EUROPEAN **HUNTINGTON'S DISEASE** NETWORK

Frequently Asked Questions about Huntington's Disease

FOR ALL WHO WANT TO LEARN MORE

Written by Diana Raffelsbauer

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Overview

1. What is Huntington's disease?
Huntington's disease (HD), also known as Huntington's chorea, is a rare degenerative inherited genetic disorder of the brain.

2. Why is it called the Huntington's disease?
HD is named after George Huntington, an American medical doctor who described the disease accurately in 1872. His description was based on observations of HD affected families from the village of East Hampton, Long Island, New York (USA), where Huntington lived and worked as a physician. He was the first person to identify the pattern of inheritance of HD.

3. What causes HD?
HD is caused by a mutation in the gene coding for a certain protein called huntingtin (Htt). This mutation produces an altered form of the Htt protein, which results in the death of nerve cells (neurones) in some areas of the brain.

4. How does the mutation cause death of nerve cells?
The exact mechanism of the disease is still unclear. Two pathways have been proposed: Firstly, the protein can no longer exert its normal function (loss of function). Secondly, the mutant protein may be toxic to the nerve cells (gain of function).

5. What happens to the brain in HD?
Certain functions of the brain, such as the ability to move, think and talk, gradually deteriorate as crucial nerve cells become damaged and die. The part of the brain most affected by HD is the striatum, which is a structure of the basal ganglia located in the central region of the brain and consisting of two main subdivisions called caudate nucleus and putamen. The striatum is primarily responsible for planning and controlling movements, but is also involved in many other cognitive (thinking) processes. Loss of cortex (grey matter in the outermost layers of the brain) occurs during disease progression which contributes to worsening of the cognitive function. In general, HD causes atrophy of the whole brain.

6. When do HD symptoms appear?
Most individuals develop the disease during mid-adult life, i.e. between 35 and 55 years of age. Approximately 10% of people develop symptoms prior

to the age of 20 (juvenile HD) and another 10% after the age of 55. More rarely, symptoms appear before the age of 10 years (infantile HD).

7. How long does HD last?
HD is a fatal illness, developing at a gradual and relentless rate. The average duration of the disease is 15-20 years, but this varies between individuals.

8. Why does HD lead to death?
People with HD do not die as a direct result of the disease, but from medical problems that arise from the body's weakened condition, particularly choking, infections (such as pneumonia) and heart failure.

9. How do I know if I have HD?
If you suspect that you have HD, you should consult an HD specialist (usually a neurologist) for diagnostic testing.

10. How common is HD?
HD is a rare disease which affects up to approximately 1 in 10,000 people in most European countries. In Germany for instance, about 10,000 people have HD and a further 50,000 are considered at risk for inheriting the HD gene because they have (or had) a parent with HD. Men and women are equally likely to inherit the gene and develop the disease.

11. Does HD occur with the same frequency in different countries?
HD can affect people of all ethnic groups, but is more common among European descendants. The prevalence of HD in countries predominantly of European descent (e.g. USA, Canada and Australia) is similar to Europe. In the USA for example, approximately 30,000 people have HD and a further 150,000 are at risk. HD is less common in Asian and African countries, where the frequency has been estimated as 1 per 1,000,000 people. However, detailed studies have not been conducted in these countries, with the exception of Japan, where it has been well-documented that there are fewer HD cases than in Europe.

Symptoms

12. How does HD start?
The first subtle signs may be slight personality or mood changes. Forgetfulness, clumsiness and random, brief, "fidgeting" movements of the fingers or toes might also be a hint. Often, medical advice is not sought during these very early stages of the disease, and some years may pass by before the disorder is medically diagnosed. Hence, the onset of HD is described as "insidious", as the disease emerges very slowly.

13. What are the symptoms of HD?
HD is characterised by a combination of motor (movement), behavioural (mood) and cognitive (thinking) disturbances. The symptoms of HD may vary in range, severity, age at onset and rate of progression from individual to individual, including among members of the same family. For instance, one person affected by HD may have a very obvious movement disorder, but only mild psychiatric symptoms and intellectual deterioration, while another may suffer from depression and anxiety for years before showing any abnormal movements.

One of the earliest **motor symptoms** of HD is **chorea** (involuntary "dance-like" movements). Chorea comes from the Greek word *choreia*, which means dance. At the beginning of the disease, these extra movements occur infrequently and are not especially large. HD patients also have slowed initiation of voluntary movements, which is often very subtle and is best described as **bradykinesia** (a slowing of movements).

As the disease progresses, these symptoms become more evident. In the middle stages, the chorea may cause relatively large movements of the muscles of the limbs, face and trunk. The slowing of movements will have worsened, but might be masked by the chorea. Furthermore, **dystonia** may become apparent. This term describes a condition in which abnormally slow and prolonged muscle contractions cause twisting and repetitive movements. The motor signs of HD are therefore a mixture of chorea, bradykinesia and dystonia, which notably affect posture, balance and gait. In some cases, the person may appear stiff and rigid. Oculomotor (eye movement) abnormalities are also frequent. The patient's **speech** gradually becomes more slurred and difficulties with **swallowing** may also appear which might lead to **weight loss.** Referrals to a speech therapist and a dietician can be helpful.

In addition to the movement disorders listed above, HD causes **personality and behavioural (mood) changes**. The most typical psychiatric symptoms seen in HD are depression, apathy, anxiety, irritability, outbursts of anger, impulsiveness, obsessive-compulsive behaviours, sleep disturbances and social withdrawal. Occasionally, delusions (false beliefs) and hallucinations (seeing, hearing or feeling things that do not really exist) are also observed.

HD is also characterised by increasing **cognitive (thinking) impairments** which involve comprehension, reasoning, judgment and memory. Cognitive symptoms include slower thinking, difficulty concentrating, organising, planning, making decisions and answering questions, as well as problems with short-term memory, impaired ability to absorb and understand new information and solve problems.

There are also a number of other symptoms that often occur during the course of the disease including weight loss, sleep disturbance and urinary incontinence.

14. How does HD progress?

According to a classification developed by Dr. Ira Shoulson, the progression of HD can be divided into five stages:

- Early Stage: The person is diagnosed as having HD and can function fully both at home and work.
- Early Intermediate Stage: The person remains employable but at a lower capacity. He/she is still able to manage daily affairs despite some difficulties.
- Late Intermediate Stage: The person can no longer work and manage household responsibilities. He/she needs considerable help or supervision to handle daily financial affairs. Other daily activities may be slightly difficult but usually only require minor help.
- Early Advanced Stage: The person is no longer independent in daily activities but is still able to live at home supported by the family or professional carers.
- Advanced Stage: The person requires complete support in daily activities and professional nursing care is usually needed.

15. Do the symptoms of juvenile HD differ from those of the adult form?

When HD starts early in life (i.e. under the age of 20), chorea is less prominent whereas slowness of movement (bradykinesia) and stiffness become more prevalent. In most cases, the rate of progression of juvenile HD tends to be faster than in the adult form. Early features of juvenile HD include strong behavioural changes, learning problems, decline at school and speech problems. Epileptic seizures occasionally occur in HD, being more common among young patients.

16. What are the symptoms when HD starts late in life?

When HD starts late in life, chorea tends to be stronger, whereas slowness and stiffness are less prominent. If HD occurs late in life, it is likely to be more difficult to establish a family history because the individual's parents may have already died, perhaps before they themselves showed signs of the disease.

Genetic background

Chromosomes contain genes, which are the basic units of inheritance. A **gene** is a sequence of DNA (deoxyribonucleic acid) which codes for a certain protein. The **DNA** is a polymer (long chain) composed of nucleotides whose structure is a double helix. A **nucleotide** is a chemical compound consisting of a DNA base (adenine, guanine, cytosine or thymine) linked to a sugar molecule (deoxyribose) and a phosphate group. The DNA carries the **genetic information**, which is translated into specific amino acid sequences within the **proteins** using the **"genetic code"**.

Humans are **diploid**. This means that they have two copies (also called alleles) of each gene, one inherited from each parent. A human cell contains 23 pairs of chromosomes. In each pair, one chromosome comes from the father and the other from the mother.

HD is an **inherited genetic (hereditary)** disease which is caused by mutations in one gene (the HD gene) present in all cells of the body since fertilisation. This means that HD can be passed on from one generation to the next.

HD is an **autosomal** genetic disease. This means that it may affect both men and women equally because the abnormal gene is located on a chromosome which is the same in both sexes (autosome or non-sex chromosome).

Most HD affected people are **heterozygous**. This means that they have two different copies of the gene: one normal copy from the unaffected parent and one abnormal copy from the affected parent. In exceptional cases, when both parents are affected, the offspring may inherit two abnormal copies of the gene (one from each parent). In this case, the child is **homozygous** (two identical copies of the gene).

HD is a **dominant** genetic disease. This means that only one abnormal gene copy from either parent is sufficient to inherit the disease. In other words, the mutation in the HD gene dominates over the normal copy from the unaffected parent.

In 1993, scientists identified the gene that causes HD. Located on chromosome 4, this gene codes for a protein called **huntingtin** (Htt). In the first part of the HD gene, there is a sequence of three nucleotides, cytosine-adenine-guanine (CAG), which is repeated several times (i.e. …CAG-CAG-CAG-CAG…). This is called a **"trinucleotide repeat"**. According to the genetic code, the trinucleotide CAG codes for the amino acid glutamine. Hence, a sequence of CAG repeats forms a chain of glutamines or **"polyglutamine"**.

A trinucleotide repeat length of up to 35 CAG units is considered normal. When the HD gene has more than 40 CAG repeats, the altered form of the huntingtin protein produced will cause the disease within a normal life time. Hence, HD is caused by a **trinucleotide repeat expansion** and is one of several **polyglutamine diseases**.

Inheritance

17. How is HD passed on?
An unaffected person always gives normal copies of the gene to the next generation. In contrast, an HD affected person can pass on either a normal or an abnormal copy of the gene with a 50:50 probability (provided that he/she is heterozygous for the HD gene). Hence, when one parent carries the HD gene, the children will inherit a normal gene from the unaffected parent and will have a 50% risk of inheriting the abnormal gene from the affected parent.

18. Can I get HD in any other way?
No, this is impossible. You must be born with the abnormal HD gene in order to develop the disease.

19. If my brothers and sisters have HD, does this mean I will have it too?
Not necessarily. The risk of being affected is 50% for each child of a person who carries the HD gene.

20. If the risk of inheritance is 50%, does this mean that half of all of the children in a family will develop the disease?
The overall risk of inheriting the HD gene is always 50% for each child (provided that only one parent is affected and carries only one abnormal copy of the gene). This does not mean that half of the children in any family will inherit the HD gene. For example, in a family of three children, it is possible for one, two or all three children to inherit the abnormal gene or for all three children to only inherit the normal gene.

21. In our family only women carry the HD gene. Does this mean that only women will ever be affected in our family?
This is a mere coincidence. HD can affect both men and women equally.

22. What does it mean if I am told that I am "at risk" of HD?
It means that either your mother, your father or one of your grandparents carries the HD gene, regardless of whether he/she has already developed the symptoms or not. If one of your parents is affected, your risk of inheriting the HD gene is 50%. If a grandparent is affected and it is not known whether your parent carries the HD gene, then your risk of inheritance is statistically 25%.

23. Am I already affected by the disease if I am a carrier of the HD gene?
By definition a carrier is not affected by the disease, unless he/she begins to show symptoms and signs.

24. What happens if I carry the HD gene?
People who inherit the abnormal HD gene may develop the disease, depending on the number of the CAG repeats, although usually not before

mid-adult life. They may pass the mutant gene on to their children. However, as explained above, their children have 50% chance of inheriting either the abnormal or normal copy of the gene.

25. What is the chance that I will have a child who carries the HD gene?

Every child of a parent who carries the HD gene has a 50% risk of inheriting the abnormal gene. If you carry a risk of 50% yourself and have decided not to undergo genetic testing, statistically your child will have a risk of 25%.

26. Can HD skip a generation?

If a person does not inherit the HD gene, he/she will not develop the disease and will not pass HD on to the next generation. The HD gene cannot skip a generation, but the symptoms can. This situation may occur if the gene carrier dies before the symptoms appear, so that it becomes more difficult to establish a family history.

27. Does the likelihood of developing HD change during lifetime?

Yes, the risk of inheriting the HD gene is 50% at birth. After you pass mid-adult life, the likelihood of developing HD decreases as you get older. If you reach 60 years of age without any symptoms, your risk of developing HD will be lower.

28. My parent became ill in old age. Will that be the same for me?

Some families have an older average age at disease onset than others. The determinants of age at disease onset are complex and currently under investigation. There is an indirect correlation between the trinucleotide repeat length and the age at onset. This means that, in general, the higher the number of CAG repeats, the earlier the onset of symptoms. However, the CAG repeat number is not the only factor that affects age of onset. This relation seems to be influenced by other genes (called genetic modifiers). Environmental factors may also play a role.

29. How important is the number of CAG repeats?

In general, HD symptoms show up when the trinucleotide repeat has more than 40 CAG units. Individuals with an intermediate CAG repeat length (36-39 CAG repeats) may be affected very late in life or may never show any symptoms at all. On the other hand, larger CAG expansions are typically associated with an early disease onset (under 20 years of age) as seen in

juvenile HD cases. There is a wide range of repeat sizes, but patients with disease onset under 10 years (infantile HD) often have more than 80 CAG repeats.

30. Is juvenile HD always inherited from the father?

In 75% of juvenile HD cases the mutation is inherited from the father and in 25% of cases from the mother. When the gene has more than 29 CAG units, the number of repeats may increase as the gene is passed on to the next generation, but this is very rare. When the gene has the number of CAG units that causes disease (36 and more) the repeat is more likely to change size when passed from one generation to the next. When the CAG repeat is inherited from the father, it is more likely to increase than decrease. The successive increase in the number of repeats leads to an earlier onset of symptoms, a phenomenon known as anticipation. As this is more likely to occur if the HD affected parent is the father, most cases of juvenile HD are inherited from the father.

31. If a man carries the HD gene, does this mean that his children will develop juvenile HD?

Juvenile onset is rare. If a man is affected, it does not follow that his children will necessarily have juvenile HD.

32. Can HD strike without a family history of the disease?

Yes, but this is very rare. "De-novo" HD mutation refers to the situation where HD appears in a family without a history of the disease. This means that a new, spontaneous mutation occurred which was not inherited from either parent. In particular cases, when the number of trinucleotide repeats shows borderline values (i.e. 35–39 repeats) in a healthy man, an increase in the number of CAG repeats may occur during the production of sperm cells, leading to affected offspring.

33. What happens if both parents carry the HD gene?

This is an extremely rare situation. If both of your parents carry an abnormal copy of the gene, your overall risk of inheriting the HD gene increases to 75%. You will have 25% risk of being homozygous, i.e. inheriting two abnormal copies of the gene. Homozygous individuals generally do not show an earlier symptom onset, but may have an increased rate of disease progression.

34. Are there other diseases like HD?
Yes, a few HD-like diseases (HDLD) have been described, although the genes responsible for these disorders are different from the one that causes HD. Moreover, the nature of these diseases and their symptoms are slightly different.

Diagnosis

35. How is HD diagnosed?
If you suspect that you have HD, you should consult an HD specialist (usually a neurologist) for diagnostic clinical and genetic testing. If you already show symptoms of HD, your doctor will make a diagnosis on the basis of your medical history and clinical findings. The results of this diagnosis are then checked by genetic tests (confirmatory testing). If you do not show any HD symptoms but are at risk because one of your parents has HD, you might be a pre-symptomatic gene carrier. In this case, the HD diagnosis will rely on genetic testing only.

36. What is a predictive test?
A predictive test is a genetic test to determine whether a person will develop a certain genetic disease. It is by definition performed in a pre-symptomatic stage, i.e. before any signs or symptoms of the disease appear.

37. One of my parents was recently diagnosed with HD. Should I undergo predictive testing?
Deciding whether to be tested for the HD gene in a pre-symptomatic stage is a personal decision. For some people, the uncertainty of whether they carry the mutant gene is very distressing. But for others, the knowledge that they will develop a fatal disease is even worse.

38. What is the procedure for the predictive test?
Living with the knowledge that you are at risk can be very worrying. You may feel that you would prefer to know for certain whether or not you have the abnormal HD gene. At this stage, genetic counselling can be very helpful. Referral to a clinical genetics team ensures that you are provided with accurate and up-to-date information about the disease. It also gives you the opportunity to talk through the choices available to you. Usually you will be

offered an appointment with a consultant to discuss your concerns about HD. If you choose to undergo predictive testing to see whether you may have HD in the future, you will be seen several times by the medical team who will guide you through the process. If you decide to have the genetic test, a small blood sample is taken. Depending on the local service, your consultant will give you your results 2-8 weeks later.

39. Where can I take the test?
Genetic testing is only provided by genetics specialists or genetics clinics. You can ask your general practitioner to arrange an appointment for you.

40. How is the genetic test performed?
The DNA is extracted from blood cells and analysed in a specialised laboratory. Your affected parent's blood may also be tested to check the original diagnosis of HD.

41. What does the genetic test detect?
The genetic test is a DNA test which determines the length of the CAG repeat in the HD gene and thus detects the mutation. The test can tell whether you carry the HD mutation, but it cannot tell you when the disease itself will start to develop.

42. How are the genetic results interpreted?
There are four different types of results: A result under 27 CAG repeats is unequivocally normal. A repeat length between 27-35 repeats is normal, but there is a small risk that the repeat may increase in future generations. Between 36-39 repeats the result is abnormal, but there is a chance that the person may be affected very late in life or even not at all. Over 40 repeats the gene is unequivocally abnormal.

43. How reliable is the genetic test?
HD was one of the first inherited genetic disorders for which an accurate genetic test could be performed. The results from the DNA analysis are usually double checked using two separate blood samples.

44. Are the test results confidential?
Yes, the test results are kept confidential and are only disclosed to another person with your written permission.

45. Will my health insurance pay for predictive testing?
You need to check with your insurance provider if they cover pre-symptomatic testing. However, before doing so, you should weigh the risks and benefits carefully. It might happen that an insurance company deny health coverage or cancel an existing policy when a person is tested HD positive. Despite the existence of laws which prohibit genetic discrimination by insurers in many countries, unfortunately it does exist in practice. Therefore, you may consider covering the costs of predictive testing yourself.

46. Should I tell other people (friends, neighbours, employer and colleagues) that I have HD?
It depends on the stage of the disease you are at and whether your gene status will affect other people. For example, you should tell your spouse or partner that you carry the HD gene. You should also inform your employer when HD starts to affect your job performance. However, you should consider that informing others about your disease may lead to loss of social contacts, and discrimination by employers and insurance companies. Before you decide whether, when and whom you tell about HD, you should discuss this with a specialist in legal issues for HD patients.

47. What is the prognosis if I am diagnosed with HD?
In the long term, the diagnosis of HD is fatal. The average duration of HD from symptom onset until death is 15-20 years. However, this varies greatly among different individuals and can range from 2 to 43 years.

Having children

48. Should I tell my children about HD in our family?
Yes, but you should do it in an age-appropriate manner and in a language that the child can understand. Children need to hear about HD from their parents and not from someone else. Otherwise they might think that the affected parent's behaviour is due to alcoholism or drug use, or that the parent does not love them.

49. When should I talk to my children about HD?
As a rule, it is important to tell children about HD if a person in the family is showing symptoms. This prevents children from drawing wrong conclusions about the person's behaviour.

50. May minor children undergo genetic testing?
In general, a minimum age of 18 is recommended, as it is hoped that at this age a person has the maturity needed to deal with the awareness of carrying the HD gene. However, in exceptional cases, it may be reasonable to perform the genetic test in children, for example if they show signs of juvenile HD or in pregnant women under the age of 18.

51. One of my husband's parents has HD and we are thinking about having children. What should we do?
In this case, you should consider genetic counselling before starting a family. Your husband may undergo genetic testing to see if he carries the HD gene. If he does not carry the mutant gene, your children will not inherit the disease. If he does carry the HD gene, then each of your children will have a 50% risk of inheriting the HD gene.

52. If I carry the HD gene, does this mean that I should not have children?
Deciding whether to have children or not despite the risk of HD is a personal decision which only you and your spouse can make. We recommend that you do it with the guidance of a genetic counsellor. There are currently special genetic procedures available in some countries to minimise the risk. You should also consider that by the time your children grow up there might be a cure for HD.

53. May I test my unborn child?
The genetic techniques currently available allow testing of unborn children, known as prenatal (before birth) diagnosis. However, testing of unborn children needs to fulfil certain medical and legal criteria, which may be country specific.

54. How is prenatal diagnosis performed?
There are two classical procedures of prenatal diagnosis: Amniocentesis (also called amniotic fluid test) is a procedure in which amniotic fluid containing cells of the unborn child is collected with a needle, usually after the 14th week of pregnancy. Collection of sample from the chorionic villi (tissue of the placenta) can be done earlier (between 9 and 12 weeks of pregnancy), but is more risky to the unborn child.

55. Can I test my unborn child without disclosing my own genetic status?
Yes. The "exclusion test" compares the genetic pattern of the unborn child with the genetic pattern of the grandparents.

56. Is it possible to conceive a child who does not carry the HD gene?
Yes, preimplantation genetic diagnosis (PGD), also known as embryo screening, is a modern diagnostic procedure combined with *in vitro* fertilisation (IVF). The embryos are screened prior to implantation. Using this technique, only embryos inheriting normal copies of the gene are implanted into the womb. Hence, PGD gives a couple the chance of conceiving a child that will not be affected by HD, regardless whether the man or the woman carries the HD gene. However, PGD is forbidden in some countries by laws aimed at embryo protection.

Treatments

57. Is there a cure for HD?
Unfortunately, there are currently no medications that are proven to effectively treat the underlying causes of HD. However, basic and clinical research has dramatically increased our knowledge of HD in the last years. Many studies are ongoing to investigate its pathogenesis and to find drugs that can prevent or slow down disease progression (called disease-modifying treatments). Several promising treatment strategies are now in the drug pipeline and may be available for clinical trials in the near future.

58. Are there any treatments for HD?
Although there is no cure for HD at the moment, some treatments do control the symptoms of the disease (symptomatic treatments) and improve quality of life. These are divided into pharmacological (drug) and non-pharmacological (non-drug) treatments. Pharmacological treatments comprise any medicines used to treat the symptoms of HD. Non-pharmacological treatments, such as psychotherapy, physical, respiratory, speech and cognitive therapies, may also improve both physical and psychological symptoms of the disease. For instance, improvements by these therapies have been reported for mood state, motor control, speech, balance, swallowing and gait.

59. What are the most important treatable symptoms of HD?
Chorea, bradykinesia, irritability, apathy, depression, anxiety and sleep disturbances have been reported as the most distressing problems of HD. There are different options for the pharmacological treatment of these symptoms.

60. What medicines are used to treat the symptoms of HD?
Certain antipsychotics (neuroleptics) for chorea and hyperkinesias; antidepressants for depression, apathy and other mood disorders; anxiolytic drugs for anxiety; and hypnotic drugs for sleep disturbances. However, many medicines can cause side effects and some of them may counteract others. In addition, the same medication may have different effects in different individuals. Thus, the ideal balance has to be determined individually by an experienced HD specialist according to the symptoms and treatment outcomes.

61. Is there a special diet for HD?
The benefit of a special diet rich in vitamins, coenzymes and other compounds (e.g. creatine, coenzyme Q10 and ethyl-EPA) for HD is much discussed but not clinically proven. In the later stages of the disease, weight loss can be a problem and high calorie diet may become necessary. Referral to a dietician may be helpful.

HD in daily life

62. What does a positive HD result mean?
A positive HD result may change your life in many different aspects, e.g. deciding whether to have children, planning for the future, rethinking priorities, negotiating appropriate housing, etc. It may also make mortgages, health and life insurances difficult. Therefore, individuals at risk of HD are advised to make decisions on long-term care insurance before definitive diagnosis and development of symptoms.

63. How will HD affect my day-to-day life?
HD will gradually affect your ability to live an independent life. Working, social activities and general daily activities will become increasingly difficult to perform. As the disease progresses, you may become more dependent on help and support from relatives, health and social care professionals.

64. May I drive if I am HD gene carrier?
This can be a very sensitive issue. In some countries you may have to inform the driver licensing authority if you have a medical condition which affects your ability to drive. People who are in the early stages of the disease are sometimes given licenses which can be reviewed on a regular basis.

65. What are the most relevant impairments in daily life?
Most HD patients and their carers perceive behavioural symptoms as more distressing than motor and cognitive impairments. These include depression, apathy, anxiety, irritability and obsessive-compulsive behaviours. In addition, cognitive impairments may change daily life to a great extent. HD affects certain regions of the brain which are normally responsible for planning ahead (executive functions) and concentrating on more than one task at the same time (cognitive flexibility). Consequently, HD patients may become overloaded with tasks or have difficulty dividing attention and adapting to changing situations. Moreover, altered sleep patterns may affect family life, either due to sleeplessness during the night or sleepiness during the day.

66. Are there any strategies how to better cope with HD?
Efficient strategies to cope with HD have to be drawn individually, depending on the affected person, the stage of the disease and the family context. HD develops very gradually, so that in general there is time to adapt to the changes caused by the disease. A better understanding of the behavioural and cognitive impairments may help developing strategies to accommodate these changes and to maintain a warm relationship with people suffering from HD. You can also get important information and valuable advice from both HD specialists and lay organisations in your respective country.

Support for HD affected people

67. How do I get in contact with the EHDN?
Choose your country from the menu http://www.euro-hd.net/html/network/locations and select the respective coordination centre. There you can find contact details (names, address, e-mail, and phone numbers). Alternatively, you may use the contact form provided at http://www.euro-hd.net/html/network/communication/contact.

68. How do I get an appointment with a specialist?
You can make an appointment either by a referral from your general practitioner or by directly contacting a neurologist via your language coordinator (www.euro-hd.net/html/network/project/langcoord).

69. Is there any way I can speak to a specialist without attending a clinic?
Independent advice about HD can be obtained from the lay organisation of your country. Choose your country from the menu http://www.euro-hd.net/html/network/locations and select the respective HD Association. A list of national HD lay organisations is provided at www.euro-hd.net/html/disease/links/hdas.

70. How do I become involved in research projects?
The EHDN hosts the largest European HD study, which is called REGISTRY (www.euro-hd.net/html/registry). REGISTRY is accessible at many study sites across the continent. To find out if there is a site near you, please visit the location (www.euro-hd.net/html/network/locations) section of this website and approach your local public contact. You can also contact your language coordinator (www.euro-hd.net/html/network/project/langcoord) who will be able to update you on research activities within your region. It is also possible for you to contact your local lay organisation (www.euro-hd.net/html/disease/links/hdas) to find out more about participating in research. Click here for more information and links to HD research (http://www.euro-hd.net/html/disease/links).

71. Is there any support group specialised in HD?
There are several lay organisations that provide support for individuals and families affected by HD. These organisations can be reached via your general practitioner or HD specialist. There is a list of HD support associations on the EHDN website. To access them, click the link www.euro-hd.net/html/disease/links/hdas.

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Chairman Prof. G.B. Landwehrmeyer,
Oberer Eselsberg 45/1, 89081 Ulm, Germany,
www.euro-hd.net.

Written by Dr. Diana Raffelsbauer,
PharmaWrite, Giebelstadt, Germany,
www.pharmawrite.de.

Designed by Gabriele Stautner,
Artifox Communication Design, Ulm, Germany,
www.artifox.com.