Visual Snow Survey Report 2019

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Abstract:

The Visual Snow, VS, Survey for 2018-2019 has been completed and the data collection process is now closed.

Approximately 700 people responded to the survey initially, and after some people without a clear case of VS withdrew, over 350 completed the survey. This is an exceptionally high percentage for any survey. Individuals took an average of 4.5 minutes to complete the survey.

During the survey, the most important trend was that VS has a significant genetic component that is passed down the female side of the family. It appears there is a mechanism within the respondents DNA that becomes active at a given time, after which the sufferer is susceptible to (develops a propensity for) VS. After that time, if the subject ingests a food, pharmaceutical or recreational drug containing a carboxylic acid group within a larger molecule, he/she is very likely to encounter VS. This chemical group is ubiquitous in our diet alone and it is therefore inevitable that the subject will develop VS shortly after becoming genetically susceptible.

Smoking Marijuana or any other recreational drug alone will not cause VS unless the smoker has an established propensity for Visual Snow.

No short-term cure or treatment has appeared from the Survey. The results continue to show VS is not progressive. Alternately, no case of VS is known to have disappeared.

It is important that anyone with VS that is strongly inclined to seek a cure become affiliated with a research facility within the medical community that has a dedicated research team pursuing VS.

Keywords: visual snow, cannabinoid, marijuana, carboxylic acid, idiopathy, maternal genetics,

Table of Contents

1.0 Introduction

The **Visual Snow Survey** conducted from April, 2018 until April, 2019 has been completed and the data collection process is now closed. This survey is a follow-on to and earlier survey in 2013. It is also a follow-up to the Conference on Visual Snow held at University of California–San Francisco, UCSF, at the Mission Bay Conference Center on 5 May 2018. A charitable foundation , http://VisualSnowInitiative.org was also formed at that conference to further awareness of the disease called **Visual Snow, VS,** in the vernacular. A scientific/clinical name for the disease awaits further definition of the disease itself.

The intent of the survey was to eliminate people with other *primary* complaints including floaters, the blue sky effect (due to the movement of white blood cells within the neural tissue of the retina), Aura, palinopsia, impaired night vision, spontaneous photopsia (bright flashes of light), and seeing white halos around objects and other symptoms. The focus was on Visual Snow as a physiological disease. The statistical results were therefore much more applicable to the researching of VS as a disease.

The purpose of this report is two-fold; first, to provide those with VS a better understanding of the state of knowledge about their community and condition. Second, to provide the medical and academic community focused on VS a better understanding of the state of physiological knowledge supporting the statistical questions and answers produced in the survey.

The overall report consists of three parts;

1. This summary report
2. The statistical report prepared by SurveyMonkey resulting from the Survey.
3. The raw data allowing those interested to explore the Excel file.

Access to this information over the Internet is provided in **Section 7.0**.

2.0 Review of earlier investigations

The author of this report has been fielding questions concerning VS from the public, the medical community and the academic community for almost two decades. With the benefit of the Internet, communiques from around the World have been involved. This led to an on-line survey, in 2013, of the perceptions about their condition held by those with VS.

2.1 Previous activities attempting to characterize VS

The 2013 survey mentioned above was structured to answer a series of basic questions relating to the physiology of VS. Its results will be discussed in **Section 4.1**.

Beginning in 2012 Schankin, Maniyar & Goadsby began a program to fund and implement an exploratory clinical investigation into the psychophysics of VS. They published two papers in 2014 that did not define the source of VS.

In 2018, a philanthropist entered the VS community with a goal of raising interest in advancing the state of public knowledge concerning VS, and implementing an expanded research program leading to an eventual cure for VS. His team developed a plan for and funded a conference to be held in the spring of 2019 at the University of California, San Francisco, UCSF.

2.2 Other activities under way to characterize VS

The University of Sussex[1] in Great Britain has announced (and is seeking participants in) a new and in-depth VS survey as of 7 April 2019. It is very detailed and Neil Salata, a.k.a the VisualSnowMan[2], estimates it will take one to two hours to complete. "The questionnaire will contain on-screen depictions and animations of flashing lights, flickering lights, bright lights, moving narrow striped patterns, moving letters, moving environments and objects moving in environments." The intent is to conclude the survey by December 2019. The results will be available on request from simon.saryazdi@sussex.ac.uk

3.0 The 2018-2019 VS survey

Developed at the time of the UCSF conference was a formal survey using the facilities of Survey Monkey, Inc. The survey was planned to be open until 4 April 2019. The 23 survey questions were optimized based on the 2013 survey and questions raised at the conference. During the one-year period of the survey, 700 people participated. After a preliminary self examination, 369 people completed the survey. The self examination was designed to cull people with symptoms clearly not clinically related to Visual Snow. Of the 369, 193 had reasonably good ideas on when they first encountered Visual Snow, and many associated a specific event with that encounter.

As noted in the detailed questions reviewed below, the demographics of the Survey were quite broad, ranging in age of onset from birth to 45 years old. The age profile is surely skewed by the fact the Survey was conducted over the Internet. 67% of the responses were from males.

Although, the disease is frequently described as a young adult disease, the profile tells a different story. Only 193 of the 369 respondents gave a specific age for their encounter. While incidence does peak in the 16-20 year age group, the overall statistics suggest a Gaussian probability function [See question 8 below]. No respondent indicated encountering the disease at an age over 45. On the other hand, more than 5 respondents indicated being born with the disease. Rather than filling in an age of incidence, many commented on the likelihood of it being present from birth in their comments (Question 19, only available in the Excel worksheet).

The genealogy of respondents

The behavioral aspects of respondents

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[1] https://universityofsussex.eu.qualtrics.com/jfe/form/SV_blrsONUZAQ19BGZ

[2] http://visualsnowman.com

Not a single respondent described their condition as becoming worse with time. Neither did any respondent indicate their VS had gone away. Only a few respondents found a variation in their Visual Snow as a function of time. 85% indicated their VS never disappeared. However, 67% indicated that they forgot about their VS for short periods when concentrating on a task.

The number of respondents also reporting tinnitus was high, 78% , as expected. All of the sensory modalities converge on a few specific areas of the brain.

3.1 Responses to localize the source of VS

Questions one through five were designed to confirm VS did not arise in the eyes themselves or the four quadrants of the visual cortex at the back of the brain. **Figure 1.1.1-1** shows the areas of the visual field defined in the survey questions. The central area 5 was included because this high acuity area of the eye, known as the foveola, is processed separately by the thalamus within the brain. The detailed schematic of the visual modality is addressed in **Section 5** below.

Q1: 98.37% of the respondents (362 of 368) said the VS was not constrained to only one eye.

Q2: 98.37% of the respondents (362 of 368) also said VS was not constrained to the left or right half of the visual field.

Q3: 97.28% of the respondents (358 of 368) also said VS was not constrained to either the upper or lower half of the visual field.

The answers to these question indicate the origin of VS does not arise in either eye either optic nerves, either lateral geniculate nucleus, along Meyer's Loops or any single quadrant of the visual cortex.

Q4: The answer to the question of whether the snow was lower or higher within area 5 compared to the surrounding area was more complex; 71.72% said it was the same, 22% said it was lower and 6% said it was higher.

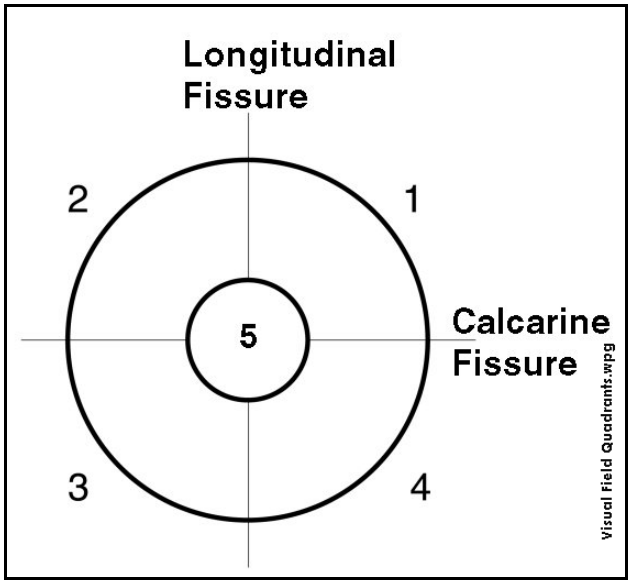

Figure 1.1.1-1 Designated areas of the visual field. The numbers correspond to both the quadrants of the visual field, plus the central high resolution area (5), as well as the quadrants of the visual cortex of the brain plus the special area of the brain labeled the thalamus (5 again). The text labels apply to the bi-sectors of the occipital lobes of the brain. See text.

The answers to question 4 indicate the origin of VS does not arise in the foveola of the eyes, the optic nerves, the perigeniculate nucleus or the sensory side of the thalamus (the thalamic reticular nucleus).

Q5: This question asked whether the VS was more prominent outside of the large circle (the peripheral signal paths) or more prominent within the large circle (the foveal signal paths). 81.68% (165 of 202) said the prominence was the same. 11.39% said lower and 6.93% said higher.

The answer to question 5 suggest again that the source of VS is not in the multiple sensory paths prior to the summation of the abstract information.

An accumulation of responses limited to only one or two of the areas identified in the figure would significantly steer the overall investigation. No such accumulation was encountered in the survey.

Together, questions 1 through 5 support the conclusion that VS arises after the visual data from the above paths is combined into a full perception of the outside environment within the brain . This localizes the search to the the association areas on the left temporal lobe of the brain, to the summation of signal performed within the thalamus, the parietal lobe (potential location of the saliency map storing all sensory information from all senses in an abstract form, or the prefrontal cortex of the brain. The fact the saliency map (where ever it is located) and the prefrontal cortex process information in an abstract form suggest the source of VS is in one of these regions or along the nerves connecting them. Virtually any small group of neurons in these region could introduce uncorrelated noise pulses into the abstract data that would effect the perception of the full visual field.

At the current time, such a small group of neurons is not resolvable with any clinical level of MRI or CAT-Scan equipment. These clinical machines typically provide imagery with a pixel containing not less than 2 million neurons each.

3.2 Responses to factual questions

Q6: Asked how suffers perceived VS as a fuzziness of the surface of objects in their field of view or as a veil outside of their eyes and between their eyes and the objects. 65.83% chose the veil as their descriptor and 34.17% chose fuzziness.

Q7: Asked whether the perceived their VS in color or only as a translucent veil or fuzziness. 27.14% said they perceived a coloration of their VS while 72.86% said their VS was colorless.

The answers to questions 6 and 7 may be useful later in our research once the possible locations of the source of VS are better localized.

Q8: Asked when a sufferer first recognized their visual snow. Many people had a vivid memory of the circumstances as will be discussed below.

Here **Figure 1.1.1-2** is presented to provide more information concerning the common feeling that it is a late teen or twenties phenomenon. As will be discussed below, acquiring the disease is more likely related to puberty than social pressure. The distribution is nearly Gaussian, also described as a Normal Distribution.

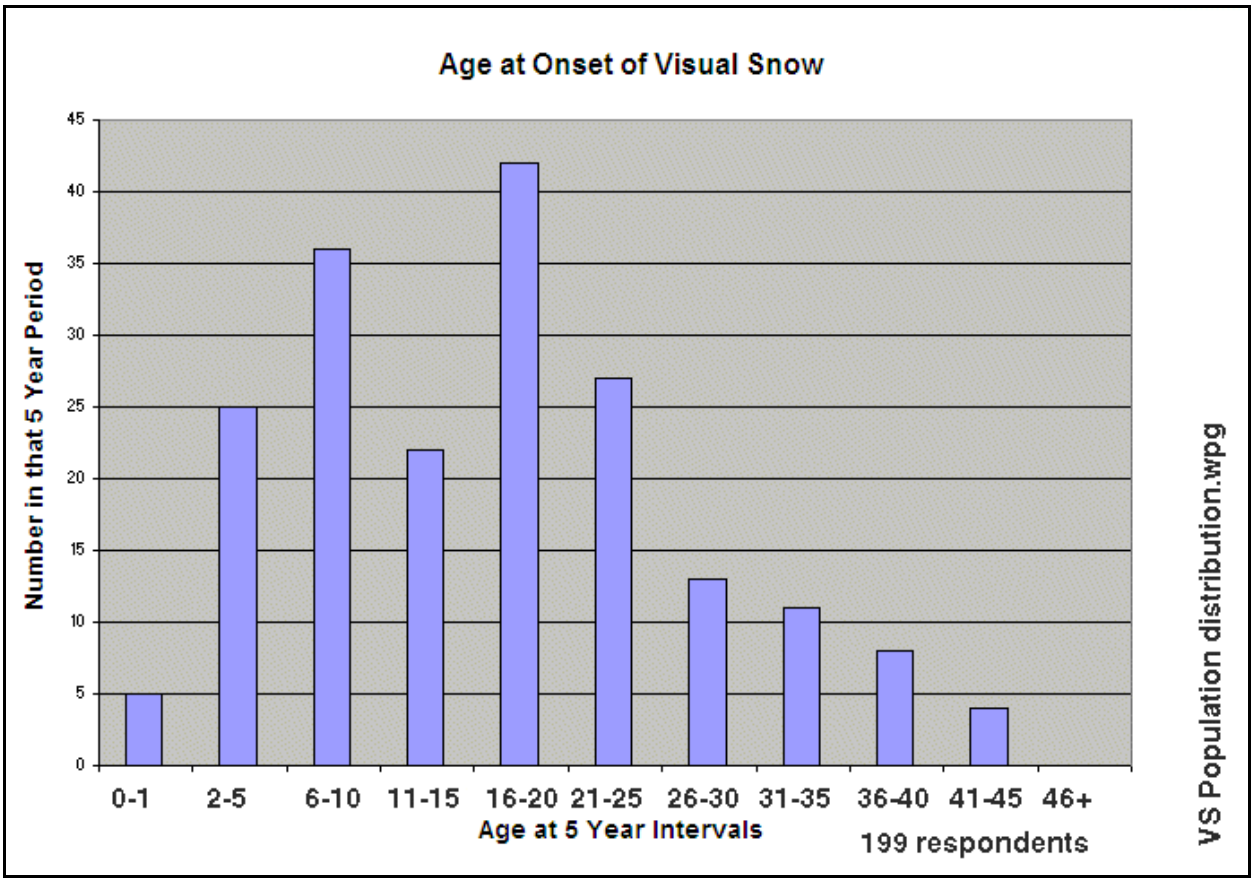

Figure 1.1.1-2 Age of onset of VS from survey. The number in the 0-1 bin is soft because some respondent only expressed their thought informally.

Figure 1.1.1-3 provides another view of the age of onset versus the current age of the respondent. The graph includes an acute line with those VS respondents encountering the disease recently nearest the acute line. While many people are reporting their recent encounter with VS, that may be because of the current discussions on social media (Which tends to be dominated by people in the 15-30 year age group. Note the large number of people reporting the onset of VS before they were 10 years of age. Note also the significant number of respondents with long term (chronic) VS.

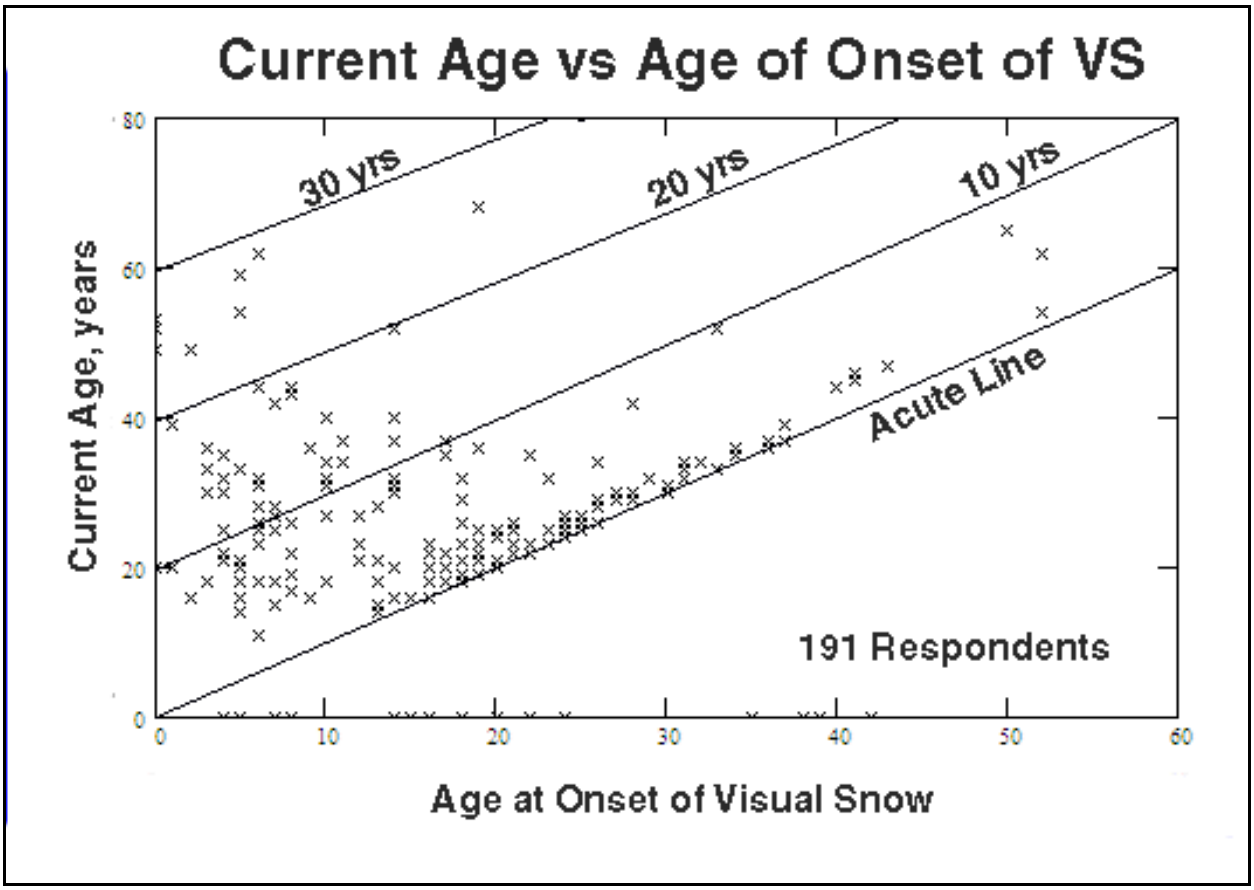

Figure 1.1.1-3 Current age vs age of VS onset. 191 respondents. 4 subjects did not report their age at onset of Vsbut did give their age (data points along the vertical axis). 15 did not report their current but did estimate their age at onset of their VS explicitly. Sometimes, they discussed it in the comments. Many people have had the condition for over ten years as indicated by the diagonal lines. With the growing familiarity of the condition, more people may come forward.

Questions nine through twelve were designed to explore any chemical cause for VS. The questions related to did you or did you not take and complex drugs, whether prescribed or recreational drugs and what were they. The answers were obscured by the number of physicians prescribing tranquilizers or anti-anxiety drugs to people exhibiting anxiety in their offices.

Q9: 194 out of 368 (53%) answered the question about prescription drugs, with 64.95% answering they had not been prescribed any medication. 13.4% said the question was not applicable to them and 21.65% answered yes.

Q10: The type of drugs listed was long. It ranged from penicillin and its modern equivalents (amoxicillin, etc) to the latest NSAID's (non-steroid anti-inflammatory drug) along with a large number of anti-depressants after onset of VS. It also included two cases where Levodopa was prescribed. Levodopa has long been implicated in VS and similar diseases.

The only recognized common element in these medications taken before VS onset; they were all complex organic chemicals; virtually all contained a carboxylic acid group as part of a larger molecule.

Q11: Asked about recreational drugs, 194 of 368 (53%) responded. 114 of the 194 (74.23%) said, they had not used them, 6.19% said the question did not apply to them and 19.59% (38 of 194) said they had used them.

Q12: The list was long, and included a variety of slang names not known to the writer. They included the commonly known cannabinoids (including marijuana), LSD, cocaine and L-Dopa (not usually available without a prescription).

These drugs, when used before VS onset, almost invariably exhibited one or more, frequently many, carboxylic acid groups as part of a larger molecule.

It is commonly heard that VS is a young adult disease and that it is linked closely to the use of recreational drugs. Further analysis of these assertions appears warranted. Therefore, the statistics for the 18-30 year olds were examined more closely. They constituted 67 of 194 (34.5%) responding to Q9 and Q11. These questions were expected to be exploratory in character and the answers reflected that fact. **Figure 1.1.1-4** summarizes the data. The ratio of recreational to prescription drug use in the population of the study responding was 3:4; the ratio of recreational to prescription drug use in the 18–30 yr cohort responding was 5:4.

These statistics, although still of limited precision, suggest;

1. The respondents were not dominated by the 18–30 year cohort of 34.5%.

2. The 18–30 year cohort reporting recreational drug use, 39%, was not dominant over those reporting prescription drug use, 31%, as the potential cause of their VS.

3. The low number of total respondents to these questions, particularly with regard to recreational drug use, suggests the sensitivity of this question in our culture at this time.

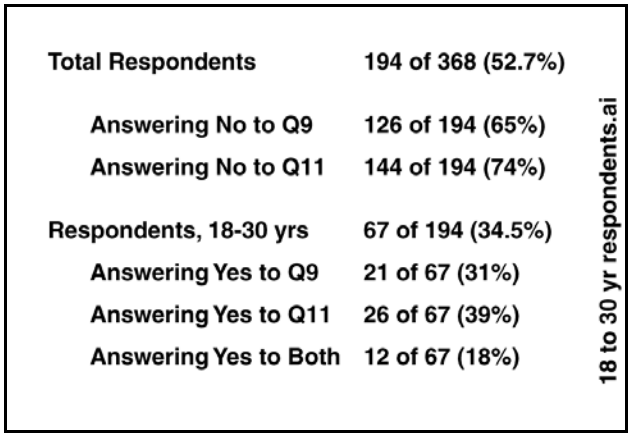

Figure 1.1.1-4 Statistics relating to the 18–30 year old cohort in the study. In the population of the study, the ratio of recreational to prescription drug usage was 3:4. In the 18–30 year cohort, the ratio of recreational to prescription drugs was 5:4. These ratios are of limited statistical value.

4. Levodopa probably should not be prescribed for patients complaining of VS. It was documented as causing short term VS at high dosage levels by Dr. Sacks in the 1960's.

5. Kennamycin probably should not be prescribed to human patients complaining of VS. It is a known danger to the receptors of the hearing modality when prescribed in excessive amounts.

6. The subject deserves follow-up using a more sophisticated protocol.

Q13: Asked if their VS ever disappeared for a few minutes to a few days. 85.13% said it never went away. 14.87% said yes. The question did not explore whether this were related to their

focus on a specific task (concentration) or their level of attention during these intervals.

Q14: As a follow-up to Q13, it asked whether your VS disappears when you are concentrating on a specific task? 66.15% said it never went away to their knowledge. 33.85% said it did.

This answer deserves more attention in future surveys, particularly if the source of VS has been further localized.

Q15: Asked to help confirm a linkage between VS and tinnitus (a neurologically similar, if not identical symptom/disease). The response was very positive, 77.95% confirmed they suffered from tinnitus; only 22.05% said they did not.

The answer to this question further solidifies the proposal that VS arises either in the saliency map (within the thalamus or parietal lobe) or the prefrontal cortex where one physiological fault could easily cause both diseases.

Q16: The respondent were balanced between 62.36% male and 37.64% female. These percentages do not suggest a strong genealogical trend. However, only 178 out of 368 answered the question.

Q17: It provided little direct information. It asked the current age of the respondent.

Q18: Provided new information of considerable significance. It showed a particularly high percentage of at least one sibling having VS, 57.89%, but even more illuminating was that 42.11% of their mother's had VS. Only 10.53% of the fathers suffered from VS. A mitigating factor is that only 19 of 368 respondents addressed the question.

All of the respondents reporting their Mother's exhibited VS were males (6). Of the respondents reporting their siblings exhibited VS, all but three were male (9). Only two respondent reported their father exhibited VS, one female and one male.

The percentages surely warrant more investigation into the possibility that VS is inherited along the female side of the family tree.

3.3 Response to invitation to add comments

Many respondents chose to contribute additional information that will be merely touched on here, but can be reviewed further in response to any particular questions received by email. The comments are also available in the raw data file mentioned in the Introduction. A common theme relates to migraine headaches and the disease, Aura. The subject of migraine headaches remains a subject of significant medical and academic research, but little progress in understanding the condition has been made recently. Aura is clearly a psychotic disease but it is possible its source might be physically near the source of VS. One mention of synesthesia probably supports the source of VS being in the saliency map described in connection with questions Q5 and Q15. Synesthesia involves the misreading abstract information in the saliency map where abstract information from many sensory modalities are stored adjacent to each other.

One respondent mentioned they had purchased a DNA analysis. However, without many people acquiring these analyses and the analyses being studied as a group, there is probably little information that can be gained. This type of analysis will become more important in the near future as more of this type of data becomes available.

Several respondents presented a technique that may be useful to early education professionals. They indicated that at an early age (5-8), they got into discussions of whether they or their playmates (or mothers) could see the "air." Those under ten years of age with VS frequently took the position that they could see the air. This "air" acted as a veil between them and objects in their visual field.

Several respondent made note of various psychotic visual disorders they had encountered. The source of these conditions will be left to a future study.

3.4 **Administrative questions**

Questions 20 through 23 were administrative in nature. Only 137 of 368 were willing to provide an email address to receive a copy of this report. The others will just watch for the results on the Internet. Responses came from all over the World, recognizing the reach of the Internet. However, only 178 of 368 disclosed their Country.

40% of the 178 were from the USA, 10% from the United Kingdom, with 7.3% from Canada, 6.4% from Germany, 5.6% from the Netherlands, 4% from Denmark, 0.56% from Brazil,

4.0 Findings

To the extent possible, the findings will be separated into three groups; a review of the 2013 findings, a review of the 2018 findings and a review of the combined findings.

4.1 Findings from VS 2013 Survey

These findings were summarized in the talk at UCSF[3] on 5 May 2018. The highlights were:

1. The cause of VS was most likely a chemical change in the porosity of the tissue of the thalamus or parietal lobe of the brain involving a very small number of neurons (1 to 100)

2. The resulting chemical environment probably caused a change in the electrical biasing of one or more neurons, resulting in electrical noise being introduced into their electrical output. This is a well known mechanism in electrical engineering, and not unexpected in the electrolytics-based neural system.

3. This change in porosity involved such a small number of neurons that medical imaging equipment was useless in identifying the area of the problem involving only a few neurons.

[3] http://neuronresearch.net/vision/ppt/VS.ppt

4. Progress in identifying the cause of VS will probably involve genetics and/or additional analyses of the chemicals ingested prior to onset of VS.

5. Innovative research requires funding. Funding requires organization & dedication. Funding also requires good statistics to win over supporters.

4.2 Findings from the 2018-2019 Survey

The surprising finding of the 2018 Survey was the prominence of a genetic factor related to the female line of genetic inheritance. While not statistically determinative, this finding deserves follow-up with genetic specialists and possibly further inquiry into the mitochondrial DNA of these individuals and their relatives. 23 & me provides an introduction to the female line of inheritance[4].

The range of pharmaceutical drugs, recreational drugs, and foods that were associated with VS was also surprising. The pharmaceutical group included penicillin and its modern sibling, amoxicillin. Statistically, it was clear that essentially all of these ingestables incorporated at least one single chemical group, carboxylic acid. The commonality of this chemical group also indicated that it could not be a determinative factor in the initiation of VS.

The genetic factor suggested two potential elements worthy of further investigation; whether there was a proclivity of some individuals to exhibit a genetic proclivity to VS, and whether this proclivity was activated at a given time in life by another element of the genetic code yet to be determined.

The statistics related to onset of VS and the length of the disease after acquisition are worthy of further analysis, but the concentration around the 20-30 years of age group is probably heavily weighted by the adoption of the Internet by this age group and the much wider awareness of VS as an identifiable disease.

4.3 **Combined Findings from both VS Surveys**

The surveys were largely complimentary but the second provided valuable and traceable statistics relating to VS. The 2018-2019 survey was designed to follow up on a number of earlier questions and provide broader, statistically adequate information in all areas (without an admixture of other diseases in the data). It also provide additional detail in statistical form about the Onset of VS versus the age of the individual. The 2018-19 survey largely confirmed the first three conclusions of the **2013 survey**;

1. The cause of VS most probably involves a change in porosity of a very localized area of the brain.

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[4] https://customercare.23andme.com/hc/en-us/articles/212880257-Maternal-Haplogroups-Diving-Deeper

2. The change in porosity occurs in such a small area that it will not be identifiable using non–invasive medical imaging equipment for at least another ten years.
3. Progress in identifying the cause of VS will probably be focused on genetics or chemical analyses of the foods and/or drugs that patients ingested prior to onset of VS

Additional findings from the **2018–2019 Survey** included;

1. The high incidence of mother and siblings sharing the disease of VS indicated a strong likelihood that a genetic factor(s) within mitochondrial DNA, mtDNA, will ultimately lead to the cause of VS. The genetic data being collected by Ancestry.com, 23 & Me.com and others will be invaluable as a source of data mining as the database expands and is collated.
2. The reported wide range of foods and drugs ingested prior to onset of VS indicates such ingestion is not a causal factor in the initiation of VS. Further analysis of the raw Survey data will shed additional light on whether recreational drugs played a disproportionate role in the acquisition of VS among the 20-30 year old group. 72% of the total cohort denied use of any recreational drug in the month prior to VS onset, whereas 65% said they did ingest prescription medications during that month.
3. The statistical evidence indicating the source of VS can be traced to the sensory association areas of the brain, the temporal lobe, the thalamus or parietal lobe. It does not arise in the eyes, the lateral geniculate nuclei, the occipital lobe of the brain or the interconnections between these entities.
    3A. The basic cause of VS arises in a specific type of neuron used to propagate signals between individual neural *engines* of the brain. These engines typically incorporate 2 million or more of information extraction neurons.

    3B. The cause of VS arises among only a few stage 3 neurons that generate monopulse signals known generally as “Action Potentials.”

    3C. The cause of the noise introduced into the system by these stage 3 neurons is a mis-biasing of the amplifier within the neuron(s). The fuzzy character of that noise is diagnostic. It is introduced at the input terminals of the encoding neurons of stage 3.

4. The fuzzy character (a specific temporal frequency distribution) of the reported VS continues to suggest the origin of VS; it almost certainly arises in a nerve of the commissure class (stage 3 neuron in the Electrolytic Theory of the Neuron). A commissure is a nerve bundle within the Central Nervous System (the brain). The origin may include many neurons, or only one, within a nerve bundle. There appears to be no need for more than one commissure to be involved.

5. The number of respondents reporting some degree of color in their VS, 27%, was higher than expected and suggests a lower frequency cutoff to the pink noise spectrum into their abstract information stream. This lower cutoff would lead to larger perceived “snow” granules that are more likely to allow the perception of color.

The search for only a specific neuron or small group of neurons out of the billions in the brain is well beyond the capability of medical and research science at the current time.

Non-invasive imaging of the MRI and CAT varieties will remain useless for locating the source of VS for at least another decade. The minimum pixel size in these imaging technologies is currently about 4-8 million neurons. OCT imaging is limited to the retina of the eye, which is not a viable location for the origin of VS.

4.4 Common experience when seeking medical help

Most of those with VS report a common scenario;

1.They visit a general medical practitioner; he/she finds nothing wrong and sends them to,
2. An Ophthalmologist or an Optometrist;, they find nothing wrong and sends them to,
3. A neurologist; he/she finds nothing wrong medically and sends them back to the practitioner.
4. As a result,

- One of the medical people prescribes an anxiety-suppressing medication and sends the patient on their way.
- The patient leaves with the feeling that, all the medical people think he is crazy.

Although the author is not a medical professional, a person with VS can tell if they are on the right track if they hear the following terms in the order listed;

- Neuropathy,
- Thalamo-neuropathy, thalamic-neuropathy or parietal neuropathy
- Neuropathy of the ganglion neurons
- and finally, idiopathy or idiopathic neuropathy (unknown source of neuropathy).

If anyone with VS receives a diagnosis of an idiopathic disease, and the person wants to pursue a cure for the condition, they should affiliate with a nearby research program, probably associated with a teaching hospital associated with a university. While no cure for VS is on the horizon, increased awareness among the medical community is spawning more intense research.

The terms retinopathy, MRI scan, CAT scan, or other imaging scans) are not believed to be appropriate for VS as a disease at this time and for at least five more years.

5.0 Potential sources of VS (of interest to the research community)

5.1 Isolation of the source of VS within the visual modality

Figure 1.1.1-5 shows the overall schematic of the visual modality used in this analysis.

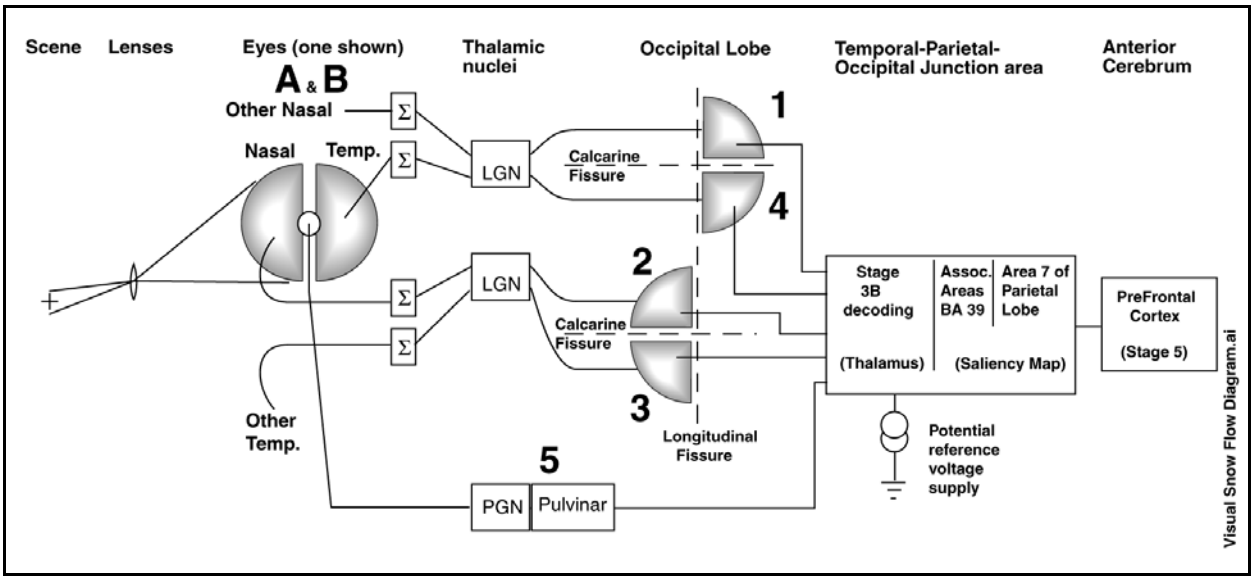

Figure 1.1.1-5 Major elements of the visual modality used to isolate the cause of visual snow.

The system is redundant in many ways to protect the visual capability. Each eye provides signals from a large area shared with the other eye. The signals from **A** & **B** are sent to two separate elements known as the lateral geniculate nuclei, LGN. The signals collated by the LGN are then sent to four distinct information extraction areas of the occipital lobe, numbered **1** through **4**. Meanwhile, the signals generated by the photoreceptors of the foveola travel a separate route via the perigeniculate nucleus, PGN and the pulvinar designated path 5.

The signals from the above information extraction elements, 1 through 5, first come together in the association areas of the temporal lobe and area 7 of the parietal lobe after passing through the "switchboard" portion of the thalamus.

Questions 1 through 5 allow the investigator to ascertain that the source of VS does not arise along paths **1** through **5**. VS arises within one of the areas following the thalamus. The current challenge is to further isolate the source with the current limitations on our knowledge of the way highly abstract information is treated within the brain.

5.2 Potential chemical causes of Visual Snow

The findings related to questions Q9 through Q12 were quite surprising. From a chemical perspective, they ranged from relatively simple molecules of known impact on the human system, to more complicated molecules not generally regarded as harmful to man, to the complex molecules associated with the cannabinoids (with highly arguable virtues and dangers).

The most surprising finding was the common association of penicillin and its more recent derivatives, including amoxicillin, as a potential cause of VS. But a more useful input was

concerning the chemical L-Dopa. L-Dopa has been known, since the work of Oliver Sacks[5] in the 1960's, to cause VS for indeterminate lengths of time. The L-Dopa is molecule is quite simple, consisting of only 25 atoms, $C_9H_{11}NO_4$ and exhibiting the ability to cross the blood-brain-barrier quite easily. Thus, it is worth looking at more closely. It contains only two known chemical groups, a carboxylic acid group and a glucol group (not to be confused with a glycol group). **Section 3.5** in "The Neuron & Neural System[6]," N&NS, reviews the role of L-Dopa and its derivatives in some detail as does **Section 1.8.6.4** in "Processes in Biological Vision[7]," PBV.

Interestingly, the carboxylic acid group found in L-Dopa is also a cornerstone in the structure of penicillin and its derivatives. Penicillin comes in a wide variety of similar compounds as well as a variety of derivatives, including the widely prescribed amoxicillin (28 million prescriptions in the USA in 2016) that also exhibits a prominent carboxylic group.

Other members of this family that showed up in the survey include; Ampicillin, Carbenicillin (Geocillin), Piperacillin / Tazobactam (Zosyn), Amoxicillin / Clarithromycin / Lansoprazole (Prevpac), and Ampicillin / Sulbactam (Unasyn) with trade names shown in parentheses. They have not been examined as to their carboxylic acid content.

Moving up the chemical complexity ladder, the cannabinoids frequently contain multiple carboxylic acid groups. The cocaines exhibit groups that may be easily modified to exhibit one or more carboxylic acid groups. Lysergic acid also contains a readily available carboxylic group but its more well known derivative Lysergide, LSD, does not. Hower LSD could easily be reduced to lysergic acid within the body.

The result is a trail of research into the role of carboxylic acid in VS is well worth more sophisticated analysis. The problem is that carboxylic acid (not to be confused with carbonic acid) is widely available in foodstuffs, although not necessarily in high concentrations after passing through the digestive track

5.3 Genetic research leading toward understanding Visual Snow

Looking at Q18, the potential for one or more genetic factors relating to VS, appears high. Although the number of respondents mentioning a familial situation involving VS, the percentages point toward an inheritable genetic factor(s) along the female line of the family tree. Unfortunately, it is an early day in widespread genetic testing and there is very little historical data among family records. And, the rarity of VS, sufficient to be considered an orphan disease by most health agencies around the world makes data collection haphazard.

A feature of the genetic code that is just becoming important in research is the recognition that many, if not all, genes are controlled as to when they are active. As an example, the genes

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[5]Sacks, O. (2012) Hallucinations NY: Alfred A. Knopf

[6]http://neuronresearch.net/neuron/

[7]http://neuronresearch.net/vision

related to puberty are always present in a persons genome, but they are only activated at specific times by “activators” or “suppressors.” It is reasonable to propose that the timing of any genetic propensity toward VS may be controlled by one of these activators.

5.4 Formally naming Visual Snow

The findings of this survey continue to point to VS as a *physiological* disease and not a *psychotic* disease (such as the class of hallucinogen persisting perception disorder, HPPD).

While the designation “visual snow” resonates with the older population who remember analog television before the early 2000's. It has less relevance among the younger generation. However, the visual snow label is likely to endure.

It should be made clear the disease under discussion has ***nothing to do with the eyes*****!** Therefore, an initial technical descriptor should relate to neuropathy rather than retinopathy. A prefix to neuropathy should describe where the neuropathy occurs. This is largely unknown at present, except that it is a disease of the Central Nervous System (the brain). An additional prefix should probably define the type of neuron exhibiting this neuropathy. The character of the noise spectrum perceived in VS can be traced to a stage 3 neuron generally labeled a ganglion neuron within the brain. The same ganglion neurons are also employed in the eye. *The ganglion neurons of the eye are not of concern in VS.* The ganglion neurons operate in the pulse mode and generate “Action Potentials.” These neurons are susceptible to noise in their input circuits.

> For any researcher interested in understanding this esoteric area of spectral noise generation, **Section 9.6** discusses it more broadly. The author would be happy to correspond with any researcher on this subject, jtfulton@neuronresearch.net.

An error in the bias *involving only one neuron* could account for all of the fuzziness in a full visual field visual perception. This neuron would almost certainly be associated with extracting information from the abstract information stored in the saliency map. This map is probably housed in the parietal lobe of the CNS. The neuron would be a stage 3A encoding type large pyramid cell operating as a ganglion pulse-encoding neuron.

The cumulative evidence relating to VS suggests a preliminary technical name for VS might be a “specific ganglion neuron neuropathy of the parietal lobe.” The designation, parietal lobe, is a stand-in, based on less substantial evidence, for the parietal lobe, left temporal lobe or thalamus.

6.0 Currently Leading Scenario for the cause of VS (of interest to the research community)

One potential cause of VS can be outlined as involving a genetic mutation in the female genetic lineage that is controlled as to when it is active, and that propensity involves or controls the ability of a molecule(s) of a carboxylic acid group to interfere with the neurons in a finite area of the brain. It could perform this interference by physically interfering with the mechanism associated with the surface (lemma) of a single neuron, or by changing the physical or chemical environment within the matrix surrounding one or more neurons.

There is more than sufficient evidence that the carboxylic acid group of L-Dopa can, and does when prescribed medically, interfere with the electrolytic biasing of individual neurons. In fact, that is its dominant role in medicine today. It is prescribed to slow the operation of the skeletal muscle system in Parkinson's Disease and to slow the operation of the ocular muscles of the eyes in certain un-named medical conditions related to ocular tremor.

As noted in the application of L-Dopa to the above condition, stereo-chemistry introduces an additional complicating factor. D-Dopa does not have the same effect as L-Dopa even though it has the identical connection between the atoms of each molecule. A further complication is that the methods used to electrolytically bias specific neurons or types of neurons is unknown. From experience in electrical engineering, the shopping list of effective methods are likely to be endless.

In summary, the current leading theory of the cause of VS involves three factors.

1. A genetic code, propagated along the female branch, mtDNA, of the genetic code leading to an individual.

2. A propensity, controlled by another element of the same genetic code, to have a small area of the central nervous system, CNS, react to the presence of a specific stereo-chemical form of a molecule with a carboxylic acid group on its outer surface.

3. The ingestion of such a stereo-chemically specific molecule, probably not often via digestion, resulting in such concentration within the thalamus, temporal or parietal lobe of the CNS to allow it to interfere with the electrolytic biasing of one or more neurons ***permanently***.

6.1 Alternate scenarios for the cause of VS

There are undoubtedly modifications to the Leading Scenario discussed above, but there are no known alternative theories supported in any detail.

7.0 Obtaining a complete copy of the 2019 Survey & related material

Additional copies of this report can be downloaded from the website, in three forms;
• This summary report at http://neuronresearch.net/vision/pdf/VSsurvey_2018_Report.pdf.
• The survey in text form with graphics but without answers to questions requiring a typed answer, http://neuronresearch.net/vision/pdf/VSsurvey_2018.pdf The major graphics responding to text entries have been included in the summary Report.

• The answers to all question, multiple choice and entered text, are available without graphics in an Excel spreadsheet, http://neuronresearch.net/vision/pdf/VSsurvey_2018.xlsx You will have a choice whether to open the file or save it to your computer.

• Other material on the broader subject of vision can be found at
http://neuronresearch.net/vision/