CHROMOSOME 21: Inherited from 5th Great Grandparents – Captain Daniel and Elizabeth (WINDECKER) YOUNG

The Purpose: The reason for writing this essay is to explore what appears to be a highly unusual genetic genealogy observation, namely the author's apparent inheritance of his maternal chromosome 21 without recombination (unchanged) from his great great grandmother, and further back in its entirety from two of his 5th great grandparents. This low probability event has triggered the curiosity of the author to better understand this phenomenon, and the particular chromosome involved here. The following report will reflect a personal approach to the subject in a way that is uncharacteristic for a scientist – so apologies for the references to self found herein.

The Ancestors: Daniel Young was born about 1755 in the Canajoharie District of the Mohawk Valley New York, U.S.A. and died 9 May 1836 at his home in Barton Township (now City of Hamilton), Wentworth County, Ontario, Canada. His father was Johann Adam Jung (Young) was born in 1717 at Foxtown, Schoharie Valley, New York, and died in 1790 at the Grand River in what is today Seneca Township, Haldimand County, Ontario. Daniel's mother was Catharine Elizabeth Schremling who was born about 1720 in the Schoharie Valley, New York and died in 1798 at the home of her son Daniel in Barton Township. Daniel was a Sergeant in Butler's Rangers during the American Revolution, and a Captain of the 5th Lincoln Militia during the War of 1812. An overview of his illustrious career can be seen here. Daniel's wife was Elizabeth Windecker, born about 1763, probably on the Windecker Tract, Canajoharie District, New York, and died at her home in Barton Township on 8 March 1829. Elizabeth's father was Hendrick Windecker (a "notorious" Private in Butler's Rangers) born about 1738 probably on the Windecker Tract, and died after 1814 likely at the Grand River, North Cayuga Township, Haldimand County, Ontario. Her mother was Dorothy Pickard born about 1743 in the Canajoharie District, likely on the Windecker Tract, date and place of death unknown.

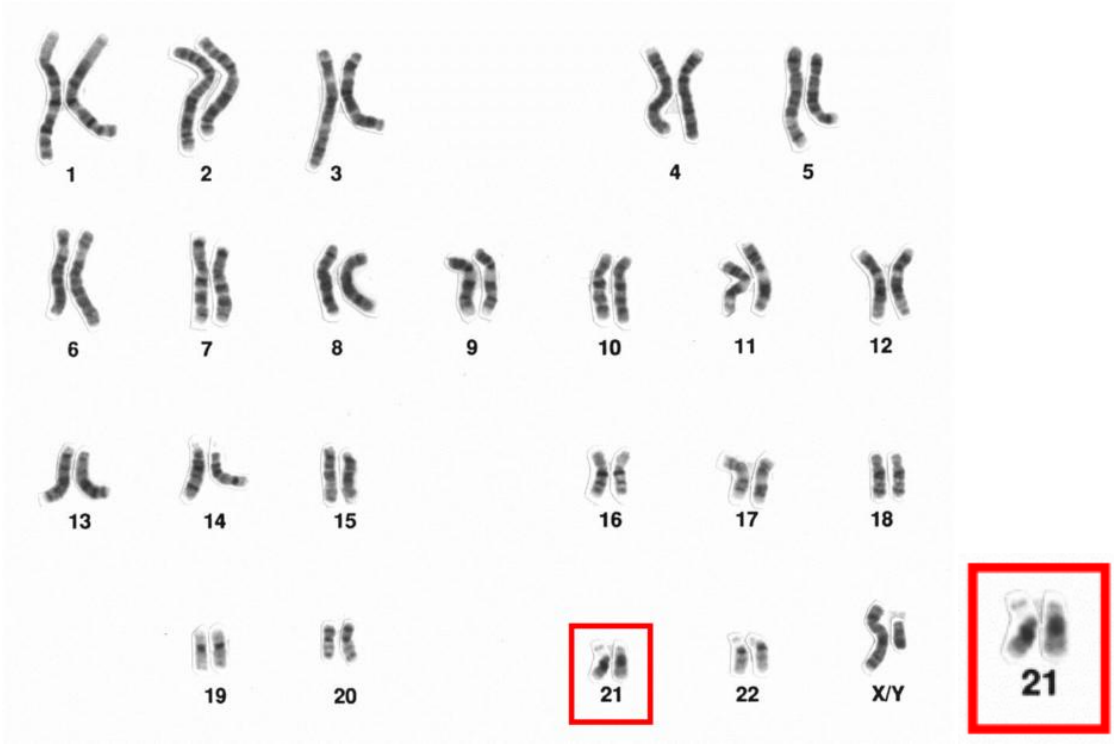
Author's Genealogical Connection: The extensive paper documentation travels back in time from David K. Faux to his mother Violet M. Williamson to her mother Eva F. Dawson to her father Joseph W. Dawson to his mother Hannah Adelia Young to her parents Henry Young and Elizabeth M. Young (first cousins). Henry's father was Henry Young Sr. and mother Rachel Young (a first cousin once removed to her husband). Elizabeth M. Young's father was George Young and her mother was Mary Terryberry. Henry Young Sr. and George Young were brothers, both the sons of the above Daniel and Elizabeth – making the latter the 5th great grandparents (twice over) to David K. Faux.

DNA Testing: The autosomal (22 chromosome pairs) DNA of David K. Faux was tested by Ancestry.com, 23andMe.com, and FamilyTreeDNA.com; and further analyzed by Gedmatch.com. Cousins of varying degrees were also tested and analyzed by one or more of the

above firms. Key matches to segments along chromosome 21 (and others) were informative cousins (close cousins share too many lineages to be certain of how to interpret the results) ranging from half third cousins once removed and 4th cousins (where a matching segment on a chromosome can be assigned to the Young / Windecker family), to 5th and 6th cousins (where the match can often be isolated to either the Young or the Windecker family) – basically those with whom the only connection, meaning ancestors in common, was via the Young or Windecker families. Thus in some instances a matching segment between two distant cousins could be deemed to be from Daniel Young or Elizabeth Windecker (and in some cases even to the level of their parents or beyond).

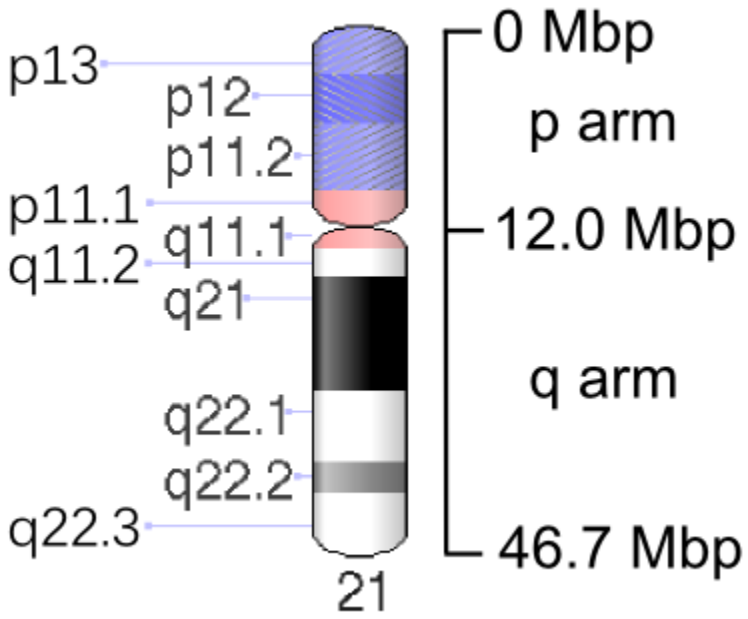
An Overview of Chromosome 21: Before delving into how the author is able to say with certainty that the entirety of his maternal chromosome 21 comes from his 5th great grandparents, it will be useful to provide a brief description of the nature of this chromosome.

To visualize chromosome 21 within the context of the human genome a good starting point is a karyogram to show what it "looks like" at least at one stage of the process of cell division. If for example a blood sample is drawn and say a white blood cell is "opened up" to view the nucleus and its contents when, during cell division, the chromosomes have isolated themselves into discrete entities, what follows is what one would see:



It can be seen that chromosome 21 is the smallest of the 22 autosomes (the 23rd pair being the sex chromosomes), and that there are two – one from the father and the other from the mother.

Another useful way of visualizing chromosome 21 is diagrammatically where the different regions can be displayed, and their distinguishing features noted, as seen below:



G-bands of human chromosome 21 in resolution 850 bphs[17]

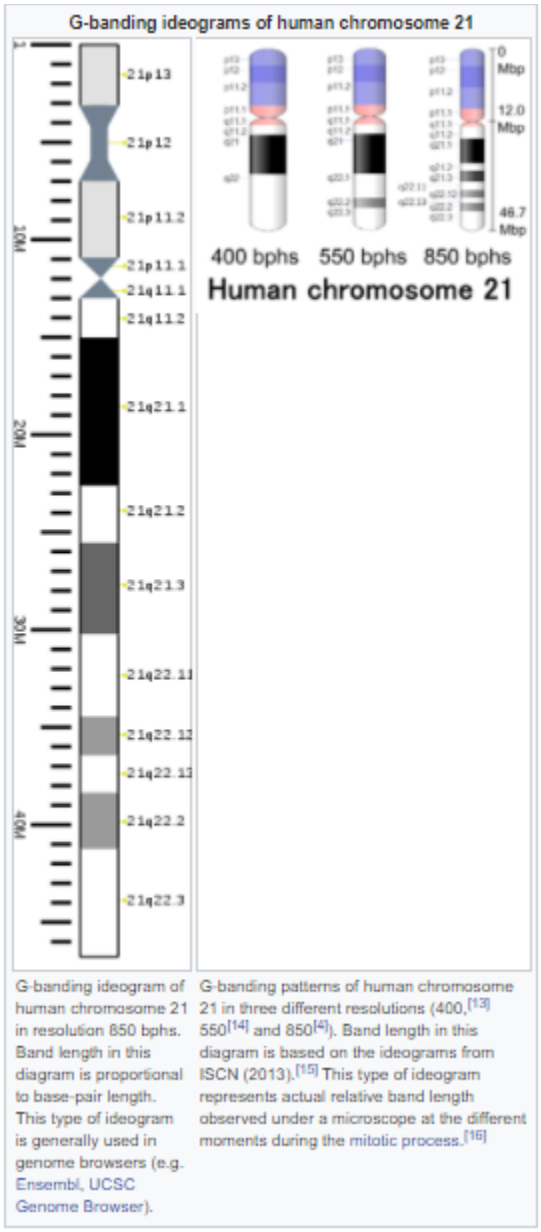
Chr.	Arm[18]	Band[19]	ISCN start[20]	ISCN stop[20]	Basepair start	Basepair stop	Stain[21]	Density
21	p	13	0	311	1	3,100,000	gvar	
21	p	12	311	683	3,100,001	7,000,000	stalk	
21	p	11.2	683	1056	7,000,001	10,900,000	gvar	
21	p	11.1	1056	1274	10,900,001	12,000,000	acen	
21	q	11.1	1274	1367	12,000,001	13,000,000	acen	

21	q	11.2	1367	1584	13,000,001	15,000,000	gneg	
21	q	21.1	1584	2019	15,000,001	22,600,000	gpos	100
21	q	21.2	2019	2144	22,600,001	25,500,000	gneg	
21	q	21.3	2144	2330	25,500,001	30,200,000	gpos	75
21	q	22.11	2330	2485	30,200,001	34,400,000	gneg	
21	q	22.12	2485	2610	34,400,001	36,400,000	gpos	50
21	q	22.13	2610	2703	36,400,001	38,300,000	gneg	
21	q	22.2	2703	2858	38,300,001	41,200,000	gpos	50
21	q	22.3	2858	3200	41,200,001	46,709,983	gneg	

The length of the chromosome in total is 46.7 Mb, meaning 46,700,000 base pairs in length (other work gives as much as 48,129,895 bp). So one may have say an A nucleotide base (a SNP or single nucleotide polymorphism) from your mother and a C from your father at one location along the chromosome – the total being over 46 million pairs of them. For our purposes this is what is most important due to what is being tested, but it is noteworthy that a chromosome included many other elements such as short tandem repeats (STRs) such as strings of say ACGACGACG, insertions, deletions and perhaps an inversion, and a host of other characteristics that do not come into play for our purposes. Although Hattori found only 127 known genes, 98 predicted genes and 59 pseudogenes, more recent research suggests that the number may be as high as between 477 and 635 genes. The above ideogram does not show all the bands that appear on chromosome 21 after staining and viewing under a microscope – each band being used to help define the location of a gene.

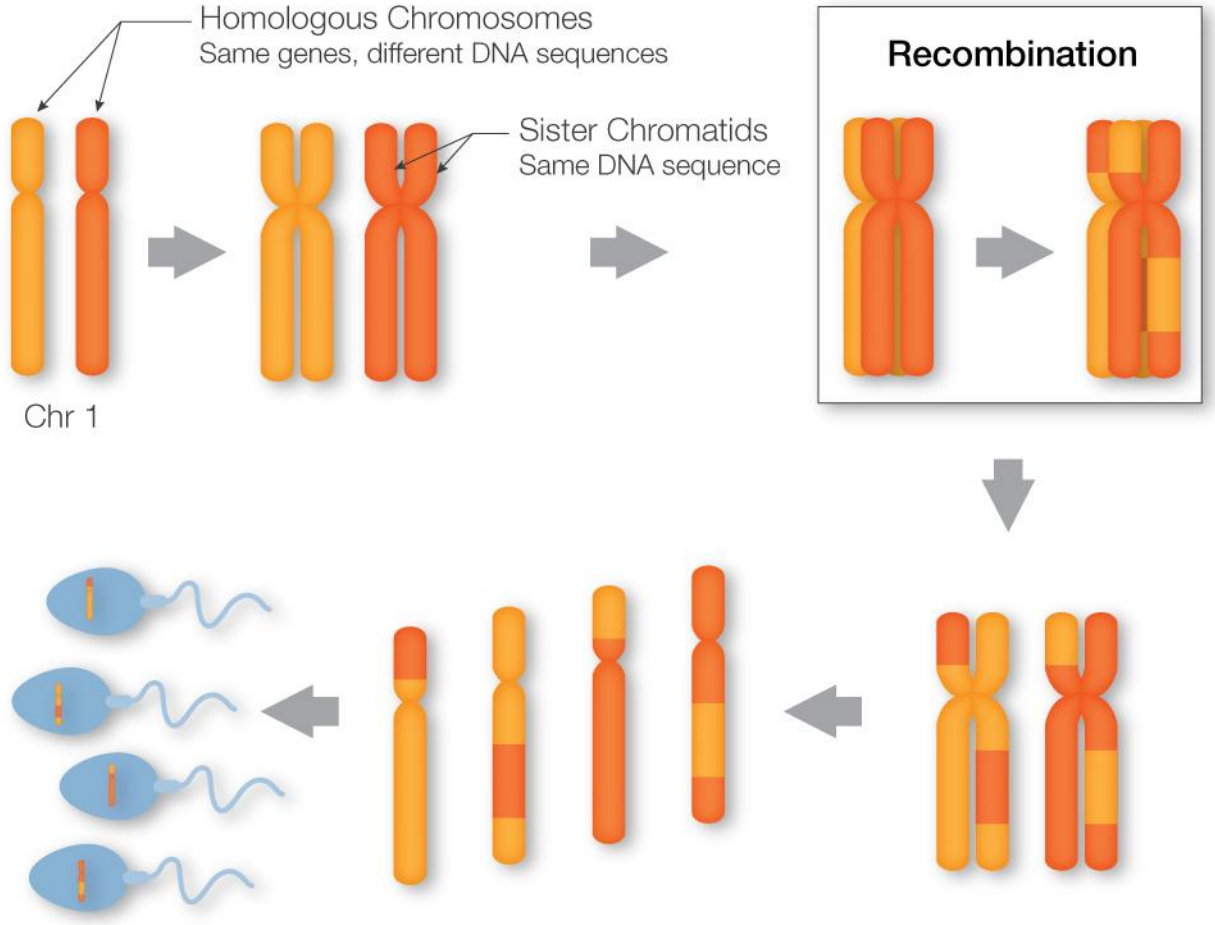
The chromosome is composed of euchromatin (the q arm) which is what is being tested by the various commercial companies, and heterochromotin (the p arm) which is a complex tangle

shown in blue above that contains few if any genes and which is lacking in many identifyable SNPs. The pink section above is the centromere, separating the p (short) and q (long) arms, and which is also very SNP poor and largely uninformative in terms of ancestry and family matching, and its origin must to some extent be inferred. Thus for all intents and purposes what can be said definitively relates to the q arm alone, and only the SNPs from about 14.6 Mb shown at the top of the dark black stained band known as q21 to the telomere (end section) of the distal end at 46.7 Mb.



Some of the "performance characteristics", particularly in terms of what happens in meiosis (the formation of eggs and sperm) of chromosome 21 have likely impacted the origin of the segments found here in the author. One key finding is that there is considerably less male recombination than female recombination during meiosis (in the order of 1.6 female recombinations to every 1 male recombination). This means that on average male recombinations will produce longer segments, and female recombinations more but shorter segments – "breaking up" the chromosome more.

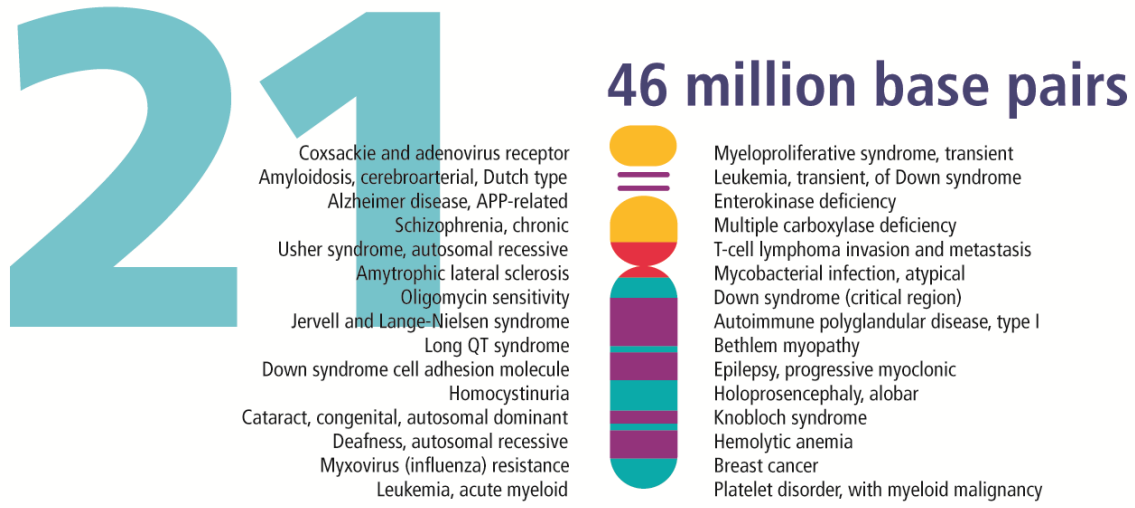
During meiosis chromosomes must go from the diploid (possessing 2 of each chromosome) to the haploid (possessing one of each chromosome) state. The X shaped chromosome joined at the centromere lines up with the sister chromatid from the mother during egg production, and the same process occurs in the father during sperm production, then the process of recombination occurs. To picture recombination, the following diagram should help:



Here, during the process of recombination, the chromatids exchange genetic material at a chiasma (point for disjunction or break apart and junction or splicing) so that each new pair will have chunks (blocks or segments) from its homologous mate, meaning that in a female the chromosome that gravitates to one of the four eggs (gametes) formed after division has perhaps two new segments sliced from the other chromosome in the pair. During fertilization one of the chromatids from the mother will merge with the contents of the gamete of the father. Then cell division from this point is called mitosis. At a certain phase of mitosis (division of body cells to form a duplicate), for example white blood cells, it is possible to isolate each individual chromosome from the mother and father as seen in the karyogram above. Hence the new chromosome compliment is a combination of the two original units (usually one or two recombintaions), one from the mother and one from the father – unless recombination did not occur.

The randomness and sheer number of possibilities of what emerges in the formation of the four gametes above is staggering. However one of the possibilities is that an entire chromosome can be passed to the next generation – but the likelihood of this happening such that someone obtains an entire maternal or paternal chromosome from 5th great grandparents is vanishingly small since there are 6 meiotic recombination events or opportunities for segments to be spliced from the other 30 maternal ancestors. It would mean that for all intents and purposes there was no recombination, effectively it did not happen at all, for the events leading up to what is inherited by a 5th great grandchild. In the author's case, the outcome was likely to some degree assisted by a cousin marriage, but this would explain only a small fraction of the whole picture.

<u>Diseases Associated with Chromosome 21</u>:



Genes Associated with Chromosome 21:

The following are some of the approximately 215 to 450 genes (the exact number is currently being debated) located on chromosome 21:

- APP: amyloid beta (A4) precursor protein (peptidase nexin-II, Alzheimer disease)[11]
- C21orf45: encoding protein Protein Mis18-alpha
- C21orf55/DNAJC28: encoding protein DnaJ homolog subfamily C member 28
- C21orf56: encoding protein Uncharacterized protein C21orf56
- C21orf59: Chromosome 21 open reading frame 59
- C21orf62: expressed in tissues of the brain and reproductive organs
- C21orf66: encoding protein GC-rich sequence DNA-binding factor homolog
- CBS: cystathionine-beta-synthase
- CLDN14: claudin 14
- CRYZL1: Crystallin zeta-like 1
- CYYR1: Cysteine and tyrosine rich 1
- DIP2A: Disco-interacting protein 2 homolog A
- DOPEY2: Dopey family member 2
- DSCR1: Down Syndrome critical region 1[12]
- DYRK1A: dual specificity tyrosine-(Y)-phosphorylation regulated kinase 1A
- FAM3B: Family with sequence similarity 3 member B
- FRGCA: encoding protein FOXM1-regulated, gastric cancer associated
- HLCS: holocarboxylase synthetase (biotin-(propionyl-Coenzyme A-carboxylase (ATP-hydrolysing)) ligase)
- KCNE1: potassium voltage-gated channel, Isk-related family, member 1
- KCNE2: potassium voltage-gated channel, Isk-related family, member 2
- LAD: leukocyte adhesion deficiency (symbols are ITGB2, CD18, LCAMB)
- PCNT: centrosomal pericentrin
- PDXK: encoding enzyme Pyridoxal kinase
- PSMG1: Proteasome assembly chaperone 1
- RNR4: RNA, ribosomal 45S cluster 4
- RRP1: encoding protein Ribosomal RNA processing protein 1 homolog A
- RRP1B: ribosomal RNA processing 1 homolog B
- RWDD2B: encoding protein RWD domain-containing protein 2B
- S100B: calcium binding protein
- SOD1: superoxide dismutase 1, soluble (amyotrophic lateral sclerosis 1 (adult))
- TMEM1: encoding protein Trafficking protein particle complex subunit 10
- TMPRSS3: transmembrane protease, serine 3

- TTC3: Tetratricopeptide Repeat Domain 3

Results of Non Disjunction of Chromatids During Meiosis:

This chromosome is the cause of Down's syndrome, Trisomy 21, there being three chromosomes rather than the normal two. Here one parent (often the mother, and more common in older mothers) transmits two chromosomes to the child, and added to the one from the other parent, yields 3 instead of 2. The child is viable (in contrast to trisomys involving other chromosomes

which result in miscarriage or early infant death). Presumably the excess proteins / enzymes or other factors due to this irregularity is what causes the features characteristic to Down's including the distinctive facial features, intellectual deficits, short fingers, thick tongue – to name a few.

Some Facts About Chromosome 21 Relating to the Failure to Recombine:

1) Chromosome 21 was first sequenced and fully described in 2000 by Hattori et al.

2) It is the smallest of the 22 autosomes and so has a greater chance of being transmitted to the next generation without recombination (a 40% chance) relative to the larger chromosomes (chromosome 1 has a 4% chance of being transmitted without recombination).

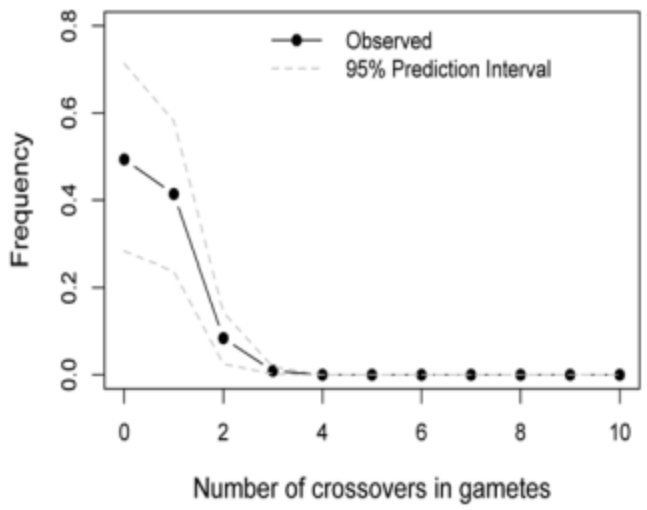
Due to this small size factor, chromosome 21 only represents between 1.5 and 2% of the total cellular DNA.

3) In general, female recombination is about 1.5 times greater on this chromosome than it is in males, with a tendency to see more near the centromere (8 times more likely) and decreases along the q arm. Male recombination in the specific telomeric regions, however, is about 1.8 times that seen in females (Lynn et al., 2000).

4) The centromere exerts a negative effect on recombination both within itself and in proximal regions. However, the exception as noted in the Lynn study is in females where the recombination rate is higher in the most proximal region (2.4 Mb) of the q arm to the centromere than along the rest of the arm. In females there is also a "recombinational hyperactive region" localized at 5 Mb from the centromere (a "hot spot"). Little is known of the recombination activity along the short arm – 21p (Laurent et al., 2003).

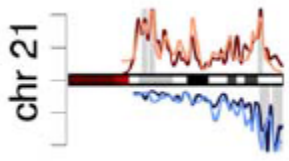
5) A recombination factor unique to chromosome 21 is the observation that it does not require a chiasma (attachment point) during meiosis. According to Fledel-Alon et al. (2009), "*chromosome 21 seems to be frequently transmitted properly in the absence of a chiasma in females*". In other words, viable offspring are standard even without the chiasma seen in other chromosomes – thus "*chromosome 21 appears to frequently segregate properly in the absence of a crossover*".

6) The chance of chromosome 21 being transmitted to the next generation without recombination is 37% in females, and 44% in males (FledelAlon et al. (2009)). The following chart profiles this probability:



So with combined male and female data, approximately 50% of the time there will be no crossovers, 40% 1 crossover event will be observed, and in 10% of the instances there are 2 or more recombination crossovers. Thus, while far from rare in chromosome 21, a no recombination – crossover event during meiosis is only seen about half the time. Also, what is seldom noted, is that due to a no recombination event, half of the transmissions (whether combined or uncombined) will see one of the pairs of chromatids "disappear" (not be passed along to the next generation) – for example the grandfather's chromosome if the grandmother's unrecombined chromosome is transmitted. With each generation extending into the past the probability of inheriting the same unrecombined chromosome becomes considerably more unlikely. This is amplified when most of the transmissions are female (the higher recombination rate means a higher probability of producing a recombined chromosome during any meiosis event).

7) A series of recombination jungles (hot spots), as well as "cold spots" or deserts, have been profiled for all chromosomes. Hotspots relate to the q arm, from about 14.5 Mb (the effective start point for the assessment of this chromosome). The q arm contains functional euchromatin versus the SNP poor heterochromatin that characterizes the p end.



What is shown in the above diagram from Chowdhury et al. (2009) are the areas of the chromosome which are most likely to include these high probability sites of recombination during meiosis (gamete formation). The bright red area to the left includes the SNP poor p region, centromere and part of the q region where recombination is unlikely to take place. The

jagged red line above the chromosome represents the probability of recombination in females, and the comparable blue line below displays probability of recombination in males. What is clear is that female recombination is most likely to occur in the region between about 14 and 20 mb, and in males at the far end (telomere) of the q region (about 35 to 46 mb). As will be discussed, these regions correspond precisely to the areas where there appears to be an amalgam of the parts of chromosome 21 inherited by the author from Daniel Young and his wife Elizabeth Windecker.

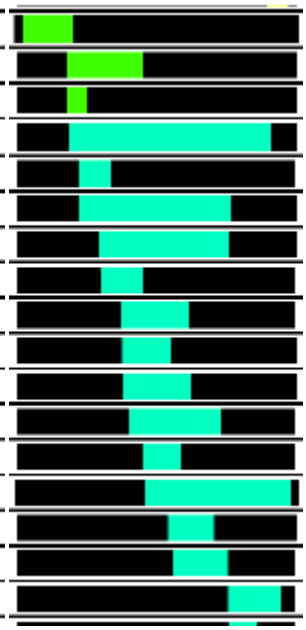
8) More information can be found in the Wikipedia article for Chromosome 21, and "Genetics Home Reference" Chromosome 21 articles online.

Genealogical Evidence that Chromosome 21 was Inherited from Daniel and Elizabeth (Windecker) Young:

The evidence comes from the matching segments of individuals with a known genealogy from half third cousins once removed to 9th and beyond cousins. Most of these can be displayed in the match profile from Gedmatch.com from individuals who uploaded their raw data to that site, and comparing this to their documented genealogy. This is seen in the diagram below, using the phased maternal data of the author. There are two versions since between the two dates, somewhat different versions of the matches emerge due to the apparent change from Build 35 to Build 37. Specifically, some individuals have been removed from the first version; some have been added to the latter; and the start and stop points for each match has changed slightly:

1) 21 October 2015 – Build 35

21	9,849,404	16,699,698	8.7	740	Ferne Bolton
21	16,658,736	26,769,135	18.7	2,801	Norm Sones
21	16,658,736	19,341,200	7.1	798	*Marti Sigsbee
21	16,969,464	43,652,459	54.3	7,962	K.L.
21	18,330,173	22,749,282	8.7	1,284	Robert Lloyd Hall
21	18,344,736	38,231,969	33.9	5,380	Lawrence William Brown
21	20,915,614	38,276,905	28.4	4,705	*Kathryn Willson
21	21,271,294	27,141,178	9.0	1,002	Gail Crichton
21	23,901,585	33,066,196	13.7	2,359	Nola Helen King
21	23,901,585	30,509,664	9.6	1,120	*RobertsMaiden
21	24,060,742	33,090,443	13.4	1,477	*sefp
21	25,012,337	37,004,682	19.8	2,031	Jocelyn Malheiro
21	26,798,079	31,793,748	7.0	1,293	Barry Shumaker
21	27,311,934	46,909,175	41.2	6,108	Ferne Bolton
21	29,944,093	36,342,520	11.0	1,730	Anthony Messuri Jr
21	30,683,684	38,222,301	14.0	1,321	Rashelle Elburg
21	38,145,277	45,159,710	19.4	2,616	Norm Sones



2) 30 May 2019 – Build 37

14670124	17772222	8.5	675	Ferne Bolton	F	
15215814	17624130	6.6	300	Jackie Yorke	F	
17736865	27845535	18.6	2669	Norm Sones	U	
17763748	20407698	6.9	543	*heather	F	
18047593	44823479	54.2	7599	K.L.	M	
19422865	39288940	33.7	5133	Lawrence William Brown	M	
19797601	34384588	23.1	974	*candai	F	
20133449	32224934	18.3	1972	*CW	M	
20701689	28821164	14.0	1564	Kristen Vogt	F	
21996784	39349932	28.3	4467	*Kathryn Willson	F	
24936629	34059352	13.6	604	*sandelk06	F	
24979714	34125319	13.7	2241	Nola Helen King	U	
25138871	34125319	13.3	1471	*sefp	F	
26090466	38075313	19.7	2028	Jocelyn Malheiro	F	
27876208	32833470	6.9	1206	Barry Shumaker	M	
28390063	47995443	40.8	5818	Ferne Bolton	F	
29514609	38075313	13.7	1407	*Cave Yodel	M	
29703656	37406849	11.7	1901	Anthony Messuri Jr	M	
31371100	38075313	12.5	1170	Linda Kinker	F	
31761813	39285453	13.9	1317	Rashelle Elburg	F	
32735471	38089097	11.0	1055	Nicole Myers	F	
39223407	46327835	19.4	2515	Norm Sones	U	

The chart names the chromosome, the start and stop matching segment in Mb, the cM (genetic size of the match), and the number of matching SNPs (single nucleotide polymorphisms). Note that a cM is a measure of genetic distance whereas Mb is a measure of physical distance. As a very general rule, one cM = 1 Mb, but there are notable exceptions. None of the new additions to the second iteration (2)) have any attached genealogy at Gedmatch or Ancestry.

With respect to the known individuals, and using the 2) version as the reference point:

The author shares the first measured part of chromosome 21 with his sister **Ferne Bolton** (14.7 Mb to 17.8 Mb) and his first cousin **Jackie Williamson Yorke** (15.2 Mb to 17.6 Mb). The p end to 14.6 Mb has so few SNPs (markers) that it cannot be measured by existing technology. Jackie shares nothing further on this chromosome – and the author's uncle **Dale Williamson** shares zero with the author on chromosome 21. This means that it is not possible to say with complete certainty anything about the p end to 14.6 Mb of the chromosome – although it is not known to recombine.

Norm Sones is a half 3rd cousin once removed with the ancestor in common being the author's ggg grandmother Elizabeth M. Young. He matches in two locations on this chromosome – the distal ends.

Marti Sigsbee is a 9th cousin, with our only shared ancestor being George Landgraff, the grandfather of Catharine Elizabeth (Schremling) Young. The match is located at the beginning of the chromosome (p end), starting at the same SNP as Norm Sones – suggesting that it is a Young (not Windecker) segment.

K.L. is the author's second cousin. Our maternal grandmothers were sisters. It appears that K.L. shares almost as much of chromosome 21 as the author, with the exception of small amounts at each end.

Lawrence William Brown is a 5th cousin once removed, a descendant of Peter Young (via his daughter Catharine Cramer), brother to the author's 4th great grandfathers, Henry Young and George Young.

Bob Hall is also a 5th cousin once removed, and a descendant of Peter Young but via his son Edmund Wellington Young. Bob's data is not on Gedmatch at this point but our match was from a start point of 18,330,173 to 22,749,282. The match is 8.7 cM.

Kathryn Willson is a 5th cousin twice removed, a descendant of Barbara Windecker (who married James Fleming), daughter of Henry Windecker (father of Elizabeth who married Daniel Young – the parents of Henry, George, Peter and others) and Dorothy Pickard.

Jocelyn Malheiro and the author's closest connection is via Hans Bellinger (born about 1618) who married Anna Bellinger about 1642 at Steinau an den Strass, Hessen, Germany; and Hans Kuhn (born 1625) who married his sister Catharina Kuhn in 1647 in Langenselbold, Hessen, Germany. Jocelyn is a descendant of Hans and Anna's son Dietrich Bellinger born about 1644 who married Barbara Gessen and had son Johannes Bellinger born 1664 who in 1690 married Anna Margaretha Kuhn born 1661, daughter of the above Hans and Catharina. The author, and the other Windecker descendants noted in this article, are descendants of Johannes' uncle, Nicholas Bellinger born about 1645, who married Anna Kuhn, the sister of the above Anna Margaretha Kuhn. This makes Jocelyn and the author 11th cousins! The genealogy above was completed by Hank Jones and found in Vol. 1 of his "Palatine Families of New York". There are no other ancestors known or likely to be shared in common – and the author has similar matches to other descendants from many lines descending from the above Johannes Bellinger. Thus, this is a Windecker connection since Henry Windecker's great grandfather was Nicholas Bellinger (again, the uncle of Johannes Bellinger).

Barry Schumaker is a 6th cousin once removed, a descendant of Margaret Windecker (who married Stephen Kitson) daughter of Henry Windecker, sister to Elizabeth (Young) Windecker. Here also there is no connection to the Young family, so the match is Windecker or Pickard.

Anthony Messuri Jr. is a distant cousin, a descendant of Elizabeth Windecker's great aunt Gertrude Windecker (8th cousin once removed) who married Jacob Pickard (7th cousin once removed) the uncle of Elizabeth.

Rashelle Elburg is a known descendant of Daniel and Elizabeth (Windecker) Young.

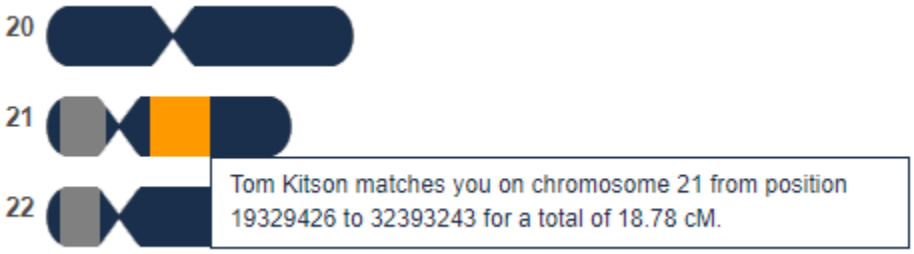
The other individuals shown in the chart above (of which there are not many) are either undocumented Young – Windecker descendants, or perhaps are simply false positives.

Using the chart above it is clear that there are some rather astounding matches to distant kin, but that the fit on chromosome 21 makes perfect sense.

Other cousin matches relating to a match on this chromosome are not found on Gedmatch:

Suzanne Longo, who is a 3rd cousin once removed who tested at 23andMe, matches between 40 Mb and 46 Mb. A closer examination of the data shows that the numbers are actually 40.4 Mb and 46.2 Mb. The match is 14.6 cM, and takes known the Young – Windecker segment to the q end telomere, offering further proof that the entire intact maternal chromosome is indeed from this lineage. The shared segment ends specifically at 46,263,539 for the author, Norm Sones, and Suzanne Longo. Academic research shows that the end of the chromosome is 46,709,983 (each testing company differs to some degree). So for all intents and purposes our match extends to the far q end of chromosome 21.

Tom Kitson, who is a descendant of Margaret Windecker, daughter of Henry Windecker and Dorothy Pickard who married John Kitson (Kitzer), and tested at FamilyTreeDNA.com. A diagram of the match from Family Tree DNA browser is shown below:



<u>Summary Observations Concerning Apparent Segments</u>: Due to the paucity of SNPs from the beginning of the p end of chromosome 21 to about 14.6 Mb; plus the fact that the region between 16,344,186 and 19,375,168 Mb in Build 37 is denominated as a "pile up region" (Li et al., 2014), the region before 19 Mb is of questionable utility. The latter region is where there is excess matching due to "identical by descent (IBD)" areas can be in fact "identical by state (IBS)" meaning random matching with others in the European gene pool.

It appeared earlier that the first Young – Windecker segment could be ascertained by the start positions of the match with Norm Sones (who is both Young and Windecker) and Marti Sigsbee (who is Young only – via the George Landgraff connection which is also that of Catharine Elizabeth (Schremling) Young (whose mother was a daughter of George Landgraff). However the IBS "issue" has resulted in the removal of the match with Marti Sigsbee in Build 37 since it falls within a pile up region and hence is not reliable – although was likely a valid match with the

author (there are zero "strays" in that area of the chromosome of the author). Build 37 has resulted in the most conservative take being used such that the informative matching starts at 19 Mb – where the above match with Tom Kitson begins. Whether the region below 19 Mb is Young – Schremling is unclear – but what is clear is that the segment shared with Tom Kitson at 19.3 cM is valid, and is entirely Windecker in origin (he does not have Young ancestors). This segment merges with the Windecker segment of Kathryn Willson and extends to 38.2 cM. This is precisely the position where the sharing with Norm Sones begins again. This may signal the beginning of a Young segment which extends to the end of the chromosome at about 46 cM. The fact that Norm Sones is descended from Frederick and George, two of the sons of Daniel and Elizabeth (Windecker) Young, and the author is also a descendant of the above George, but also George and Frederick's brother Henry does complicate matters. However it is more likely that what we share (at the beginning and at the end of the chromosome) is via George, our mutual ancestor. The segment at the very end of the q part of chromosome 21 is also shared with Suzanne Longo who is also a descendant of George Young, but as with the author, also George's brother Henry Young. It may be that these two shared segments are from Daniel Young (beginning and end of chromosome). However, the data shows more clearly that the large middle portion is, without a doubt, from Elizabeth Windecker. This segment was perhaps contributed via her son Henry Young, the "other" Daniel and Elizabeth ancestor of the author and Suzanne, but not Norm (although Norm is also a descendant of George and Henry's brother Frederick).

So the entire chromosome may have descended from Henry Windecker – but to add another monkey wrench into the mix, it also may have come in whole or in part from Henry Windecker's wife Dorothy Pickard. However although Anthony Messuri also has Windecker and Pickard ancestors, Joscelyn Malheiro does not – she only has Bellinger ancestors (not Young or Windecker) – her ancestors (shared with the author) being the progenitors in the author's line leading to Henry Windecker.

Some Cautions: While the conclusions below would seem to rest on solid ground, there are a few potential "flies in the ointment". The problem with the p end to 14.6 Mb and 17 to 19 Mb of the q end of the chromosome has been noted above. Furthermore, while not shown in the above Gedmatch table, just below "Norm Sones" (a known Young – Windecker descendant), there is a stack of unknown matches between about 39 Mb and 45 Mb – none of whom have a useful genealogical tree. This is apparently a "pile up region" so may be matches well beyond a genealogical time frame. Li et al. (2014) only identified the segment between 17 and 19 Mb (shared with the author's sister and first cousin) as falling in this category for chromosome 21 – despite having used an array of statistical approaches. However there is no "pile up" evidence relating to anywhere in this area of the author's maternal chromosome 21. Hence the 39 to 45 Mb observation is not easily explained.

Secondly, the distal (telomere) match could have arisen from Terryberry ancestors. Both Norm Sones and Suzanne Longo are, as is the present author, descendants of Mary Terryberry. The

segment shared with Norm and Suzanne is more likely (statistically) to have occurred in a male recombination (as noted above, see Chowdhury et al., 2009). The most probable candidate is George Young (born 1796) "donating" his father Daniel Young or mother Elizabeth Windecker's telomere end to his daughter Elizabeth M. (Young) Young. While there is nothing that would prove or even suggest that all or part of these chunks of DNA are Terryberry derived (rather than Young) – it cannot at present be ruled out.

Conclusions: Combining all of the data above, it can be seen that there appear to be no clear gaps, the author has apparently inherited his maternal chromosome 21 from 5th great grandparents Daniel and / or Elizabeth (Windecker) Young, via his great great grandmother Hannah Adelia (Young) Dawson. This is without a doubt a rare event (statistically improbable), but has been reported by others to at least the great grandparent level. For example, in the blog by Kitty Cooper she has a posting entitled, "*Using the Chromosome Mapper to make a four generation inheritance picture*". Here it was noted that "Byrnne" had four generations of her family tested, meaning all 8 of her great grandparents, and that one observation is that it was, "*Interesting that she has chromosome 21 intact from her paternal great-grandmother*". The author did not have this luxury / blessing of testing even grandparents, but the data still shows clearly (by a more circuitous route) that his maternal great great grandmother was the source of the maternal chromosome 21.

Beyond the great great grandparent Hannah Adelia level it becomes difficult to parse out the contributions of her double great grandparents, the husband (Daniel Young) and wife (Elizabeth Windecker). However, for at least one component we can be certain (via genealogical – DNA matches) that Elizabeth (Windecker) Young was the one who contributed the span from 19,329,426 to 38,077,824 Mb. The origin of the p end and the q telomere segment is more up in the air.

A hypothesis that fits with the data is that of the two contributing sons of Daniel and Elizabeth, George Young (via daughter Elizabeth M. (Young) Young provided the beginning and end (telomere) of the chromosome which the author shares with Norm Sones; and likely George's brother Henry Young (via his son Henry Young Jr.) contributed the mid section, the portion that is shared with Lawrence William Brown (starts 18,344,173 ends 38,239,633).

This pattern of distal end versus central section tallies precisely with the "jungle" areas with relative high probability recombination noted above by Chowdhury et al. (2009). However from Hannah Adelia (Young) Dawson down to the author no further recombination opportunities here or elsewhere were seized upon as it passed to her son Joseph William Dawson, to his daughter Eva Fern (Dawson) Williamson, to her daughter Violet May (Williamson) Faux, to her son David K. Faux.

The entire weight of data poses an interesting question. The evidence shows that a substantial part of this chromosome does not come from Young or the Pickard line (wife of Henry

Windecker). Since both Henry Young Sr. and George Young were **Windecker** descendants and therefore **Bellinger – Kuhn** descendants, and considering the relatively large Bellinger - Kuhn match to 11th cousin Jocelyn Malhiero, it can be asked whether the entire mid portion of the chromosome came down intact from the early 1600s (through many potential recombination possibilities) via **Hans Kuhn** born 1625 and his wife **Catharina (Kuhn) Kuhn** of Huttengesas / Langenselbold, Hessen, Germany. Since they were siblings, it is worth considering whether there was some inherited factor (known from genetic studies) that might have affected recombination (or lack thereof). The author has a number of other matches tracing back to marriages of close kin, generally first cousin marriages, and here also there is a tendency for relatively long segments to be preserved down to the present through many generations (opportunities where recombination could have cut the segment into smaller units). At one level there seems to be some sort of "stickiness" factor at work, where the chromosome had 12 chances to recombine (one recombination per generation is typical). It is more likely that the correct interpretation is that what we are seeing the manifestation of a very low probability occurrence in multiple generations of one family. However, recall that the author's uncle did not inherit any of this chromosome from his mother Eva Fern (Dawson) Williamson, and must have received his chromosome 21 in its entirety from his father Gilbert Williamson.

What can be said with a high degree of certainty is that each SNP on chromosome 21 "experienced" the American Revolution (via Sgt. Daniel Young of Butler's Rangers and / or his father in law Henry Windecker) – in a manner of speaking. Similarly, it must have been present during the War of 1812 (via Henry Young and George Young who were Sergeant's in their father's Regiment). In addition to genetic inheritance, an additional factor is epigenetic inheritance (an ancestor's life experiences impacting descendants through methylation – histone "wrapping" of some DNA strands) which may have had an enduring effect lasting for generations. The result is that some traits or characteristics linked to chromosome 21 are influencing the physiology or behavior of descendants 200 or more years later. While this could be said of any segment or chromosome, it is more persuasive when it can be proven that an entire chromosome was passed to a descendant via one or at most two great grandparents (Daniel and Elizabeth (Windecker) Young) of a great great grandparent (Hannah Adelia (Young) Dawson).

A Question for Consideration: After this study, the most salient personal question is: which of the author's children and grandchildren will have none of his maternal chromosome 21, how many will have some part, and whether perchance one or more will have the entire package? If there is an inherited factor (as described by Coop et al. 2008) inhibiting recombination, it means that the author's maternal chromosome 21 may have a greater than 50% chance of being transmitted intact to some of his children. Hopefully at some point each child and grandchild will opt for DNA testing, and in the process yield an to answer this question.

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