Neurofibromatosis Type II (NF2), is a condition that affects an individual with tumors throughout their life. NF2 is sometimes also known as:

- MISME Syndrome (Multiple Inherited Schwannoma, Meningioma, and Ependymoma)
- Bilateral Acoustic NF (BAN)
- MERLIN derived from the Moesin-Ezrin-Radixin-Like Protein, also called Schwannomin, is the missing or nonfunctional protein that causes NF2 tumor growth.

Neurofibromatosis Type II (NF2) is a rare genetic autosomal dominant, neurological, and neurodegenerative, tumor predisposition condition which results in slow-growing typically benign (noncancerous) tumors, primarily located in the central nerve system (CNS); brain and spinal cord. NF2 typically cause numerous issues during an individual's lifetime. However, not all issues are tumor related. Tumors can occur in other parts of the body not just the CNS but can also include tumors along the peripheral nervous system (PNS) along; arms, legs, and other regions of the body. PNS tumors can include tumors seen as firm bumps under the skin. NF2 tumors can result in different forms of nerve, brain, and body damage. NF2 is a neurological disorder that results in tumor growth as well as eye issues unique to NF2; some are unrelated to tumor nerve damage, such as juvenile cataracts.

There is no cure for NF2. A cure for NF2 would need to be a treatment that:
- permanently stops tumor growth
- prevent additional tumors
- destroy all of what might be potentially hundreds of tumors an individual may have
- manage tumors without dangerous side effects

Treatment options available today can only treat one tumor at a time. Surgery to completely remove a tumor, for individual tumor management is the only way to guarantee a tumor will do no additional damage.

While NF2 is a genetic condition, fifty percent (50%) of people born with NF2 are the result of spontaneous mutation, no family with NF2. Since NF2 is rare, it makes it difficult for early intervention of issues. Delays of problematic tumors can result in serious consequences.

Tumor growth rate at different points in the life of an individual will vary; the reasons may include; exact NF2 mutation, hormones, and life/environment factors. Each tumor in an individual will also grow at different rates.

**NF2 gene and MERLIN**

The NF2 gene provides instructions for the production of a protein called Merlin (moesin-ezrin-radixin-like protein), also known as Schwannomin. This protein is made in the nervous system, particularly in specialized cells called Schwann cells that wrap around and insulate nerves.

Merlin helps regulate several key signaling pathways that are important for controlling cell shape, cell growth, and the attachment of cells to one another (cell adhesion). This protein functions as a tumor suppressor, preventing cells from growing and dividing too fast or in an uncontrolled way.

The NF2 gene mutations that cause Neurofibromatosis Type 2 are classified as Germline, which means they are present in all of the body's cells. Most NF2 gene mutations result in an abnormally shortened version of the merlin protein. This short protein cannot perform its normal tumor suppressor function in cells. Research suggests that the loss of Merlin's function allows certain cells in the nervous system, especially Schwann cells, to multiply too frequently and form tumors.

More than four-hundred (400) mutations in the NF2 gene have been identified in people with the condition Neurofibromatosis Type 2 (NF2).

**Cytogenetic Location:** 22q12.2

**NF2 Health Problems**

The different mutation types of the NF2 condition alone does not determine what issues an individual may or may not have to face. Knowing the possibilities in advance can help raise personal awareness of doctor types an individual should see, frequency, and have the information at hand for questions during doctor visits and final selection in treatments or other things that can be done.

**Typical Issues**

- Tumor Growth
- CN8 damage: Loss of Hearing and Balance (Disequilibrium)
- Tinnitus
- Headaches
- Eye Problems/Vision Issues

**Common Issues**

- CN7: Facial Nerve damage
- Permanent Side Effects from Treatments
- Aid in Nerve Damage Recovery
- Poor Immune System
- Pain: Physical and Emotional

**Possible Issues**

- Cerebrospinal-Fluid Flow (CSF) Issues
- Kidney Failure
- CNS: Trigeminal Neuralgia
- CN10: Vagus Nerve damage
- Swallowing Difficulties
- Additional Cranial Nerve Damage
Case report of a Meningioma in a rare location for meningioma growth, but it includes a good description of the general development and locations of meningioma, photos of tumors, and brain MRIs.

2. Rare cranial nerve schwannomas: A retrospective review of nontrigeminal, nonvestibular cranial nerve schwannomas.

http://www.ruralneuropractice.com/article.asp?issn=0976-3147;year=2018;volume=9;issue=2;spage=258;epage=263;aulast=Deora

"Intracranial nerve schwannomas are slow-growing benign tumors that arise from Schwann cells. Most of these (60%) arise from cranial nerves with a preponderance of origin from the sensory nerves. Motor nerves give rise to schwannomas usually in association with neurofibromatosis.

Among the nonvestibular schwannomas, trigeminal nerve predominates with an incidence of 0.8%-8% of the total intracranial nerve schwannomas. Other nerve schwannomas are thus exceedingly uncommon and in the descending order of frequency involve the glossopharyngeal, vagal, facial, accessory, hypoglossal, oculomotor, trochlear, and abducens nerves.

Olfactory nerve as an extension of central nervous system (CNS) lacks Schwann cells. Nerve sheath tumors arising from the anterior cranial fossa (ACF) base are uncommon. They usually arise in relation to olfactory groove. Hence, these groove schwannomas are rarely reported."
3. Tongue Schwannomas Associated with Neurofibromatosis Type 2.

Highlights:
- Neurofibromatosis type 2 (NF2) is a rare genetic disorder
- Oral manifestation in patients with NF2 is rare in the clinical setting
- We report tongue schwannomas in a 36-year-old woman with NF2
- This is the first report of histologically confirmed NF2-related oral schwannoma

4. No Longer Living in the Dark After Receiving Houston’s First Bionic Eye.
http://blog.memorialhermann.org/no-longer-living-dark-receiving-houstons-first-bionic-eye/

First Argus II Retinal Prosthesis System, a retinal implant commonly referred to as the “bionic eye.” Implant for people with severe to profound retinitis pigmentosa.

5. GET Tinnitus and Eye Movement.
http://www.arquivosdeorl.org.br/additional/acervo_eng.asp?Id=451

Hearing loss from Cochlear Nerve damage does not mean the end of Tinnitus. The Subjective Tinnitus becomes Gaze Evoked Tinnitus (GET).

6. GET Tinnitus and Hearing Implants.
https://journals.lww.com/otology-neurotology/Abstract/2017/01000/Tinnitus_Suppression_After_Auditory_Brainstem.18.aspx

ABI's don't help with just sound, they can help manage tinnitus.
Hearing loss from Cochlear Nerve damage does not mean the end of Tinnitus. The Subjective Tinnitus becomes Gaze Evoked Tinnitus (GET). Some sources inaccurately assume surgery is the only time GET may develop.

7. Swallowing Exercises for Dysphagia.
https://www.verywell.com/swallowing-exercises-3146018

Dysphagia is the medical term used to describe difficulty swallowing. Dysphagia includes difficulty starting a swallow (called oropharyngeal dysphagia) and the sensation of food being stuck in the neck or chest (called esophageal dysphagia).
The most common neurological conditions associated with dysphagia include:
- Stroke
- Head trauma
- Multiple sclerosis
- Cerebral palsy
- Dementia
- Tumors of the brain or spinal cord
- Cervical spine injury
- Motor neuron disease
- Myopathy

(Follow link to read about exercises.)

8. The World’s First Bionic Kidney Is All Set to Replace Dialysis in Just Two Years.
Troab. (April 2018)

Individuals with Neurofibromatosis (NF) are at risk of Kidney Failure as a result of treatments to manage health. While waiting for alternatives to things that leave an individual at risk for more damage to kidney’s a possibility to manage kidney issues easier and longer is not far away with an implant for dialysis.
The kidneys are a pair of vital organs that perform many functions to keep the blood clean and chemically balanced. It is easier to maintain healthy kidneys than it is to recover from poor kidney health. Some of the treatments that can damage the kidneys include:
1. MRI Contrast Injection Contrast (Gadolinium)
2. CT (CAT) Scan Contrast Injection Contrast
3. Tumor Drug Treatments
4. Pain Medication: Ibuprofen (Motrin)
5. High Hemoglobin - Overconsumption of Iron in food or vitamins https://www.nf2is.org/kidney_failure.php


10. Newly-discovered human organ may help explain how cancer spreads.
New Scientist. (March 27, 2018)

“A newly discovered network of fluid-filled channels in the human body may be a previously-unknown organ, and it seems to help transport cancer cells around the body.”

“But as well as protecting organs, the network may also aid the spread of cancer. When Theise’s team looked at samples taken from people with invasive cancers, they found evidence that cancer cells that had worked their way out of their original tissues could find their way into these channels, which took them directly to the lymphatic system.”

11. Integration of cancer associated genes from human genome by manual curation of biological databases.

“Cancer has remarkable molecular circuitry connections. Cancer treatment is complex and depends on a number of factors, including genetic, transcriptomic, epigenetic and environmental factors. Advanced Cancer therapeutics research requires molecular data integration and network and pathway analysis. It is essential to know the genes and proteins involved in cancer networks and pathways for the better treatment of cancer.”

Read further for genes involved in cancer and public biological databases material for the cancer associated genes in the human genome.

http://jmg.bmj.com/content/52/10/699.long

“Noefibromatosis 2 (NF2) is an autosomal-dominant tumor predisposition syndrome characterised by bilateral vestibular schwannomas, considerable morbidity and reduced life expectancy. Although genotype–phenotype correlations are well established in NF2, little is known about effects of mutation type or location within the gene on mortality. Improvements in NF2 diagnosis and management have occurred, but their effect on patient survival is unknown.”

13. Risk of a second breast cancer can be better quantified in women carrying a BRCA mutation.
The European Cancer Organisation (ECCO). Science Daily. (March 21, 2018)
https://www.sciencedaily.com/releases/2018/03/180321091354.htm

The risk of a second breast cancer in patients with high-risk BRCA gene mutations can be more precisely predicted by testing for several other genetic variants, each of which are known to have a small impact on breast cancer risk.

14. Researchers observe the switching of Ras protein in detail.
https://www.sciencedaily.com/releases/2018/03/180321094742.htm

Using a combination of methods, the team confirmed the hypothesis that the binding partner of Ras in its bonded form does not contain any hydrogen atoms in the phosphate groups.

15. EPH Receptor Signaling as a Novel Therapeutic Target in NF2-deficient Meningioma

“Meningiomas are the most common primary brain tumor in adults, and somatic loss of the neurofibromatosis 2 (NF2) tumor suppressor gene is a frequent genetic event. There is no effective treatment for tumors that recur or continue to grow despite surgery and/or radiation. Therefore, targeted therapies that either delay tumor progression or cause tumor shrinkage are much needed. Our earlier work established mammalian target of rapamycin complex mTORC1/mTORC2 activation in NF2-deficient meningiomas.”
“Co-targeting mTORC1/2 and EPH RTK/SFK pathways could be a novel effective treatment strategy for NF2-deficient meningiomas.”

[https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5723899/](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5723899/)

NF2 gene inactivation has been reported in a significant proportion of sporadic meningiomas, vestibular schwannomas and malignant mesotheliomas. Furthermore, the protein product of the NF2 gene, Merlin, has been found to antagonize tumor initiation and/or progression in breast, colorectal, prostate, hepatobiliary and medullary thyroid cancers as well as in glioblastoma and melanoma. Therefore, there is tremendous interest in understanding the precise roles that NF2 gene loss and the protein Merlin play within diseased and normal cells, respectively, in order to develop new therapies for cancer treatment.

17. NF2/Merlin Inactivation and Potential Therapeutic Targets in Mesothelioma.

Abstract:
“The NF2 gene encodes merlin, a tumor suppressor protein frequently inactivated in schwannoma, meningioma, and malignant mesothelioma (MM).

The sequence of merlin is similar to that of ezrin/radixin/moesin (ERM) proteins which crosslink actin with the plasma membrane, suggesting that merlin plays a role in transducing extracellular signals to the actin cytoskeleton.

Merlin adopts a distinct closed conformation defined by specific intramolecular interactions and regulates diverse cellular events such as transcription, translation, ubiquitination, and miRNA biosynthesis, many of which are mediated through Hippo and mTOR signaling, which are known to be closely involved in cancer development.

MM is a very aggressive tumor associated with asbestos exposure, and genetic alterations in NF2 that abrogate merlin’s functional activity are found in about 40% of MMs, indicating the importance of NF2 inactivation in MM development and progression.

In this review, we summarize the current knowledge of molecular events triggered by NF2/merlin inactivation, which lead to the development of mesothelioma and other cancers and discuss potential therapeutic targets in merlin-deficient mesotheliomas.”

18. Neoplastic myelopathies.

This article discusses the diagnosis and management of neoplasms that affect the spinal cord as well as spinal cord disorders that can occur due to cancer treatments.

19. The location of constitutional neurofibromatosis 2 (NF2) splice site mutations is associated with the severity of NF2.
[http://jmg.bmj.com/content/42/7/540](http://jmg.bmj.com/content/42/7/540)

“Neurofibromatosis 2 (NF2) patients with constitutional splice site NF2 mutations have greater variability in disease severity than NF2 patients with other types of mutations; the cause of this variability is unknown. We evaluated genotype-phenotype correlations, with particular focus on the location of splice site mutations, using mutation and clinical information on 831 patients from 528 NF2 families with identified constitutional NF2 mutations. The clinical characteristics examined were age at onset of symptoms of NF2 and number of intracranial meningiomas, which are the primary indices of the severity of NF2. Two regression models were used to analyse genotype-phenotype correlations. People with splice site mutations in exons 1–5 had more severe disease than those with splice site mutations in exons 11–15. This result is compatible with studies showing that exons 2 and 3 are required for self-association of the amino terminal of the NF2 protein in vitro, and that deletions of exons 2 and 3 in transgenic and knockout mouse models of NF2 cause a high prevalence of Schwann cell derived tumors.”

20. Lung Cancer Patients Live Longer with Immune Therapy.
NY Times. (April 2018)

Early intervention.
Odds of survival can greatly improve for people with the most common type of lung cancer if they are given a new drug that activates the immune system along with chemotherapy, a major new study has shown.

The findings, medical experts say, should change the way doctors treat lung cancer: Patients with this form of the disease should receive immunotherapy as early as possible.

21. The Role of Inflammation in Subventricular Zone Cancer.

Definition:
The subventricular zone (SVZ) is a term used to describe both embryonic and adult neural tissues in the vertebrate central nervous system (CNS).

Abstract:
"The adult subventricular zone (SVZ) stem cell niche has proven vital for discovering neurodevelopmental mechanisms and holds great potential in medicine for neurodegenerative diseases. Yet the SVZ holds a dark side - it can become tumorigenic. Glioblastomas can arise from the SVZ via cancer stem cells (CSCs). Glioblastoma and other brain cancers often have dismal prognoses since they are resistant to treatment.

In this review we argue that the SVZ is susceptible to cancer because it contains stem cells, migratory progenitors and unusual inflammation. Theoretically, SVZ stem cells can convert to CSCs more readily than can postmitotic neural cells. Additionally, the robust long-distance migration of SVZ progenitors can be subverted upon tumorigenesis to an infiltrative phenotype. There is evidence that the SVZ, even in health, exhibits chronic low-grade cellular and molecular inflammation. Its inflammatory response to brain injuries and disease differs from that of other brain regions. We hypothesize that the SVZ inflammatory environment can predispose cells to novel mutations and exacerbate cancer phenotypes. This can be studied in animal models in which human mutations related to cancer are knocked into the SVZ to induce tumorigenesis and the CSC immune interactions that precede full-blown cancer. Importantly inflammation can be pharmacologically modulated providing an avenue to brain cancer management and treatment.

The SVZ is accessible by virtue of its location surrounding the lateral ventricles and CSCs in the SVZ can be targeted with a variety of pharmacotherapies. Thus, the SVZ can yield aggressive tumors but can be targeted via several strategies."

22. Familial Syndromes Involving Meningiomas Provide Mechanistic Insight into Sporadic Disease.

Abstract:
"Currently, there is an incomplete understanding of the molecular pathogenesis of meningiomas, the most common primary brain tumor.

Several familial syndromes are characterized by increased meningioma risk, and the genetics of these syndromes provides mechanistic insight into sporadic disease. The best defined of these syndromes is neurofibromatosis type 2, which is caused by a mutation in the NF2 gene and has a meningioma incidence of approximately 50%. This finding led to the subsequent discovery that NF2 loss-of-function occurs in up to 60% of sporadic tumors.

Other important familial diseases with increased meningioma risk include nevoid basal cell carcinoma syndrome, multiple endocrine neoplasia 1 (MEN1), Cowden syndrome, Werner syndrome, BAP1 tumor predisposition syndrome, Rubinstein-Taybi syndrome, and familial meningiomatosis caused by germline mutations in the SMARCB1 and SMARCE1 genes. For each of these syndromes, the diagnostic criteria, incidence in the population, and frequency of meningioma are presented to review the relevant clinical information for these conditions.

The genetic mutations, molecular pathway derangements, and relationship to sporadic disease for each syndrome are described in detail to identify targets for further investigation. Familial syndromes characterized by meningiomas often affect genes and pathways that are also implicated in a subset of sporadic cases, suggesting key molecular targets for therapeutic intervention. Further studies are needed to resolve the functional relevance of specific genes whose significance in sporadic disease remains to be elucidated."

23. The association of NF2 (neurofibromin 2) gene polymorphism and the risk of medulloblastomas.
https://link.springer.com/article/10.1007/s10072-018-3327-0

Abstract:
"To explore the relationship between NF2 promoter gene mutation and the risk of medulloblastomas (MBs). We collected tissues from 16 MB patients and 7 age-matched non-MB controls."
"NF2 mRNA expression was higher in controls than patients; whereas NF2 protein level was higher in patients than in controls."


Conclusions:  
"This is the largest series of revision surgery for vestibular schwannomas to date. Our preferred approach is the translabyrinthine craniotomy, which can be readily modified to include the transcochlear approach for improved access. CSF leak rate slightly exceeds that of primary surgery, and gross total resection is achievable in the vast majority of patients."


"Inactivation of the tumor suppressor Merlin leads to the development of benign nervous system tumors of neurofibromatosis type 2. Merlin deficiency is also observed in human malignancies including colorectal and lung cancers. Causes of Merlin inactivation include deleterious mutations in the encoding neurofibromin 2 gene (NF2) and aberrant Merlin proteasomal degradation. Here, we show that NF2 is also regulated by microRNAs (miRNAs) through interaction with evolutionarily conserved miRNA response elements (MREs) within its 3'-untranslated region (3'UTR)."

27. Multicenter, phase 2 study of bevacizumab in children and adults with neurofibromatosis 2 and progressive vestibular schwannomas: an NF Clinical Trials Consortium study (S23. 004).  
https://www.biorxiv.org/content/early/2018/01/16/249177  

Conclusions:  
"Bevacizumab treatment at 10 mg/kg every 2 weeks is associated with hearing and radiographic response rates at 6 months that are similar to previous studies using lower doses. Future analyses will address the durability of hearing and radiographic responses during 18 months of maintenance therapy."

28. Mifepristone (RU-482)  
Status: Phase 0 - NF2 Pre-Clinical  
Tumor Target: Vestibular Schwannoma (VS)  
Class: Progesterone Receptor Antagonist  
https://nf2is.org/RU482.php  

Massachusetts Eye and Ear researchers have shown that Mifepristone (RU-482), a drug currently FDA-approved for chemical abortion, can also prevent the growth of vestibular schwannoma.  
"Adverse effects for mifepristone include mild fatigue, hot flashes, nausea and rash. Long-term use of mifepristone has been studied in clinical trials for other tumors, with minimal adverse effects reported after years of usage.[5]  
"Side Effects; nausea, vomiting, diarrhea, weakness, or dizziness may occur. Serious side effects; fever of 100.4 degrees F (38 degrees C) or higher, fainting, fast heartbeat, stomach/abdominal pain or tenderness."

Sources  

29. Clinical trials may be based on flimsy animal data

Science. (April, 2018)

In an article from the American Association of the Advancements of Science. (AAAS) “Clinical trials may be based on flimsy animal data.” The trials were not for any specific condition or treatment, but a general concern for all mouse trials.

The discussion on arguments of issues of trials in people do not match the results from animal studies, but in review “89% of the animal studies were not published at all, making it impossible for the IRBs to know whether the study had been reviewed by other experts. Additionally, fewer than 5% included important information on whether bias-reducing methods such as randomization of the experimental groups were used, they report today in PLoS Biology. (This could mean that the studies weren’t set up to avoid bias, or that the measures were not reported in the investigator brochures, for instance to keep the packets concise.)” [AAAS]

“Lastly, 82% of the brochures only reported studies that had positive effects” … [AAAS]

“It’s not clear why investigator brochures often lack data... Some companies may prefer to keep information confidential because they worry about the competition, or they may believe that animal efficacy studies are too complex for most IRB members-which usually include nonscientists to review.” [AAAS]

About the IRB:

“Institutional Review Boards (IRBs) and Protection of Human Subjects in Clinical Trials. Under FDA regulations, an Institutional Review Board is group that has been formally designated to review and monitor biomedical research involving human subjects.” [FDA]

Question

Is it possible this is also happening with Phase 1 trial results in people?

Sources

2. FDA. “Institutional Review Boards (IRBs) and Protection of Human Subjects in Clinical Trials.” https://www.fda.gov/AboutFDA/CentersOffices/OfficeofMedicalProductsandTobacco/CDER/ucm164171.htm

30. Poor Balance (Disequilibrium) and Vestibular Rehabilitation Therapy

NF2 Information and Services. (April 21, 2018)

People with Neurofibromatosis Type 2 (NF2) develop walking issues for many reasons. Most often walking issues starts as a result of imbalance from tumor growth that results in vestibular nerve damage, which is a branch of cranial nerve 8 that runs from the brainstem to the vestibular system for which provides the inner ear for balance.

The growth of a schwannomas tumor on the vestibular nerve, vestibular schwannomas (VS)/acoustic neuroma (AN), will result in an individual facing single-sided vestibular system damage and will be the start of equilibrium issues; disequilibrium or vestibulopathy. Single-sided vestibulopathy is characterized by vestibular system damage on one side of the head. It results in imbalance issues that an individual can recover from in a few days or a few months.

Bilateral vestibulopathy is vestibular system damage to the nerve on both sides of the head. “The symptoms typically include imbalance and visual disturbance. The imbalance is worse in the dark or in situations where footing is uncertain while spinning vertigo is unusual. The visual symptoms referred to as "oscillopsia," only occur when the head is moving (J.C., 1952).” [1] Bilateral vestibulopathy results in complete loss of the part of the body that distinguishes motion. With time, a section of the brain will help with adaption to motion, but vestibular rehabilitation therapy (VRT) is necessary.

Without VRT it is easy to develop body weakness that can make it even more difficult to recover balance and ability to walk naturally.

Learn More:


31. Too much sitting: The Brain and Memory

NF2 Information and Services. (April 16, 2018)

Do you have problems with memory? How many hours a day do you sit without walking, moving, or standing? "researchers have found that in people middle-aged and older, a brain structure that is key to learning and memory is plumpest in those who spend the most time standing up and moving." [LA Times]

"The findings are based on interviews and tests of 35 cognitively healthy people between the ages of 45 and 75. Researchers at ‘UCLA's Semel Institute and its Center for Cognitive Neuroscience’ queried the volunteers about their physical
activity patterns and scanned their brains in an MRI. Then they gauged how self-reported sitting time or physical activity levels corresponded to a thickness in these critical brain structures. [LA Times]

"The study subjects reported average sitting times of three to 15 hours a day. After adjusting for their subjects’ ages, the researchers found that every additional hour of average daily sitting was associated with a 2% decrease in the thickness of the medial temporal lobe." [LA Times]

**Sources:**

**32. Reason to Walk: Heart**

_NF2 Information and Services. (April 10, 2018)_

Something to consider for individuals with high blood pressure, or at risk for heart-related issues, for example, if taking a tumor-drug medication; “walking briskly can lower your risk of high blood pressure, high cholesterol and diabetes as much as running.” [AHA]

“Researchers analyzed 33,060 runners in the National Runners’ Health Study and 15,045 walkers in the National Walkers’ Health Study. They found that the same energy used for moderate-intensity walking and vigorous-intensity running resulted in similar reductions in risk for high blood pressure, high cholesterol, diabetes, and possibly coronary heart disease over the study’s six years.” [AHA]

“Just get started, even if it’s a few additional minutes per day.” [AHA] “It’s not all or nothing; it’s step by step.” [AHA]

“The findings are consistent with the American Heart Association’s recommendations for physical activity in adults that we need 30 minutes of physical activity per day, at least 150 minutes of moderate activity per week or 75 minutes of vigorous activity per week to derive benefits.” [AHA]

“Split up your walks into 10-15 minutes each.” [AHA]

**Source:**
1. American Heart Association (AHA). “Walk, Don’t Run, Your Way to a Healthy Heart.” http://www.heart.org/HEARTORG/HealthyLiving/PhysicalActivity/StartWalking/Walk-Dont-Run-Your-Way-to-a-Healthy-Heart_UCM_452926_Article.jsp#.Wtk7COch1mB
2. NF2 Information and Services. https://www.nf2is.org/move-it-move-it.php#heart

**About NF2 Information and Services**

NF2 Information and Services has been a corporation since September 2013 and a charity under section 501(c) (3), as a Public Charity Status of 509(a) (2) since July 2014. The organization is Dedicated to the education of Neurofibromatosis Type 2 (NF2) health issues and treatments. Charitable funds raised will go to: support and education, print and online Educational Material, medical center education and research.

There is no cure for NF2, but the constant advances in medical development today have only recently started to be truly helpful managing some of the symptoms that patients with NF2 endure. We, as an NF community, need to be aware of how to find NF2 specialists and the newest available treatments and procedures to consult doctors about. We endeavor to thus improve our quality of life.

NF2 Information and Services is dedicated to helping people with NF2 who are looking to learn and find the information and services needed to live better, feel better and do better as they deal with their NF2 issues.

The NF2 Information and Services organization and affiliated website, are not run by medical professionals.

**Information and Services - Goals: NF2 Education**

‘NF2 Information and Services’ is an education charity looking to share information with doctors, researchers, people with NF2 and their families to help ensure better health for individuals with NF2.

NF2 Information and Services is operated by volunteers with NF2 and family members of individuals with NF2.

Research on up to date information for Treatment Pros, Cons, and Options for:

- Disability Prevention - Sharing Information about Options to Remain Independent Longer
- Prevention When Possible
- Recovery of Health Issues
- Improved Quality of Life
NF2 Move It Move It: Walk for Health

NF2 Day - 2018

Goal: Help Yourself if in need of Balance Recovery or Building Muscle Mass

Plan a walk with a sponsor during one day listed, aim for multiple walk days during listed days with other people.

‘NF2 Information and Services’ is encouraging walkathons across the world on NF2 Day. The start of a movement to improve balance, focusing on one step at a time will and increase of muscle strength.

Dates: Sat. May 19, Sun. May 20, Tue. May 22, Sat. May 26, or Sun May 27.

Tuesday, May 22 is NF2 Day. Plan your day for your walk with a supporter.

Location: Your Choice.

A Park Race Track, Tennis Court, School Track, Pre-selected Flat Path in your town, or Mall of Your Choice. Short paths may be ideal with a plan to walk the path as many times as possible.

Costs: Free – Unless you do not own a portable 2Lb folding chair ~ $10

Rules:

- This Event Is Not a Race. Goal is walking; better, precision walking, and proper posture.
- A supporter is required present to walk with you for assistance during your walk.
- Do not walk faster or further than your supporter.
- Added Recommendations and Competitions: See added Challenges at www.NF2IS.org/move-it-move-it.php

Learn More about NF2 and NF Awareness Month:

https://www.nf2is.org/awareness.php

#NF2ISMoveItMoveIt

NF2 Information and Services