist - in theory, you either have them or you don't, and if you have them you got them from your parent, who got them from their parent... way back to the very first person who had the SNP mutation. Individual Y-DNA configurations - 'haplotypes' - can be classified into groups -'haplogroups' -according to the number of SNPs, and these groups are taken to form an alphabetical tree which grows in a simple-to-complex direction.

Closest to the root of this tree is the haplogroup that has undergone the least SNPs, and from here we supposedly proceed in a strictly simple-to-complex direction to the top of the tree and the haplogroup that has undergone the *most* SNPs (these trees are being continually modified, but all of the modifications involve the general assumptions to be challenged here).



Presumably then, there are two complimentary ways to look at the question of whether Edward III is a shared ancestor of Richard III and the Somersets. Assuming a certain mutation rate for SNPs ($\frac{1}{10^9}$ per base pair per generation), Richard Y-DNA belongs to the wrong haplogroup -it is on the wrong branch of the Y-DNA tree- for he and the Somersets to share an ancestor who lived as recently as Edward III.



And assuming a certain mutation rate for STRs (1 mutation per marker per 500 generations), this number of mismatched markers is *too great* for Richard and the Somersets to share an ancestor who lived as recently as Edward III.

Richard III	14	22	15	10	13	14	12	13	11	30	18	16	22	11	15	15	16	10	21	21	10	12	12
Somerset 1	13	23	15	10	12	15	11	14	11	30	20	14	18	10	14	17	17	10	21	26	13	12	12
Somerset 2	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	19	12	23	22	12	13	10
Somerset 3	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	18	19	12	23	22	12	13	10
Somerset 4	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10
Somerset 5	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10

But how much confidence should we have in these mutation rates, and in the assumption that they were always thus? After all, they are based on observations made in the tiny window of the near-present, and extrapolated on faith to the unobserved and vast window of the distant-past. Imagine a wheel that turns at a seemingly constant rate within the context of the present, but in fact turns ever more slowly in the direction of the past. The assumption that the wheel turned in the past as it turns in the present results in an overestimation of the total number of revolutions undergone by the wheel. If genealogy rather than genetics is right when it comes to the relationship between Richard III and the Somersets, then one way to account for the discrepancy between the two is with the idea that nature's clock is just such a wheel. The significance of Richard III's DNA for this question, and for genetic genealogy generally, is that (at the time of writing) his is the only ancient genome belonging to someone with identifiable living relatives to be sequenced. We are provided then with an opportunity to make a Y-DNA comparison between family members separated in time by 5 centuries, and but for the example of Richard III, we are -from an empirical point of view- speculating as to what such a comparison might reveal.

PART II

THE Y-DNA OF THE LIVINGSTONS OF CALLENDAR

Richard III's Y-DNA presents us with a conflict with genealogy and genetics: genealogy says one thing -that Richard and the modern Somersets are descended from Edward III- but genetics says -or at least seems to say- another. A good example of the conflict between genealogy and genetics is to be found by studying data collected by the *Livingston/-MacLea DNA Project*. Genealogy tells us that no less than 5 sets of Livingston men in the DNA project descend from a family known as 'The Livingstons of Callendar' prominent in Scotland, from the 13th to the 18th century. The surname is derived from the village to the southwest of Edinburgh, named after a man called 'Leving' ('Levings's Town') who settled in this area sometime during the reign of King Edgar (1097 - 1107). The trouble is that DNA-analysis seems to show that the MRCA of these supposed lowland Livingstons lived much earlier than Leving. If the Y-DNA mutation rates given above apply, not merely to the present time, but to *all times*, then either one of more of these family traditions are mistaken, or rogue male chromosomes lurk somewhere in the lowland Livingston past. Here is size of the problem: there are at least than 5 sets of Livingston Y-DNA, some of whose owners have excellent genealogical reasons to believe that they derive from an unbroken line of Livingston men leading back to by Andrew de Livingston (1240 - 1297), but none of whom match according to Y-DNA mutation rates observed in the present. Some of the mismatches are such that the TMRCA is 10s of 1000s of years ago.

Let

$g =$ length of a generation

$n =$ number of STR mutations

$m =$ sum of mutation rates

TMRCA = time the most recent common ancestor

and we have the simple formula

$$\text{(f1) Present year} - \frac{g\,n}{2\,m} = \text{TMRCA}$$

which broadly captures the way in which the TMRCA is usually computed. Applying *(f1)* to the 3 Callendars that at least share the same haplogroup, we arrive -on the assumption of a mutation rate of 0.002- at TMRCAs of 189, 415 BC, and 215 BC:

Callendar 3:	13	24	14	10	11	14	12	12	12	13	13	29	16	9	9	11	11	22	15	19	27	15	15
Callendar 4:	13	24	14	10	11	15	12	12	12	13	13	29	16	9	10	11	11	25	15	19	29	15	15

17	17	11	11	20	23	17	15	20	17	36	38	12	12
17	17	11	11	19	23	16	15	17	18	36	39	12	12

$$2017 - \frac{30 \times 9}{2 \times 0.074} = 192.676$$

Callendar 3:	13	24	14	10	11	14	12	12	12	13	13	29	16	9	9	11	11	22	15	19	27	15	15
Callendar 5:	13	24	15	11	11	14	12	12	12	13	13	29	17	9	10	11	11	24	15	19	31	15	15

17	17	11	11	20	23	17	15	20	17	36	38	12	12
16	17	11	11	19	23	15	15	19	18	35	38	12	12

$$2017 - \frac{30 \times 12}{2 \times 0.074} = -415.432$$

Callendar 4:	13	24	14	10	11	15	12	12	12	13	13	29	16	9	10	11	11	25	15	19	29	15	15
Callendar 5:	13	24	15	11	11	14	12	12	12	13	13	29	17	9	10	11	11	24	15	19	31	15	15

17	17	11	11	19	23	16	15	17	18	36	39	12	12
16	17	11	11	19	23	15	15	19	18	35	38	12	12

$$2017 - \frac{30 \times 11}{2 \times 0.074} = -212.73$$

□	Callendar 3	Callendar 4	Callendar 5
Callendar 3	□	192	−415
Callendar 4	192	□	−212
Callendar 5	−415	−212	□

It is thus impossible that any more than 1 of these men is a *bona fide* Callendar Livingston because as indicated the eponymous founder the Callendar Livingstons lived during the reign of Edgar (1097 - 1107). The time-scales naturally blow out *much further* if we consider the Callendar Livingstons that *don't* share the same haplogroup.

A more sophisticated formula than *(f1)* is

$$\text{(f2) Present year} - \pi\left(\frac{g\,n}{2\,m}\right) = \text{TMRCA}$$

It is by the repetition of SNPs that STRs are created and, just as we have a distinction between the unique and the repetitive elements of DNA, we have a more fundamental distinction between the unique and the repetitive elements of the number line. As every school child knows, a prime number is an integer that is divisible only by 1 and itself. Less well known perhaps is a result called the Fundamental Theorem of Arithmetic which tells us that every integer is either prime or composed of repeated primes. The symbol π in the formula stands for a function that gives the number of prime numbers not greater than some integer. For example, there are 25 primes not greater than 100, 168 not greater than 1000 and so on. If we apply *(f2)* to the data, we get an entirely different chronology, and one that -whether by accident or design- is in extremely close agreement with the genealogy. Callendar 3

...has a family tree connecting him to the well-known Livingstons of Clermont in New York, via John Livingston, the son of Robert Livingston the "third lord" of Clermont, who was in turn grandson of Robert Livingston the "first lord".

Callendar 4

...is said to descend from the Livingstons of Clermont via two sons of "Robert of Clermont", 3rd lord of the manor, Robert "Cambridge" Livingston (line carrying the Livingston paternal DNA), and John "of Oak Hill" Livingston (through two of his grand-daughters).
The great-grandfather of the participant was Col. Charles Edward Livingston of Red Hook, New York, who served with the Union N.Y. 76th Infantry Volunteers during the American Civil War. He was the son of Robert Francis Livingston, a Surveyor and Civil Engineer, who, in turn, was the son of Robert Swift Livingston. Robert Swift Livingston was the son of Robert "Cambridge" Livingston and Alice Swift.

The MRCA of Callendar's 3 and 4 is according to this information Robert Livingston 3rd Lord of Clermont Manor 1708 - 1790, and *(f2)* and a 37 marker comparison gives us a TMRCA 1733 using the same assumptions that produced the times above.

$$2017 - \pi\left(\frac{30 \times 9}{2 \times 0.074}\right) = 1736$$

Note that while changing g or m, and modifying other assumptions, changes the predicted times dramatically, *(f2) always* predicts a common ancestor who lived much more recently than the time predicted by *(f1)*.

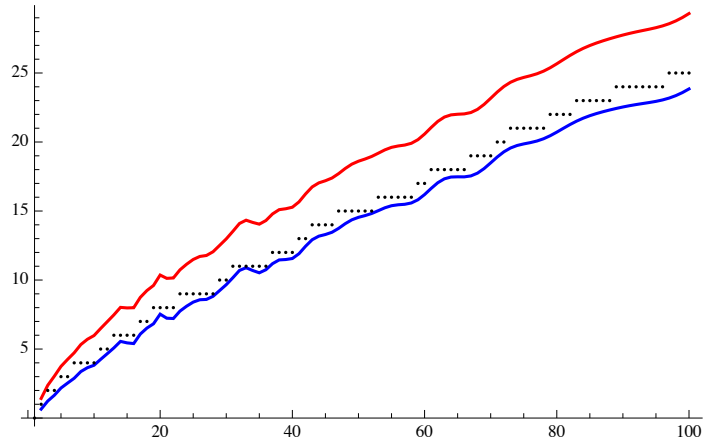
We can be refine *(f2)* on the grounds that $\pi(n)$ lies between the sums

$$\sum_{n=2}^{x} \frac{1}{H_n} - 2\,\mathrm{Re}\left(\sum_{n=1}^{\infty} \mathrm{Ei}(\rho_{-n}\log(x))\right)$$

and

$$\sum_{n=2}^{x} \frac{1}{\log(n)} - 2\,\mathrm{Re}\left(\sum_{n=1}^{\infty} \mathrm{Ei}(\rho_{-n}\log(x))\right)$$

Graphically



This gives us

$$\text{(f3) Present year} - \sum_{n=2}^{\frac{g\,n}{2\,m}} \frac{1}{\log(n)} <= \text{TMRCA} <= \text{Present year} - \sum_{n=2}^{\frac{g\,n}{2\,m}} \frac{1}{H_n}$$

The graph above depicts several interlocking ideas:

- Globally speaking, prime-density decreases as a function of arithmetic increase.
- Local fluctuations in density -which involve either a random increase or a decrease in prime-density- decrease as a function of arithmetic increase.
- There are limits on both the global rate of decrease and on the size of the local fluctuations.

$\pi(x)$ is what is called a 'step function', and it has been set up so that the jump from one step to another takes place only when a prime number appears in the number line. As we travel down the line, we find that these jumps become rarer and rarer, for the primes thin out. Despite this global decrease in prime-density, there are local irregularities. The red and blue colored staircase-curves keep the slope of the central staircase from being either impossibly steep (steeper than the red staircase) or impossibly gentle (less steep blue than the blue staircase). This means that they serve as criteria by reference to which it can be judged whether a change to the steepness of the central staircase represents a local decrease or rather a local *increase* in prime-density. If we identify prime-density with genetic simplicity, it follows that while the global direction of the Y-DNA tree is toward the upper bound and further genetic complexity, any local direction may with equal probability be toward either bound. If we accept the proposal that the mutation rates of SNPs correspond to the primes in the manner indicated by *(f2)* and by *(f3)*, we must reject the assumption that genetic mutations occur at a constant rate *and* the assumption that they always follow the one-way simple-to-complex direction of the ISOGG Y-DNA tree.

Applying *(f3)* to the Calendar 3 and 4 data:

$$\frac{30 \times 9}{2 \times 0.074} = 1824.32$$

$$2017 - \sum_{n=2}^{1824.32} \frac{1}{\log(n)} = 1725.66$$

$$2017 - \sum_{n=2}^{1824.32} \frac{1}{H_n} = 1752.43$$

Callendar 5 is 'thought to be related' to Philip 'the Signer' Livingston 1716 - 1778. Philip's father -Philip Livingston 2nd Lord of the Manor (1686 - 1749)- was also the father of Robert Livingston 3rd Lord of the Manor. The MRCA of Callendars 3, 4, and 5 could have lived no earlier than Robert Livingston 1st Lord of the Manor 1654 - 1728.

$$\frac{30 \times 12}{2 \times 0.074} = 2432.43$$

$$2017 - \sum_{n=2}^{2432.43} \frac{1}{\log(n)} = 1646.28$$

$$2017 - \sum_{n=2}^{2432.43} \frac{1}{H_n} = 1678.61$$

$$\frac{30 \times 11}{2 \times 0.074} = 2229.73$$

$$2017 - \sum_{n=2}^{2229.73} \frac{1}{\log(n)} = 1672.46$$

$$2017 - \sum_{n=2}^{2229.73} \frac{1}{H_n} = 1702.98$$

□	Callendar 3	Callendar 4	Callendar 5
Callendar 3	□	1725 – 1752	1646 – 1678
Callendar 4	1725 – 1752	□	1672 – 1702
Callendar 5	1646 – 1678	1672 – 1702	□

The basic idea behind *(f2)* and *(f3)* is that the relationship between time and genetic mutations can be modelled on that between arithmetic increase and changes in prime-density. More particularly, the idea is that genetic simplicity corresponds to prime-density, and genetic complexity to prime-sparsity. Because there are relatively fewer primes as one counts away from unity, *(f2)* works by either by increasing Y-DNA mutation rates or decreasing the length of a generation in the direction of the past, depending on one's point of view. If we look back in time on the assumption that a generation has constant length, we find that to get the right TMRCA without the benefit of $\pi(x)$ we need to allow *m* to increase dramatically. Equivalently, if we look back in time on the assumption that mutation rates are constant, we find that to get the right TMRCA without the benefit of $\pi(x)$ we need to allow the length of a generation to decrease. Using the example of Callendars 3 and 4, whose MRCA lived as indicated between 1708 - 1790:

$$\frac{30 \times 9}{2 \times 0.074} = 1824.32$$

$$2017 - \sum_{n=2}^{1824.32} \frac{1}{\log(n)} = 1725.66$$

$$2017 - \left(\frac{30 \times 9}{2 \times 0.463379} \right) = 1725.66$$

$$2017 - \left(\frac{4.79092 \times 9}{2 \times 0.074} \right) = 1725.66$$

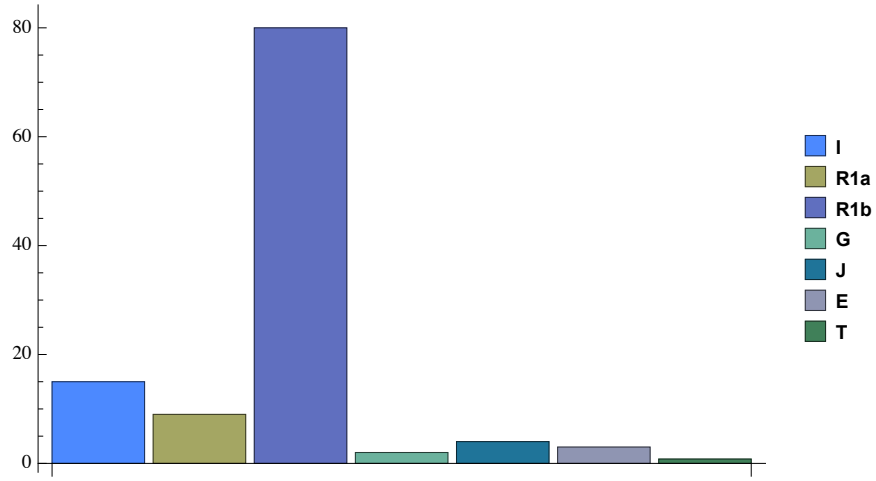
And it will prove important to consider also that the same thing can be accomplished by allowing 2 to vary while the other values remain constant:

$$2017 - \left(\frac{30 \times 9}{12.5237 \times 0.074} \right) = 1725.66$$

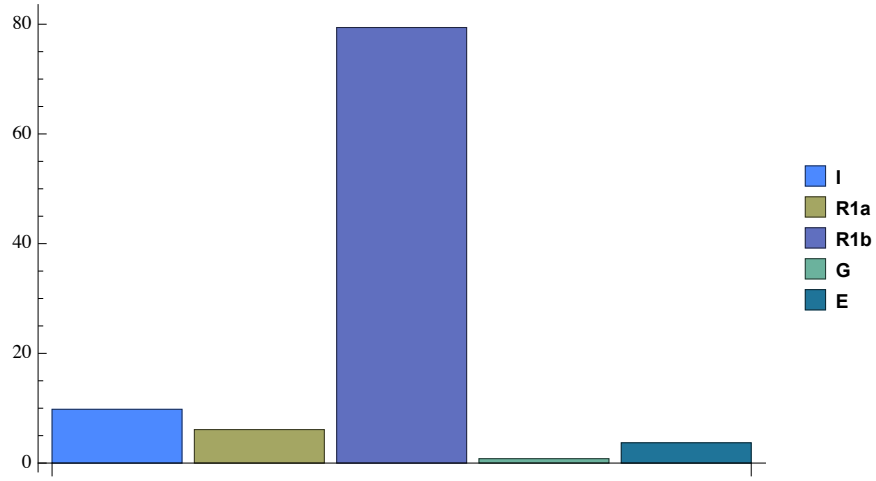
PART III

In 1998 a DNA study by Foster et al. effectively validated rumours that Thomas Jefferson fathered children by his slave Sally Hemmings, when the haplotypes of one of TJ's great nephews was found to match that of the descendant of Sally Hemmings' youngest son. The result was especially convicting since the haplogroup -T-was a rare one (T is a sub group of K which is present at low levels throughout the world, particularly in the Near East). It was at first puzzling that British Y-DNA should belong to such a rare and Near Eastern-seeming lineage, but it was found at the time that the haplotypes of 2 of 85 British Jeffersons belonged to T. This proportion is mimicked by the *Livingston/-MacLea Y-DNA Project* in respect of G rather than T, and on the surface at least, this data-set resembles the British average.

Approximate average distribution of Y-DNA in Britain:



Livingston/MacLea:



The majority of haplotypes in British Y-DNA surname projects belong to the youngest haplogroup (R1b), some belong to European haplogroups such as I, and R1a, and a tiny minority belong to haplogroups such as G and T. This is a quite pointless exercise if evolutionary chronology is correct (and the mutation rate of SNPs is a constant $\frac{1}{10^9}$ per base pair per generation or similar), but running a comparison between the older and the younger haplotype with *(f2)* or *(f3)* inevitably reveals a MRCA who lived in the ball park of the time they were must have lived if the two men are related in recent times. Here is the Livingston example:

Callendar 1 (G2a):	14	22	15	10	14	16	11	14	11	12	11	29	16	9	9	11	11	22	16	21	30	12	13
Callendar 3 (R1b):	13	24	14	10	11	14	12	12	12	13	13	29	16	9	9	11	11	22	15	19	27	15	15

13	14	11	11	20	20	13	15	17	18	37	37	11	10
17	17	11	11	20	23	17	15	20	17	36	38	12	12

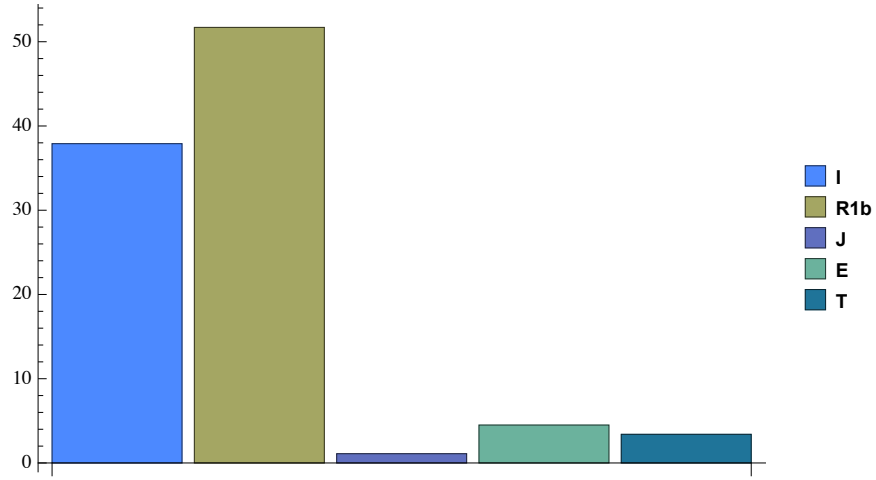
$$2017 - \frac{30 \times 26}{2 \times 0.075} = -3183$$

$$\frac{30 \times 26}{2 \times 0.075} = 5200.$$

$$2017 - \sum_{n=2}^{5200.} \frac{1}{\log(n)} = 1309.48$$

$$2017 - \sum_{n=2}^{5200.} \frac{1}{H_n} = 1363.93$$

Another example can be drawn from data on file at the *Carson Y-DNA Project*. The Carsons are an ancient family from the Dumfries and Galloway area of Scotland, who branched out to Northern Ireland, and to America. The earliest recorded spelling of the name is from a charter signed in 1276 by a cleric named 'Robert de Carson'. According to ancestry.com, 'Carson' is probably a variant of 'Curzon,' which is a variant of Courson, in which case the first British 'Carson' may have been Robert de Courson, a knight from Notre Dame de Courson in the Normandy region of north-western France. This possibility is bolstered by the fact that William de Ferrers (*c* 1240 - 1287), acquired lands in Dumfries/Galloway between 1251 and 1256, 20 - 25 years prior to the appearance of Robert de Carson in the local record books - the Ferrers' were the Courson's overlords in France, and both Henry de Ferrers (1036 - 1088) and Robert de Courson fought alongside William at the Battle of Hastings...



A majority of haplotypes in the project belong again to R1b, a number belong to I, and there are 3 closely matching T haplotypes. Running a comparison between an R1b haplotype and one of the T haplotypes with *(f3)*:

Carson 1 (T):	13	24	13	10	13	16	11	11	12	15	13	31	16	9	9	11	12	25	15	18	33	11	14
Carson 2 (R1b):	13	24	14	11	11	14	12	12	12	14	13	30	17	9	10	11	11	25	15	18	31	15	15

14	16	10	10	23	24	14	13	17	18	32	37	13	9
17	17	11	11	19	23	16	15	18	18	36	38	11	12

$$\frac{30 \times 26}{2 \times 0.075} = 5200.$$

$$2017 - \sum_{n=2}^{5200.} \frac{1}{\log(n)} = 1309.48$$

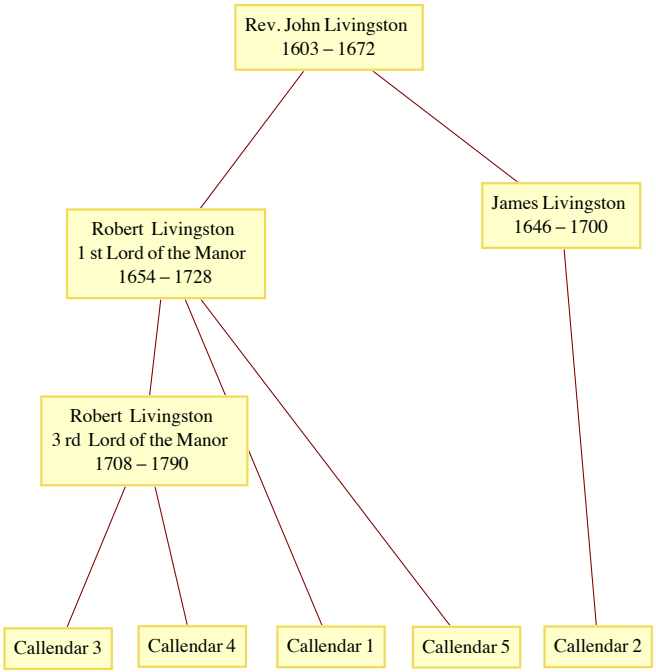
$$2017 - \sum_{n=2}^{5200.} \frac{1}{H_n} = 1363.93$$

In the *light* of *(f3)*, we find that it is mathematically possible that *all 5* Callendars are -regardless of their haplogroups- from the Livingston family traceable to Andrew de Livingston (1240 - 1297):

□	Callendar 1	Callendar 2	Callendar 3	Callendar 4	Calledar 5
Callendar 1	□	1251 – 1309	1298 – 1353	1204 – 1265	1204 – 1265
Callendar 2	1251 – 1309	□	1322 – 1375	1369 – 1420	1466 – 1510
Callendar 3	1298 – 1353	1322 – 1375	□	1729 – 1756	□
Callendar 4	1204 – 1265	1369 – 1420	1722 – 1749	□	1669 – 1699
Callendar 5	1204 – 1265	1466 – 1510	1643 – 1675	1669 – 1699	□

But whilst the TMRCAs offered by *(f3)* fit the genealogical data far better than those offered by *(f1)*, they nonetheless fail to fit it in a way that fully agrees with genealogy. Robert Livingston 1st Lord of Clermont Manor (1654 - 1728) was a descendant of the Callendar Livingstons via his father Reverend John Livingston, a direct descendant via the make line of Andrew de Livingston. It was Robert who established the Livingston family in New York, following the exile of his father from Scotland to the Netherlands for refusing to swear allegiance to Charles 11. Callendar 1 -whose Y-DNA is the same as Richard III's- is described by the administrators at the *Livingston/MacLea Y-DNA Project* as having the 'best paper trail' claim to be a direct descendant of the Livingstons of Clermont Manor, the estate founded by Robert the 1st Lord, and hence of the Livingstons of Callendar. The G haplotype is 'extremely unusual at DYS388 and was in fact not recognized as such by Family Tree DNA's software... if aristocratic families often have unusual Y DNA, either because they come from a very old line, or because they come from a very geographically distant line, then this is such a DNA signature.' William Cutter in the *Genealogical and Family History of Central New York* writes that the ancestor of Callendar 2 is 'said by good authorities' to descend from Reverend John Livingston via Robert's elder brother James Livingston (1662 - 1700). As indicated, Callendars 3 and 4 both have strong claims descend from Robert Livingston the 3rd Lord of the Manor (1705 - 1790). Callendar 5 is thought to be related to Philip 'The Signer' Livingston (1716 - 1778), a son of the 2nd Lord.

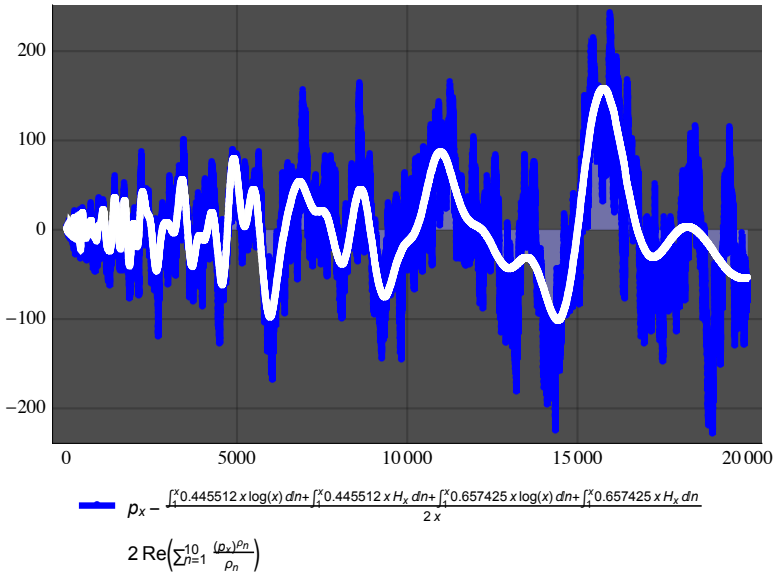
The kind of family tree the DNA test results should support -but apparently do not- is this one:



Reverend John Livingston -the man said by genealogy to be the MRCA of all 5- lived between 1603 and 1672, and so while Callendars 3, 4, 5 have a MRCA that lived around the time he is supposed to have lived have, this is not true of Callendars 1 and 2. This is because the haplogroups of Callendars 1 and 2 differ from those of Callendars 3, 4, 5, which introduces many more mismatched markers. But in spite of any superiority over *(f1)* and *(f2)*, *(f3)* is yet a very blunt tool, and it is easy to see that it will fail to give the TMRCA of distantly related haplotypes, and that it will generally yield inaccurate results when there are but a few mismatched markers. So let's continue to suppose that the genealogies *are right*, and see if there is a mathematically clear way to sharpen the tool sufficiently to bring the desired unity to genealogy and genetics.

THE UNCERTAINTY PRINCIPLE

It is evident that we can locate the primes by summing waves of various kinds:



In the example above, we are pushing and pulling the blue coloured waves arising from the difference between p_x and

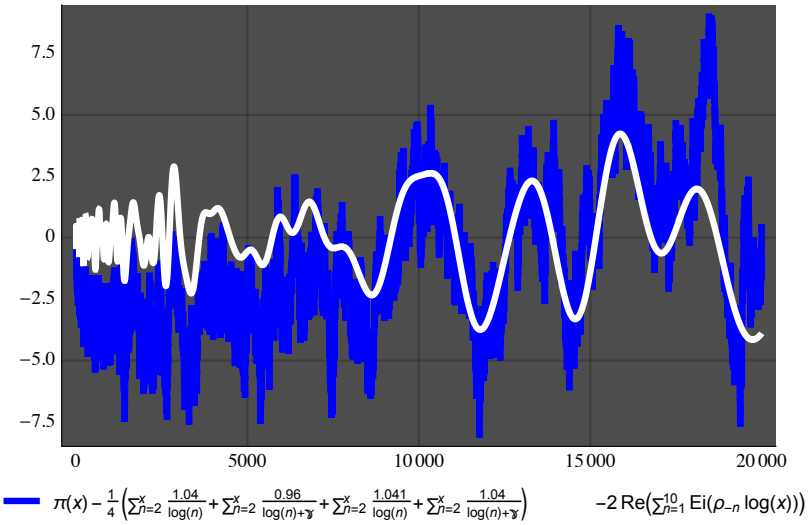
the simulating function $\frac{1}{2x}\left(\int_1^x 0.445512\, x\, H_x\, dn + \int_1^x 0.657425\, x\, H_x\, dn + \int_1^x 0.445512\, x \log(x)\, dn + \int_1^x 0.657425\, x \log(x)\, dn\right)$ so that they have increasingly taken on the shape of the white coloured waves corresponding in the limit to the superposition

$$2\,\mathrm{Re}\left(\sum_{n=1}^{\infty}\frac{(p_x)^{\rho_n}}{\rho_n}\right)$$

This matching process is to be accomplished by adding functions of the form $a_n\, x \log(x)$ and $a_n\, x\, H_x$ to the function

$$\frac{\int_1^x a_n\, x\, \log(x)\, dn + \int_1^x a_n\, x\, H_x\, dn + \int_1^x a_n\, x\, \log(x)\, dn + \int_1^x a_n\, x\, H_x\, dn \ldots}{n\, x}$$

Here is a simpler perspective on the same phenomenon:



Here we are pushing and pulling the blue waves in the graph above so that they increasingly take on the shape of the white waves corresponding to the superposition

$$-2\,\mathrm{Re}\sum_{n=1}^{\infty}\mathrm{Ei}(\rho_{-n}\,(\log x))$$

This pushing and pulling occurs as functions of the form $\sum_{n=2}^{x}\frac{a_n}{\log(n)}$ and $\sum_{n=2}^{x}\frac{a_n}{H_n}$ are added to the function

$$\frac{1}{n}\left(\sum_{n=2}^{x}\frac{a_n}{\log(n)} + \sum_{n=2}^{x}\frac{a_n}{H_n} + \sum_{n=2}^{x}\frac{a_n}{\log(n)} + \sum_{n=2}^{x}\frac{a_n}{H_n} + \ldots\right)$$

As appropriately weighted functions are added, the accuracy of the simulation grows. However these functions are associated with waves that lack a constant length (waves associated with spirals), and the accuracy of the simulation depends on allowing the unit numerators in our prime-counting functions to fluctuate, i.e. to take on values *other* than 1.

In every act of quantification there is a counted-progression and a counting-progression, and prime-counting functions artificially increase or decrease the balance of prime-density (simplicity and complexity/uniqueness and repetition) possessed by the counting progression. Take Riemann's prime-counting function $R(x) = \sum_{n}^{\infty}\frac{\mu(n)\,\mathrm{li}\left(x^{1/n}\right)}{n}$ for example. Despite any appearances to the contrary, it possesses limited accuracy - for sufficiently large x, it is no more accurate than $\sum_{n=2}^{x}\frac{1}{\log(n)}$ and/or $\int_2^x\frac{1}{\log(n)}\,dn$). The reason for the illusion is that the accuracy of *R(x)* for small *x* derives from compensations for the early *inaccuracy* of the logarithmic counting/simulation functions: it maintains its accuracy only whilst the number line is relatively prime-dense (as it is in the surveyable portions thereof) when the Moebius function μ(x) is may be used to boost the prime-density of the counting progression by eliminating non-square free integers.

$$\begin{aligned}\mu(x) = \{\,&0 \;=\; x \text{ has } a \text{ repeated factor},\\ &1 = x \text{ has an even number of distinct factors},\\ &-1 = x \text{ has an odd number of distinct factors}\}\end{aligned}$$

When these compensations are no longer necessary -when the number line is *prime-sparse* and $\sum_{n=2}^{x}\frac{1}{\log(n)}$ and is quite accurate- then the Moebius-based accuracy of *R(x)* will inevitably be lost. This is why we need the kind of prime-

counting function represented by $\frac{1}{n}\left(\sum_{n=2}^{x}\frac{a_n}{\log(n)}+\sum_{n=2}^{x}\frac{a_n}{H_n}+\sum_{n=2}^{x}\frac{a_n}{\log(n)}+\sum_{n=2}^{x}\frac{a_n}{H_n}+...\right)$ for, unlike Riemann's function, this kind of function contends with every possible state of prime-density that can be encountered on a number line... There is an uncertainty principle here that can be stated in this way: there is a square-root sized inaccuracy effecting the determination of the prime-density of a counted unit n and a counting unit n^* (https://figshare.com/articles/On_a_Question_Concerning_the_Littlewood_Violations_pdf/4240424).

That we must adopt a similar approach to time-counting as to prime-counting in order that *(f3)* have universal application can be shown by reference to the example of two members of the T Carson male line, that differ by 1 marker out of 37.

Carson 1 (T):	13	24	13	10	13	16	11	11	12	15	13	31	16	9	9	11	12	25	15	18	33	11	14
Carson 2 (T):	13	24	13	10	13	16	11	12	12	15	13	31	16	9	9	11	12	25	15	18	33	11	14

14	16	10	10	23	24	14	13	17	18	32	37	13	9	
14	16	10	10	23	23	14	13	17	18	32	37	13	9	

Given a mutation rate of 0.002, *(f2)* might predict a TMRCA between 1966 and 1974:

$$\frac{30}{2\times 0.074}=202.73$$

$$2017-\sum_{n=2}^{202.703}\frac{1}{\log(n)}=1966.58$$

$$2017-\sum_{n=2}^{202.703}\frac{1}{H_n}=1974.17$$

But the MRCA of these men must by the genealogical facts have lived no later than 1704, and possibly much earlier. By allowing the length of the numerator to vary until we hone on what we know from genealogy to be the right dates, we can quantify and compensate for any surrounding uncertainty.

$$\text{(f4) Present year}-\left(\sum_{n=2}^{\frac{g\,n}{2\,m}}\frac{\text{a1}}{\log(n)}\right)<=\text{TMRCA}<=\text{Present year}-\sum_{n=2}^{\frac{g\,n}{2\,m}}\frac{\text{a2}}{H_n}\Bigg)$$

$$2017-\sum_{n=2}^{202.703}\frac{6.20783}{\log(n)}==1704$$

$$2017-\sum_{n=2}^{202.703}\frac{7.30839}{H_n}=1704$$

Reviewing Callendars 3 and 4 with a view to achieving the greatest possible accuracy:

$$\text{MRCA = Robert Livingston }(1708-1790)$$

$$n=9$$

$$m=0.002$$

$$\frac{30\times 9}{2\times 0.074}=1824.32$$

$$2017-\sum_{n=2}^{1824.32}\frac{1.06062}{\log(n)}=1708$$

$$2017-\sum_{n=2}^{1824.32}\frac{0.858003}{H_n}=1790$$

Comparing Callendars 2 (R1a) and 3 (R1b):

Callendar 2 (R1a):	13	25	15	11	11	14	12	12	10	14	11	31	15	9	10	11	12	23	14	20	32	12	15
Callendar 3 (R1b):	13	24	14	10	11	14	12	12	12	13	13	29	16	9	9	11	11	22	15	19	27	15	15

16	16	11	11	19	20	16	18	18	18	34	38	12	11	
17	17	11	11	20	23	17	15	20	17	36	38	12	12	

$$\text{MRCA} = \text{Rev. John Livingstone } 1603 - 1672$$

$$n = 25$$

$$m = 0.002$$

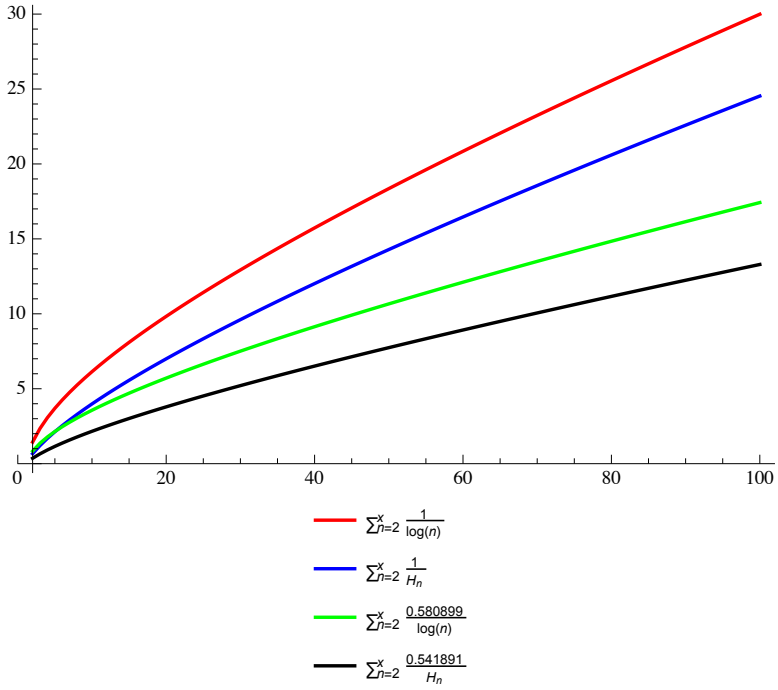
$$\frac{30 \times 25}{2 \times 0.075} = 5000$$

$$2017 - \sum_{n=2}^{5200.} \frac{0.585139}{\log(n)} = 1603$$

$$2017 - \sum_{n=2}^{5000.} \frac{0.546645}{H_n} = 1672$$

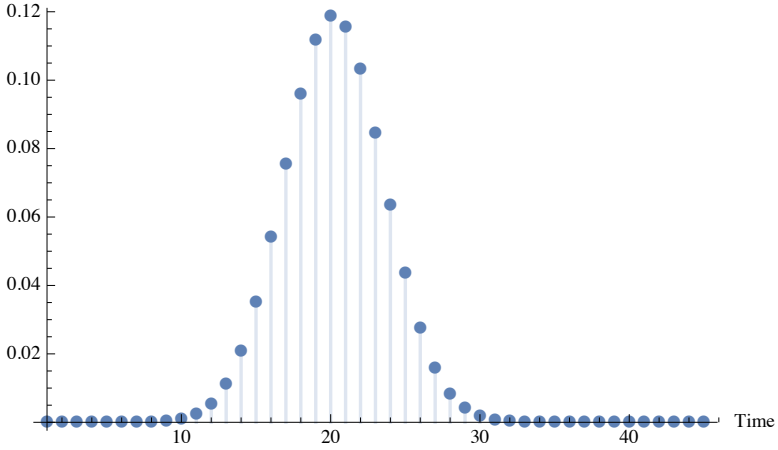
The natural objection is that we could use this shrinking and stretching technique to produce more or less *any* TMRCA we like, but this is not quite so. We have seen that by adding functions of the form $\sum_{n=2}^{x} \frac{a_n}{\log(n)}$ and $\sum_{n=2}^{x} \frac{a_n}{H_n}$ we are able to simulate π(x), from which it follows that the rigid $\pi\left(\frac{g\,n}{2\,m}\right)$ can be regarded as a sum of these more fluid sums. $\pi\left(\frac{g\,n}{2\,m}\right)$ derives from the canonical arithmetic progression 1, 2, 3... but an infinite number of prime-staircases can be generated by use of the formulation $q\,n + a$ $(n = 0, 1, 2...)$, and a result known as 'Dirichlet's Theorem' tells us that if q and a share no common factor other than 1 -if they are 'co-prime'- then the progression contains infinitely many primes. There are then prime staircases corresponding to an infinite number of progressions, each of which is constrained by a pair of staircases, each of which possesses a different slope, and climbs in a different way...

As indicated below, each of the pairs of sums $\sum_{n=2}^{x} \frac{a_n}{\log(n)}$ and $\sum_{n=2}^{x} \frac{a_n}{H_n}$ represents a pair of prime staircases, each of which possesses a certain slope, and climbs in a different way:



The fluidity of the sums associated with *(f4)* is thus constrained by their relationship to $\pi(\frac{gn}{2m})$, meaning that their are limits on how much shrinking and stretching is possible before the *wrong* TMRCA is produced.

In fact, TMRCA calculators don't give us a single date, but a wide range of times, each of which is associated with a certain probability distribution.



We have supposed for the sake of simplicity that *(f1)* corresponds to the *peak distribution*, and that this gives the most likely TMRCA. However if *(f4)* is the right formula to find the TMRCA, then in the light of its reliance on complex and negative numbers, we can say classical probability theory needs to be re-vamped in terms of these numbers, or what is the same thing, that classical probability needs to be re-vamped in terms of the idea that probabilities don't ever really add up to 1. Briefly... we can observe when a series of fair coins are flipped that the limit 1/2 is associated with a square root sized error - assuming the number of tosses forms a uniform arithmetic progression. But if we assume instead that the balance is *exactly* 1/2, then this error is transferred -without effecting the outcome of the measurement- from the balance of heads and tails to the balance of integers and coin flips. Rather than the sum

$$\frac{1}{2} + \frac{1}{2} = 1$$

this gives us

$$\left(\frac{1}{2} + m\,i\right) + \left(\frac{1}{2} - m\,i\right) = 1$$

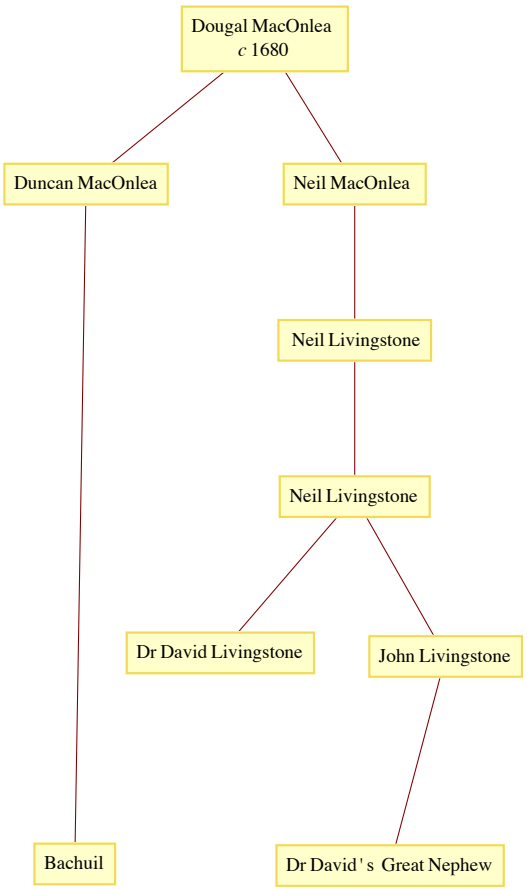
where i is $\sqrt{-1}$ and m is a square-root sized margin of error. Reflecting on the fact that

$$\left(\left(\sqrt{\frac{1}{2}}^{2} + m\,i\right) + \left(\sqrt{\frac{1}{2}}^{2} - m\,i\right) = 1\right) = \left(\frac{1}{2} + m\,i\right) + \left(\frac{1}{2} - m\,i\right) = 1$$

and given that the real part of ρ in the superpositions $2\,\mathrm{Re}\left(\sum_{n=1}^{\infty} \frac{(p_x)^{\rho_n}}{\rho_n}\right)$ and $2\,\mathrm{Re}\sum_{n=1}^{\infty} \mathrm{Ei}(\rho_{-n}(\log x))$ is always equal to $\frac{1}{2}$ (https://figshare.com/articles/On_a_Question_Concerning_the_Littlewood_Violations_pdf/4240424), we can regard be regard classical probability as a special case of quantum probability for counted and counting-progressions such that their balances of prime-density and sparsity are symmetrical.

DR LIVINGSTONE I PRESUME

The nature of mutation rates can be further revealed by reference to the genealogically distinct Livingston(e) family, that of the chiefs of the Scottish highland clan 'the Livingstones of' Bachuil'). The founders of the clan were the high priests of the ancient Gaelic over-kingdom of Dalriada (500 - 900), so called because they were the guardians of a sacred staff or 'Bachuil'. According to genealogy, Dr David Livingstone of 'Dr Livingstone I presume fame' belongs to this family. Dr David was by all accounts the 2nd great grandson of former clan chief Dougal MacOnlea, but a Y-DNA comparison of a Bachuil Livingstone and known descendant of Dougal MacOnlea, with Dr David Livingstone's great nephew made using *(f1)* gives a TMRCA of 823 BC, when genealogy says that the true TMRCA was circa 1680:



Livingstone 1 (Great Nephew):	13	24	14	11	11	13	12	12	11	13	13	29	18	9	10	11	11	25	15	19	28	14	15		
Livingstone 2 (Bachuil):	13	24	14	10	11	14	12	12	13	13	13	29	18	9	10	11	11	25	15	19	31	15	15		

16	18	11	11	19	23	17	15	18	17	35	37	12	12	11	9	15	16	8	11	10	8	10	10	12	23	23	17	10	12
17	17	11	12	19	22	15	15	17	17	36	38	12	12	12	9	15	16	8	10	10	8	10	10	12	22	23	16	10	12

12	15	8	12	22	20	13	12	11	13	11	11	12	12
12	17	8	11	22	20	13	12	11	13	11	11	12	12

$$MRCA = \text{Dougal MacOnlea } c \text{ } 1680$$

$$n = 20$$

$$m = 0.002$$

$$\frac{30 \times 20}{2 \times 0.134} = 2238.81$$

$$2017 - \sum_{n=2}^{2238.81} \frac{0.974807}{\log(n)} = 1680$$

$$2017 - \sum_{n=2}^{2238.81} \frac{1.06948}{H_n} = 1680$$

A significant consequence of *(f4)* is the possible relatedness in recent generations of men with dissimilar Y-DNA profiles. Another is the possible *non-relatedness* in recent generations of men with *similar* Y-DNA profiles. These consequences arise from the jaggedly curved shape of Y-DNA mutation rates. The first consequence we know from the case of the Livingstons of Callendar, the second consequence can be illustrated by reference to the relationship between Livingstons of Callendar and the Livingstones of Bachuil: some of the Callendar and the Bachuil Livingston(e)s appear according to *(f2)* to have a recent common ancestor:

□	Callendar 3	Callendar 4	Callendar 5
Callendar 3	□	□	□
Callendar 4	□	□	□
Callendar 5	□	□	□
Bachuil 1	1654	1683	1632

But genealogy tells us that Bachuil 1 descends from Dougal MacOnlea *b* circa 1680, and that it was not until the 18th century that his descendant John MacOnlea became the first MacOnlea to adopt the name 'Livingstone'. According to a 1743 document, Alexander Livingston Earl of Linlithgow (d 1648), a Callendar Livingston, believed that his family originated from the same line as the MacOnleas:

Lord Callender who also was Earl of Linlithgow [the date indicates that this would be Alexander, second Earl of Linlithgow] ... told him that tho' their family at that time made a figure in the world, and that tho' the McLea's in the Highlands made no great figure there, yet that their family were descended of the McLea's in the Highlands...

And so we might have to go back as far as the 6th century to find the MRCA. This is because in theory the founder of the clan was Saint Moluag *c* 530 - 592.

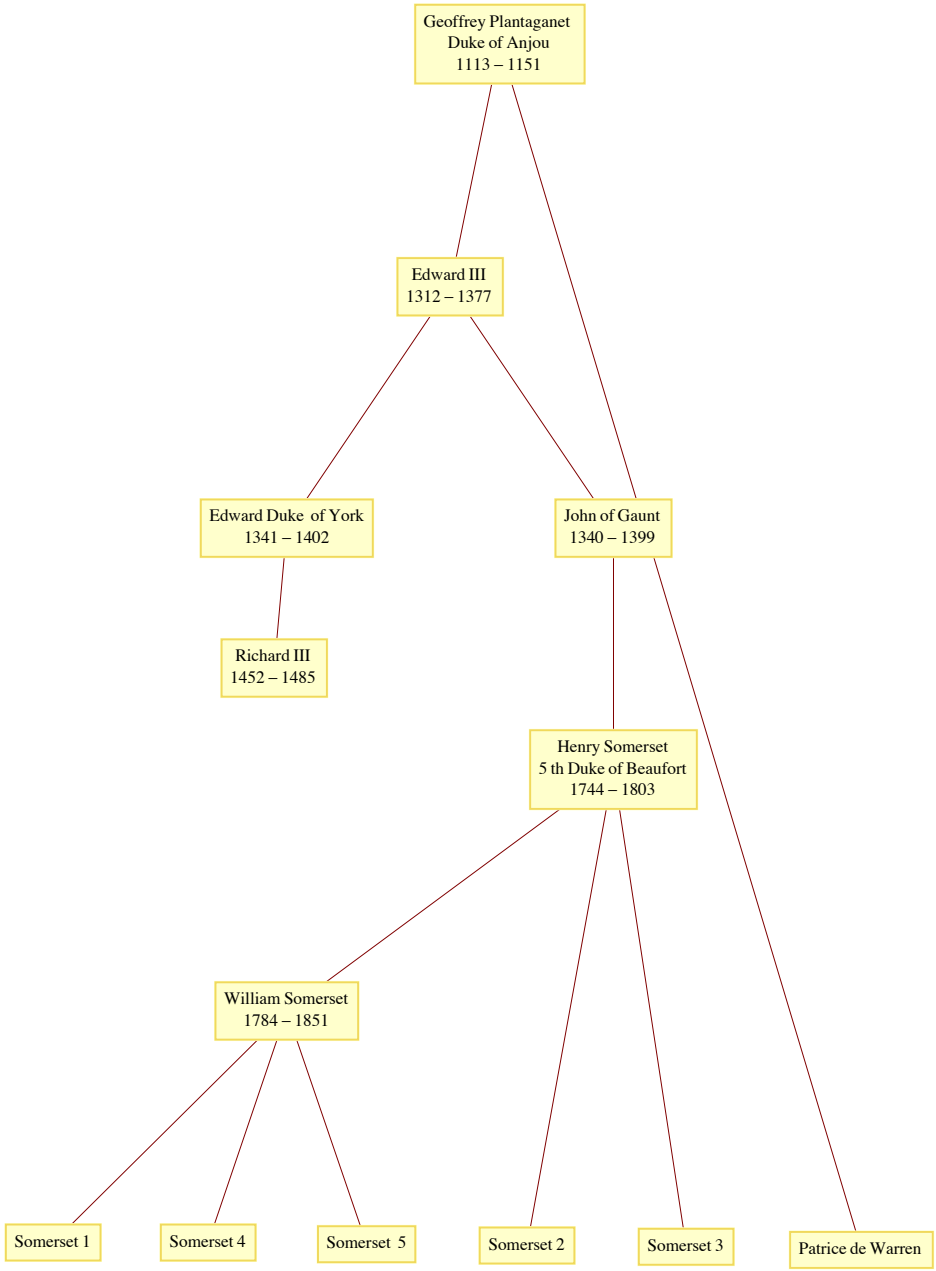
$$\frac{30 \times 13}{2 \times 0.074} = 2635.14$$

$$2017 - \sum_{n=2}^{2635.14} \frac{3.74913}{\log(n)} = 530$$

$$2017 - \sum_{n=2}^{2635.14} \frac{3.93091}{H_n} = 592$$

PART IV

THE TMRCA OF RICHARD III AND THE SOMERSETS



On next to the title conflict -Richard III v the Somersets- to which we can apply the mathematical methods outlined above. If we look at the comparison between Richard III and Somersets 2, 3 4 and 5 we see that 18 out of 23 markers are mismatched.

Richard III	14	22	15	10	13	14	12	13	11	30	18	16	22	11	15	15	16	10	21	21	10	12	12
Somerset 2	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	19	12	23	22	12	13	10
Somerset 3	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	18	19	12	23	22	12	13	10
Somerset 4	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10
Somerset 5	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10

Working with the Richard III and Somerset data (2, 3, 4 , 5) and our first modified formula -(*f2*)- we readily get an accurate TMRCA:

$$2017 - \pi\left(\frac{27 \times 18}{2 \times 0.046}\right) = 1316$$

$$\text{MRCA} = \text{Edward III}\,(1312 - 1370)$$

$$n = 18$$

$$m = 0.002$$

$$\frac{27 \times 18}{2 \times 0.046} = 5282.61$$

$$2017 - \sum_{n=2}^{5282.61} \frac{0.983129}{\log(n)} = 1312$$

$$2017 - \sum_{n=2}^{5282.61} \frac{0.9926}{H_n} = 1370$$

Somerset 1:

$$2017 - \pi\left(\frac{27 \times 15}{2 \times 0.046}\right) = 1418$$

$$2017 - \pi\left(\frac{15 \times 30}{2 \times 0.046}\right) = 1363$$

$$2017 - \pi\left(\frac{30 \times 18}{2 \times 0.046}\right) = 1244$$

$$n = 15$$

$$m = 0.046$$

$$\frac{30 \times 15}{2 \times 0.046} = 4891.3$$

$$2017 - \sum_{n=2}^{4891.3} \frac{1.05023}{\log(n)} = 1312$$

$$2017 - \sum_{n=2}^{4891.3} \frac{1.03372}{H_n} = 1377$$

SOMERSET 1

Somerset 1 doesn't match Somersets 2, 3, 4, and 5 in the traditional sense of the term 'match', prompting geneticists to appeal again to the 'false paternity event'. Somersets 2, 3, 4, and 5 match closely - there are at most 2 mismatched markers out of 23 between them:

Somerset 2	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	19	12	23	22	12	13	10
Somerset 3	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	18	19	12	23	22	12	13	10
Somerset 4	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10
Somerset 5	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10

In the case of the 1 marker mismatch, we can hone in on their supposed MRCA Henry Somerset (1744 - 1803) in this way:

$$\text{Henry Somerset } 1744 - 1803$$

$$n = 1$$

$$m = 0.002$$

$$\frac{30 \times 1}{2 \times 0.046} = 326.087$$

$$2017 - \sum_{n=2}^{326.087} \frac{3.75507}{\log(n)} = 1744$$

$$2017 - \sum_{n=2}^{326.087} \frac{3.39641}{H_n} = 1803$$

Somersets 2, 3, 4 and 5 differ from Somerset 1 by as much as 20 out of 23 markers:

Somerset 1	13	23	15	10	12	15	11	14	11	30	20	14	18	10	14	17	17	10	21	26	13	12	12
Somerset 2	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	19	12	23	22	12	13	10
Somerset 3	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	18	19	12	23	22	12	13	10
Somerset 4	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10
Somerset 5	13	23	14	11	11	14	12	13	13	29	18	14	20	12	15	19	18	12	23	22	12	13	10

$$\text{Henry Somerset } 1744 - 1803$$

$$n = 20$$

$$m = 0.046$$

$$\frac{30 \times 20}{2 \times 0.046} = 6521.74$$

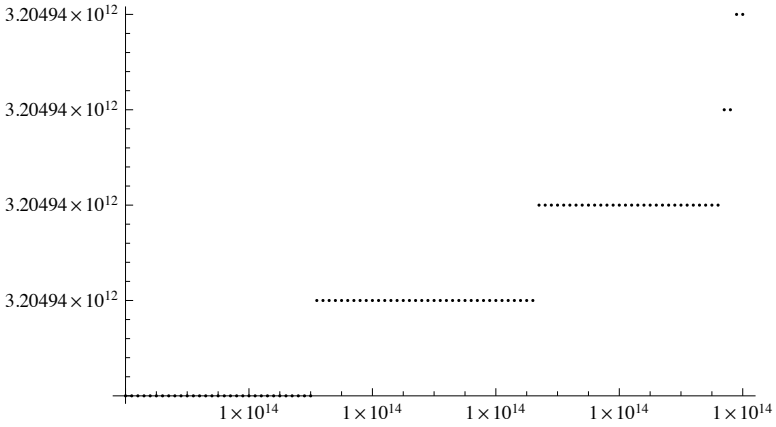
$$2017 - \sum_{n=2}^{6521.74} \frac{0.317506}{\log(n)} = 1744$$

$$2017 - \sum_{n=2}^{6521.74} \frac{0.26889}{H_n} = 1803$$

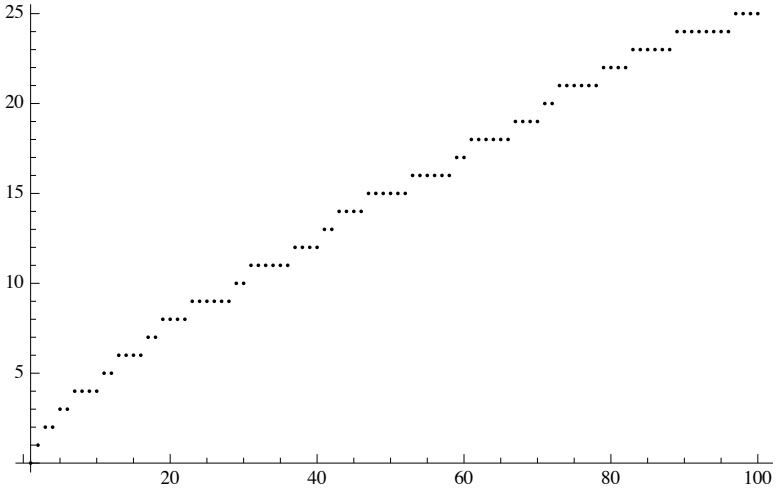
PART V

THE PRIME-DENSITY OF THE FAMILY TREE

Numerically speaking, a change in prime-density occurs if and only if a new prime number appears in an arithmetic progression, an occurrence that becomes ever more rare as the progression continues, until eventually one could not in one's lifetime count between one prime and the next.



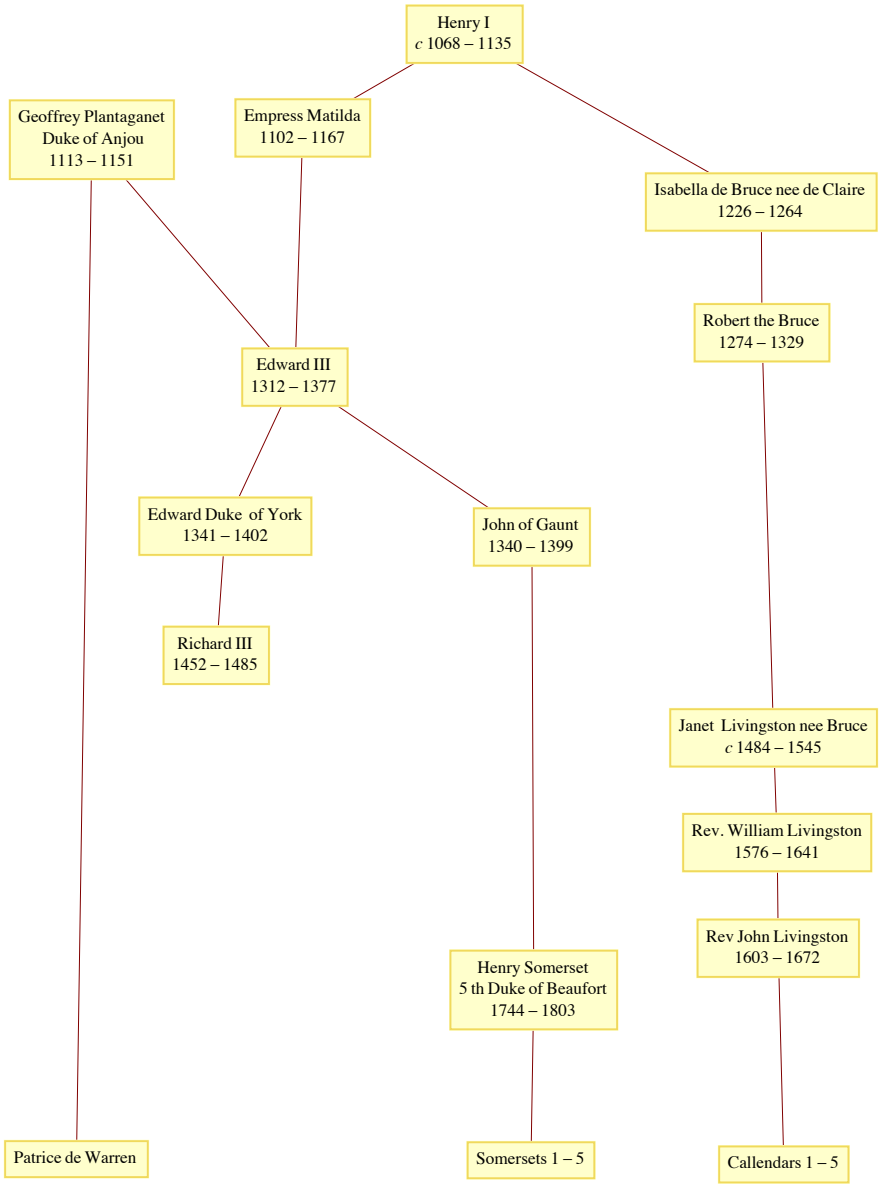
But in the *early* stages of an arithmetic progression new prime numbers appear frequently.



The central idea canvassed in this note is simply that SNP mutations correspond -albeit in a sophisticated sense- to changes in prime-density. When the upper and lower density-limits are employed, we see that a) these changes are constrained to follow a globally simple to complex/unique-to-repetitive pathway, but that b) local changes in prime-density are randomly either simple-to-complex/unique-to-repetitive or complex-to-simple/repetitive-to-unique. Because of their relationship to prime numbers, SNPs following either of these directions appear with great infrequency at sufficiently late stages of a Y-DNA time-line, but with *great frequency* at sufficiently early stages of a Y-DNA time-line. If we could survey a sufficiently dense section of a Y-DNA time-line, and perform DNA tests, we would -it is proposed- discover that what may seem, by reference to less dense sections or less dense lines, to be genetically impossible forward and back-mutations are common place. In the context of a dense time-line, the haplogroup of the son, it follows from this proposition, commonly differs from that of the father, and is commonly *simpler* rather than more complex than that of the father, i.e. SNPs commonly both come into existence and go out of existence in a prime-dense section of a time-line. It is inevitable in terms of this mathematical model that some Y-DNA sub-trees will at any point in time possess significantly more density than those around them, and will thus be mutating at a significantly faster and more erratic rate. These trees are in an objective rather than subjective sense younger than those around them, and therefore more volatile.

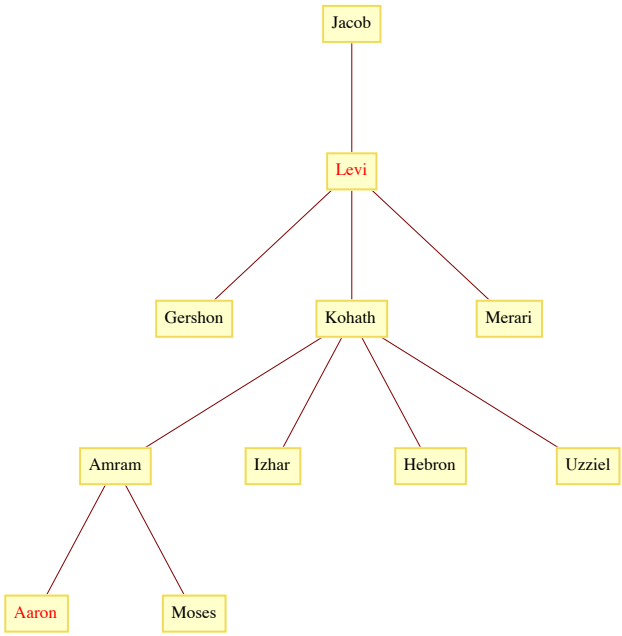
Any instability surrounding the Y-DNA of the Plantaganets and the Livingstons of Callendar, that does not also presently surround the Y-DNA of other men, is to be explained in this way. The density of a family tree may be maintained down the generations by limiting its number of unique nodes, which would be equivalent from a numerical

point of view to limiting the length of a time-line, and thus the loss of prime-density of the line. In the same way that the number of primes in a number line is inversely proportional to the length and prime-density of that line, the number of unique nodes in a family tree is inversely proportional to the size and 'genetic-density' of that tree. It may be stated that the genetic density of a tree is inversely proportional to its number of unique nodes, and it is arguable that the Plantagenet and Callendar Livingston trees in particular contain a lessor number of unique nodes than the average (whether by accident or design these turn out to form a single tree in recent times):



Y-CHROMOSOMAL LEVI AND AARON

There is in Judaism a distinction between a Cohen (a priest) and a Levite. A Cohen is a descendant of Aaron; a Levite is a descendant of Levi, but not necessarily a descendant of Aaron.



This topic has been carefully studied by geneticists, and Behar et al. in a 2003 study found that both Ashkenazi (Eastern European) and Sephardi (Spanish) Cohens tend overwhelmingly to belong to haplogroup J, but that the majority of Ashkenazi *Levites* belong of haplogroup R1a. A significant number of Sephardi Levites belong to haplogroup T.

Haplogroups	Ashkenazi Cohen	Sephardi Cohen	Ashkenazi Levite	Sephardi Levite	Ashkenazi Isrealite	Sephardi Isrealite
E1b1b	3.9 %	4.35 %	20 %	9.68 %	22 %	19.05 %
T	2.6 %	2.9 %	3.3 %	22.5 %	8 %	12.7
R1a	1.3 %	5.8 %	51.6 %	3.2 %	4 %	1.59 %
J1 and J2	86 %	75.3 %	10 %	32.2 %	37 %	36.51 %

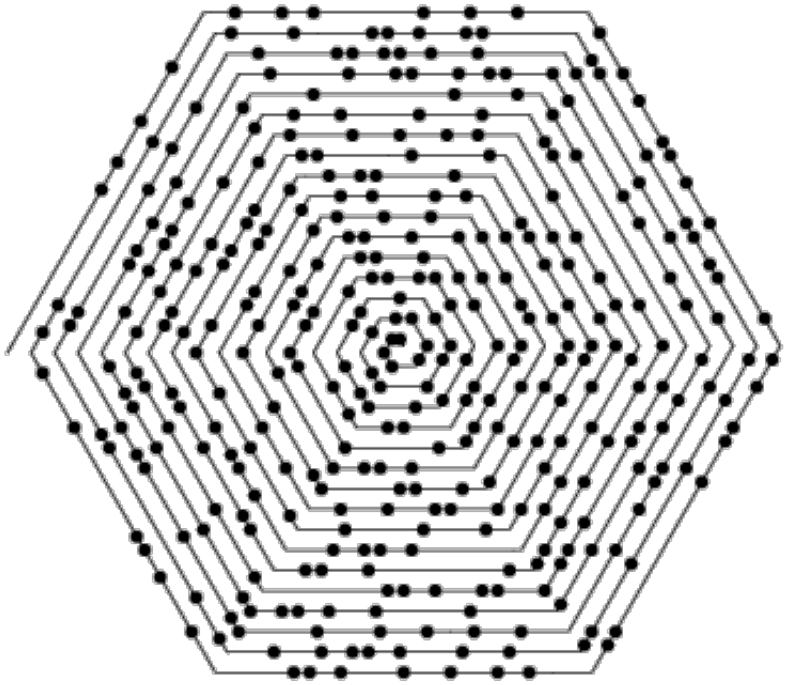
But if all of these men descend from the one man, then how in the light of observed mutation rates can one group of Cohens or Levites possibly have a different type of Y-DNA than another? And how, given that all Cohens are Levites, can Cohens have a different male heritage than Levites? (Note that one cannot become a Cohen or a Levite except by birth.) The only available answer, in the light of typical assumptions, is that no more than one of these lineages can be truly that of Levi and/or Aaron, and therefore that at certain points in history non-Levites and non-Cohens must both have assumed this role, or been appointed to it, and have subsequently have founded false bloodlines. Behar for instance takes the high percentage of R1a Ashkenazi Levites to show that these lines have a recent and European foundation:

Comparisons with other Jewish and non-Jewish groups suggest that a founding event, probably involving one or very few European men occurring at a time close to the initial formation and settlement of the Ashkenazi community, is the most likely explanation for the presence of this distinctive haplogroup found today in >50% of Ashkenazi Levites.

The same general problem arises in regard to the multiple haplogroups to which Jewish men belong. If all Jewish men are the sons of Abraham, Isaac, and Jacob, why are there so many different kinds of Jewish Y-DNA? Abraham lived so recently (about 4000 years ago) that the Y-DNA all of his male descendants should belong to the same haplogroup. Again, the obvious answer is that Jewishness has no firm genetic basis, and that men from a wide variety of ethnic

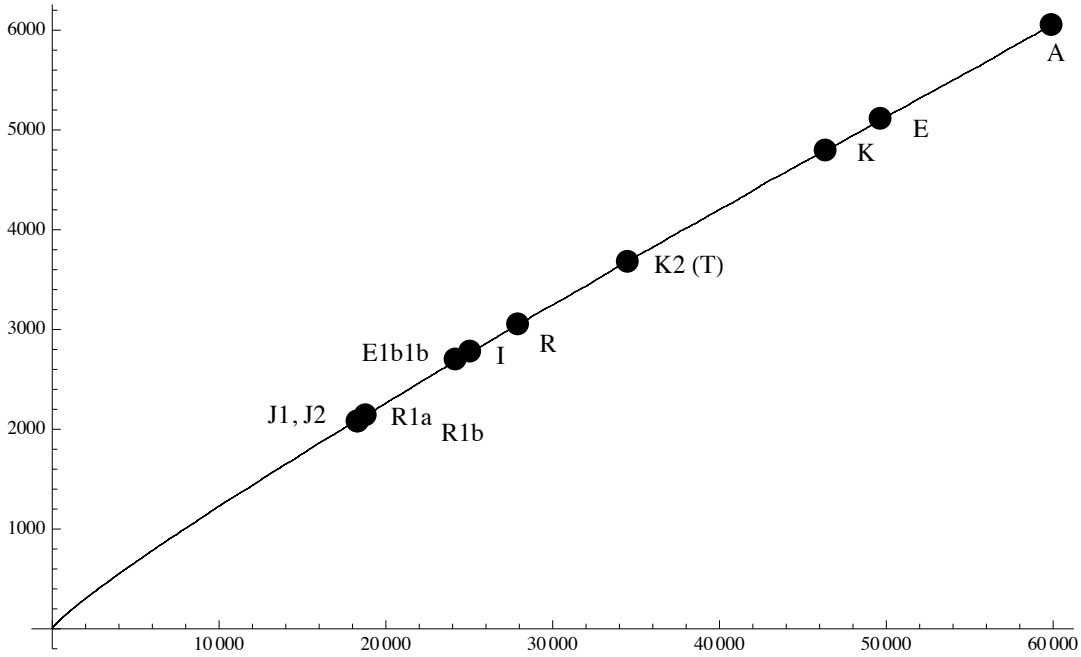
origins have often converted to Judaism and founded non-Abrahamic blood-lines. A problem with a Behar-style explanation, as far as Levites/Cohent are concerned, is how seriously these traditions are taken by those of the Jewish faith: whilst it is quite possible to convert to Judaism without being in any way a biological child of Abraham, it is *unthinkable* in the context of Judaism that these roles could be played by someone lacking the appropriate patrilineal descent, and oral and written records have been scrupulously kept for centuries to ensure the integrity of the inheritance.
The quest for the *haplogroups* of biblical Levi and Aaron is clearly a hopeless one: although there are genealogies -such as those of the Samaritan high priests- that extend back many centuries, none goes back nearly as far as necessary. The genetic signature known as the 'Cohen modal haplotype' (DYS19 = 14, DYS388 = 16, DYS39 = 23, DYS391 = 10, DYS392 = 11, DYS393 = 12) has been found to be more common amongst self-declared Cohens than any other group, but putting aside the objections that the signature is found amongst several lineages, and amongst both non-Cohens and non-Jews, there is in any case no trace back to Aaron.... But arguably we don't need a paper trail leading from the present to Levi or to Aaron, or a model haplotype common only to Levites or Cohens for *all* such traditions to be at least credible. Arguably we have *a priori* reasons to accept the possibility that patriarchs such as Levi and Aaron founded, not one or two, but many lineages present to the world today.

As indicated, the distinction between unique and repetitive change that underlies genetic development also underlies the number line. Every prime number is unique, and every non-prime number is a repeated prime. 2 is unique, 3 is unique, 4 is a repeated 2, 5 is unique, 6 is a repeated 2 and a repeated 3, 7, is unique, 8 is a repeated 2, and so on and so on... The number line forms a tree structure whose branches correspond to the prime numbers, but since prime numbers don't change in a uniform way, they can be most easily be envisioned as a spiral:



They never run out altogether, but they spiral outwards, and the further one counts down the line the fewer of them there are. To cut a potentially long story short, it has been proposed in essence that UEPs are the unique elements of the arithmetic process of reproduction and that as such they are governed by the same principles that are responsible for the distribution of the primes. If this proposal is *right*, then by subtracting the non-prime years from the ages of haplogroups estimated on the assumption that UEPs change at a uniform rate, we should arrive at a more realistic genetic time-line. If the proposal is right, then the failure to take account of this relationship is bound result in an absurdly *unrealistic* genetic time-line.

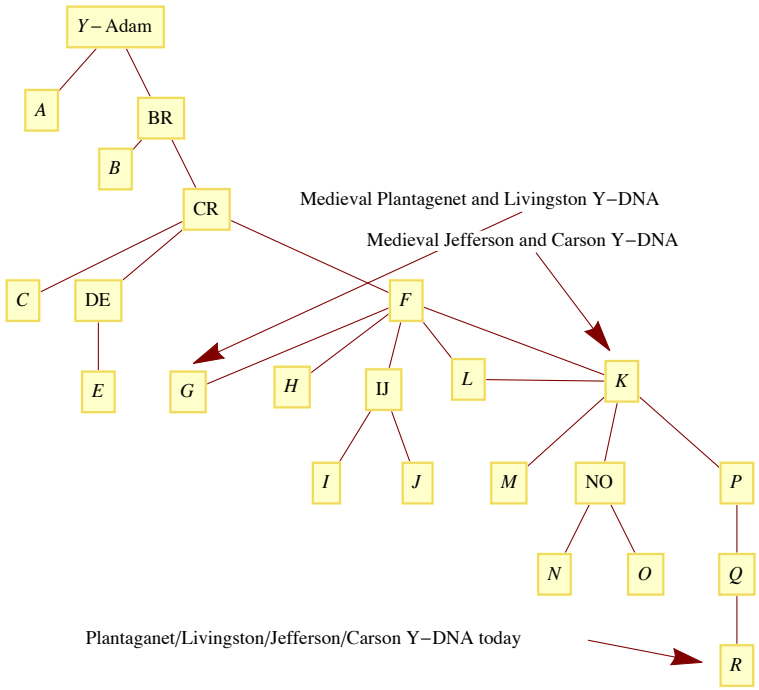
The following chronology is necessarily rough and speculative, but it illustrates the difference this approach to UEPs makes:



Haplogroup	Possible Time of Origin Accoridng to Evolution (ybp)	Possible Time of Origin According to (*f2*) (ybp)
A	60 000	6057
E	50 000	5133
K	40 000	4851
K2 (T)	30 000	3245
J	30 000	3245
R	28 000	3085
E1b1b	26 000	2860
I	25 000	2762
R1a	21 000	2360
R1b	20 000	2262
J1	20 000	2262
J2	19 000	2158

Of course it is widely believed that Y-Adam are far older than 6000 years (the latest figure is 300, 000 odd), but once in possession of the idea that unique changes are subject to entropy, we will find *all* of the usual methods for estimating the age of ancient things to be prone to exaggerate this age. Take for example the method of radiocarbon dating. When an animal dies, its level of Carbon 14 is approximately equal to that of the atmosphere at that time, and it subsequently decreases from 'beta decay', i.e. the loss by the nucleus of an atom of electrons or positrons. By measuring the amount of the stable Carbon 12 the remains of the animal contains, and comparing it to that of the unstable Carbon 14 isotope, we can estimate the date of death. But if the rates at which atomic nuclei emit energy follow the primes rather than the integers, then the assumption that the ratio of Carbon 12 to Carbon 14 decreases at a uniform rate will lead to inaccurate readings. Imagine again a clock that runs more quickly in the present than in the past : if we assume that this clock has always run at its presently observable rate, and if we try to calculate how much time has elapsed since some earlier rotation, we will find that the calculation tends to over-estimate. Since the clock *spirals* rather than circles, the accuracy of the estimate will be inversely proportional to the number of rotations. However since this is a rough rather than smooth spiral, the equation is probabilistic. Correspondingly, the accuracy of a radiocarbon date may be inversely proportional to the age of the object being dated - the older the object, the more likely it is that any estimate of its age based on the assumption of uniformity will be inflated. Ironically, this mathematical framework derives from *On the Number of Primes Less Than a Given Magnitude*, a work published by Bernhard Riemann in the same year and in the same month (November 1859) that Charles Darwin published *On the Origin of the Species* (the latter work is the most significant source of the evolutionary world-view and its simplistically linear chronology). In the light of the Riemannian framework, the possibility exists that rare Near Eastern haplogroups such a T and G are traces of haplogroups that were relatively common in the eras of Andrew de Livingston, Robert de

Carson, and Richard III, but have since mutated, creating the illusion that relatives whose haplotypes belong to different haplogroups are unrelated in recent times.



In the Darwinian framework there simply *is* no such possibility. Mutation rates are assumed to be glacially slow, wholly linear, and the MRCA of the men with the same surname but haplotypes belonging to different haplogroups must by these assumptions have lived, not 100s of years ago, but *10s of 1000s* of years ago. But if genetic mutations follow the primes in the manner described above, then this possibility is a real one. And if the estimated times of origin for hapolgroups such as G and T are more or less right ones based on mutation rates observed in the present, then *(f2)* says that earliest ancestors lived, not in one of the fantastic times offered by evolutionary genealogy -37, 000 BC for example- but in a historically documented time.

$$2017 - 40\,000 = -37\,983$$

$$2017 - \pi(40\,000) = -2186$$

The 'Riemannian' framework explains, where the Darwinian framework does not, the existence of multiple Plantagenet haplogroups, and multiple Livingston haplogroups arising from a recent common ancestor. No doubt there are *many* spurious genealogies, but within the Riemannian mathematical framework alone arises the possibility that all of those men whose genealogical traditions assert that they are the descendants of Levi and/or Aaron and/or Abraham really did descend from these patriarchs.

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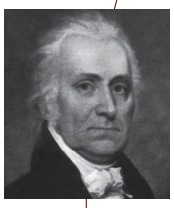
APPENDIX

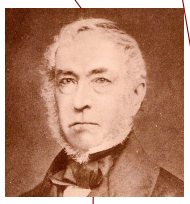
Rev. John Livingstone
1603 – 1672

Robert Livingston 1 st Lord
1654 – 1728

Robert Livingston 3 rd Lord
1705 – 1790

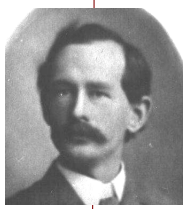


John Livingston
1750 – 1822



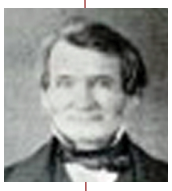
Robert Swift Livingston
1781 – 1867

Col. Charles Edward Livingston
1837 – 1893



Charles Granger Livingston
1865 – 1940

Dr. William Livingston
1768 – 1860



James Gray Livingston
1802 – 1890

Callendar 3 (R1b)

Callendar 4 (R1b)

Callendar 1 (G2a)

Callendar 5 (R1b)

Callendar 2 (R1a)