



## EDITORIAL

As reported inside scientific research into the genetics of ataxia continues to yield impressive results. Several ataxia-genes have already been located, and this has led to the development of predictive tests for some diseases. However the very success of this type of research has itself created a range of ethical problems, concerning the availability of these tests. These issues have yet to be answered. A step in this direction was taken at the last board-meeting of EURO-ATAXIA in Brussels on 12 March. There it was decided to establish a working-party on the ethics of the various applications of new genetic research, such as predictive testing (PT). In our lead article Dr. Eric Legius (Leuven University, Belgium) examines some of the ethical questions raised by PT.

Minor changes to the make-up of the board were also made at the Brussels meeting. Manfred Van den Kerchove replaces Michael Morgan as Vice-President. Michael Morgan continues as editor of the Newsletter, although contributor's attention is drawn to his recent change of address.

It is important that EURO-ATAXIA pays attention not only to scientific issues but also to those of everyday life for ataxic people. In this context we include an article on sexuality and ataxia. A Dutch reader, Mr. Lieuwe Koopmans, sent us part of an impression of a trip he made some time ago with his son Erik, who has FA, to Bangladesh. It sheds a different light upon the every day problems we have in dealing with our disabilities.

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## POTENTIAL PROBLEMS ASSOCIATED WITH PRESYMPTOMATIC DNA TESTS FOR DOMINANT ATAXIA'S

### Presymptomatic testing

When the exact location of a gene on a chromosome is known and especially when the mutation in a gene that causes a disease is known, it is possible to track that gene in a family. In an autosomal dominant disorder an abnormal gene is present on one chromosome of a pair, and this results in a genetic disorder. An example of this is the autosomal dominant spinocerebellar ataxia. It is known that several types of this disorder are present. Spinocerebellar ataxia type I (SCA1) is caused by a mutation (genetic fault) in a gene on one chromosome of the 6th chromosome pair (Orr et al., 1993). Every person with such a mutation on chromosome 6 will eventually develop symptoms of this disease at a certain point in his/her life, and has a risk of transmitting it to the offspring. There is a 50% chance of transmitting the disease to the offspring with every pregnancy. Males as well as females can transmit the disease to their sons and daughters. The disease is often

## CONTENTS

Editorial .....	1
Potential problems associated with presymptomatic DNA tests for dominant ataxias .....	1
Hereditary ataxias: an overview . . . .	2
Sex and ataxia: a young persons guide .....	4
Bangladesh .....	6
Forthcoming conferences .....	7
EURO-ATAXIA: members & contacts . .	8

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present in successive generations in a family. It is possible to look for the mutation in the blood of a healthy relative from a family with SCA1. If the mutation is present, the individual will develop the disease in the future, and he or she will also be able to transmit the disease to his or her offspring (50% risk). Testing of healthy individuals at risk for developing the disease is referred to as presymptomatic testing. Dominant spinocerebellar diseases are progressive disorders, they usually begin in adult life and since no effective therapy is known at the moment, one has to be careful with this presymptomatic testing. In order to perform a test, the person should actively request testing, and should be informed completely regarding every aspect of the disease. Presymptomatic testing of minors should be avoided, as well as testing without permission or under third party pressure (family, spouse). Confidentiality is also a very important issue (insurance, employment). Some people rather don't want to know if they have a harmful gene, and this should be respected. Other people cannot live with uncertainty, and are very concerned of transmitting the disease to their children.

#### **Prenatal testing**

If someone has the SCA1 mutation on chromosome 6, and wants to have children there is always a 50% risk of transmitting the disease. There is a possibility to perform prenatal testing early in pregnancy to find out if the mutation on chromosome 6 has been transmitted. There are two techniques for prenatal diagnosis, the first is chorion villus biopsy (CVS) and the second is amniocentesis. A CVS is performed at 10 to 12 weeks of pregnancy (counting starts at the first day of the last menstrual period). Some obstetricians perform a biopsy through the cervix of the uterus with a catheter, and others use a needle for a transabdominal aspiration under local anaesthesia. Once chorion villi are aspirated the DNA of these cells is examined in the lab for the mutation, and this allows to predict if the unborn child will have the disease or not. Amniocentesis is performed at 15 weeks of pregnancy. Amniotic fluid is aspirated transabdominally, and the few fetal cells present in this amniotic fluid are then cultured, and analysis for the mutation can be performed. If the mutation is present and the couple doesn't want to continue the pregnancy an abortion can be performed for genetic reasons. However this is a difficult decision because in these autosomal dominant disorders there is usually a certain (long or short) period

without symptoms of the disease in the future life of the unborn child and the severity of the disease can be variable.

Presymptomatic testing and/or prenatal testing will also be possible for those dominant ataxias where linked markers can be used. In these situations it is very important that enough affected family members are present and willing to cooperate to perform the testing.

*Dr. Eric Legius*

Naples, September 1993

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### **HEREDITARY ATAXIAS: AN OVERVIEW**

At the Department of Neurology of Federico II University in Naples, patients with early onset (before age 20 years) cerebellar ataxias represent 63% of total and are more common than patients with late onset (after age 20 years) cerebellar ataxias (29%). Friedreich's disease (FD) is the most common diagnosis (44%), followed by autosomal dominant cerebellar ataxias (ADCAs, 24%). I will present some epidemiological and molecular genetic data about these two forms of hereditary ataxias (HAs).

We performed an epidemiological survey on HAs in Molise, a small region in Southern Italy, in 1989. This study identified again FD as the most common form of HA with a prevalence of  $2.1 \times 10^{-5}$  population. The total expected number of FD patients would be about 1200 in Italy. Recent epidemiological studies indicate that the FD prevalence varies from 0.4 in Libya to  $4.7 \times 10^{-5}$  population in Spain. We found no patient with ADCAs in Molise. ADCA prevalence is about  $1 \times 10^{-5}$  population in Libya, Northern Spain, and Northern Italy. The expected number of patients with ADCAs should be about 600 in Italy. Much higher prevalence rates are found in some regions as in Holguin, Cuba, where the occurrence of the disease is more than 100 times higher.

The 'direct genetic' approach of the Quebec Cooperative Study on Friedreich's Ataxia failed because the primary biochemical defect in FD was not identified despite the valuable efforts of many researchers. Since 1980 the technique of the recombinant DNA has been applied to human genetics. The 'reverse genetic' approach made possible to localize the gene of Huntington's disease on the short arm of chromosome 4 in 1983. Using this approach, Chamberlain et al mapped the FD gene on chro-

mosome 9 in 1988. They studied the linkage of the FD gene to two markers of chromosome 9: the gene for interferon and MCT112, an anonymous DNA marker. Chamberlain's findings were confirmed by Fujita et al in a French population; by Pandolfo et al in an Italian population; by Palau et al in a Spanish population; by Chamberlain et al in the French-Canadian and Acadian population, which shows a milder course of the disease; by Pianese and ourselves in Southern Italy. Further markers of the FD region of chromosome 9 have been identified. It has been now clarified that the order of the markers from the distal part to the centromere on the long arm of chromosome 9 is: GS4, MCT112, MS, GS2, 26p, FD1, MLS1; and that the FD gene lies beyond MLS1, closer to the centromere. The Strasbourg group reported a candidate gene of this region (X11), which is expressed in the cerebellum and spinal cord, but not point mutation was found in DNA samples from ataxic patients. A new candidate is now under investigation.

Harding et al described in 1985 a spinocerebellar degeneration associated with a selective defect of vitamin E absorption, which mimicks the FD phenotype. The gene has been now mapped by Ben Hamida et al on the long arm of chromosome 8.

We studied 16 patients from 8 families with FD phenotype and onset after age 20 years ('late onset' FD, LOFD), which usually shows a milder course. We showed that the gene causing LOFD maps on chromosome 9 as 'typical' FD. Klockgether reached the same conclusion studying a German family. Moreover, the coexistence of patients with 'typical' FD and patients with LOFD in the same family suggests that, at least in some cases, the same mutation can cause both 'typical' and 'late onset' FD; and that environmental factors and genetic background may affect onset and course of the disease.

The second most common form of HAs are ADCAs, which are characterized by autosomal dominant inheritance, progressive cerebellar ataxia, possibly associated with pyramidal signs, optic atrophy, ophthalmoplegia, extrapyramidal features, peripheral neuropathy and dementia. There are several large pedigrees reported in literature: the Schut family of Dutch ancestry in Minnesota; the Nino family of Prussian ancestry in Michigan; a large family in Eastern Siberia; a large Cuban kindred; a black family in Texas; three Italian families, probably related, in Calabria, Southern Italy. Between 1972 and 1976, three apparently different families have been described in US: the Machado and Thomas

families in Massachusetts and the Joseph family in California. All of them originated from the Azore islands of Flores and San Miguel and represented different phenotypes of the same disease.

The linkage studies in ADCAs began in 1974, when Yakura et al showed in a small Japanese family that the disease locus (SCA1) was close to the HLA locus on the short arm of chromosome 6. Further studies showed that the linkage studies may be performed, using polymorphic markers of DNA such as D6S89 and D6S109, which are located on the short arm of chromosome 6 closer to SCA1. Resulted to be 6-linked: Schut and Nino families, the families from Texas, Calabria and Siberia; a linkage to chromosome 6 was excluded in Machado-Joseph disease (MJD) and in the Cuban family. Last June, Orr et al communicated that an unstable trinucleotide cytosine-adenine-guanine repeat is present in the SCA1 gene. The number of repeats varies from 25 to 36 in healthy unaffected individuals, whereas the affected members possess about twice the number of repeats. The authors also showed a direct correlation between the number of repeats and the severity of the disease as indicated by the onset age.

Other genes responsible for ADCAs have been mapped. A collaborative Cuban, German and English study led to the localization of the gene responsible for the Cuban ataxia on the long arm of chromosome 12. Takiyama et al mapped the gene for MJD on the long arm of chromosome 14.

Therefore, as far as ADCAs are concerned, it is now possible to distinguish at least 4 different forms: SCA1, which links to chromosome 6; SCA2, which links to chromosome 12; MJD which links to chromosome 14; and other forms, which are neither 6 or 12 or 14 linked. Even though large series of patients are in still under investigation, the current opinion is that the 6-linked families represent about one third of ADCAs; that MJD is present in Japan, Portugal, France and other countries; that SCA2 families appear to be rare outside Cuba.

In conclusion, the molecular genetic studies led to mayor advances in definition and classification of HAs and will lead, hopefully, to a better understanding of their pathogenesis in the next future.

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## SEX AND ATAXIA: A YOUNG PERSON'S GUIDE

The following article is taken from a guide written specifically for young people with ataxia. Given the importance of sexuality in any individual's life, we think it is important for everyone who deals with ataxia in whatever way to be aware of these problems. Therefore we reproduce this article here in the hope it may stimulate researchers to look at these questions in greater detail.

OK, so you're young, attractive to and attracted by boys/girls, have aeons of social graces at your command and are a Really Interesting Person once people get to know you. But you're also ataxic and gradually becoming more and more disabled. Your status as a sexual and social player is, you feel, going to be undermined as a result – even before you've had the chance to assert it, it seems. However, as reality is never as bad as your imagination makes it out to be, it pays to take a closer look at the issues of sex, sexuality and ataxia before despair sets in.

First of all, the biological facts. *Sexual dysfunction* as the medical experts call it is not a major problem to most young people with ataxia. Actually, little work has been done on the physiological and neurological specifics, and much of what is known has been inferred from general neurology rather than analysed in situ. For example, poor erections can be quite a big problem in a number of neurological disorders because of impaired nerve supplies, and it's *assumed* that ataxia does the same. This could well be an important area for further research.

Problems may also be caused by the disabling effects of ataxia rather than the disease itself. The gymnastics involved in sex might become more difficult for ataxic people as time passes – although, of course, the same holds true for many so-called 'normal' people as well. Hence, depending on the sort of sexual activity in mind, the possession of an ataxic body usually doesn't cause problems this soon – even when the individual is already a wheelchair user.

Problems may be gender-specific too. Menstruation can be a difficult time for girls with ataxia, who may experience problems in inserting sanitary towels, for example. Young ataxic women can and do get pregnant as easily as anybody else, it may be assumed. In this having ataxia makes no difference at all. Handling a pregnancy is arduous for any

woman but with ataxia added may cause extra problems for some.

For young people with ataxia, as for young people generally, the immediate issue is not sex itself but rather sexuality – a point frequently overlooked. Sex may simply refer to the mechanics of the thing – a matter of physiological and neurological functioning. Sexuality, however, refers instead to interpersonal communication – a different reality almost. It encompasses sexual interaction, the making and sustaining of sexual relationships within a shared social world.

Puberty is generally regarded as the beginning of adult sexual life as physiological and hormonal changes within the bodies of young males and females transform them into fully functional sexual beings. The ongoing development of sexual characteristics, both primary and secondary, in turn causes the intense pre-occupation with physical appearance that is such a marked feature of the adolescent years. For those who deviate from the 'norm' this can be a trying time. Ataxia, moreover, is so ungainly and awkward a condition, *especially* in the early pre-wheelchair years when staggering becomes more and more pronounced as the condition worsens, that there is nothing at all romantic about it. Kate Bush will never sing a song about an ataxic heroine.

The key issue now is how potentially negative experiences like these may rebound on the emerging adult self. Isolation and a lowering of confidence levels are the twin dangers here.

Isolation may originate physically too but it refers primarily to an inner psychological state. Unchecked, it may lead to psychological withdrawal, to an inability or even refusal to negotiate with external reality, and this may be highly damaging for the self.

Sexual interaction is best thought of as a game, albeit a serious one. It is a 'game' whose rules and governing principles are laid down during adolescence – the 'dangerous bend' between childhood and adulthood when the experience of puberty transforms every aspect of individual existence and identity. Adolescence is universally seen as a rite of passage into adult existence, troublesome, arduous even, but above all natural – a necessary grounding in the realities of life faced by every individual. It is where the rules governing male/female interaction – or erotic grammar – are first laid down. Not to engage at this crucial stage – for whatever reason – is to risk losing out on a vitally

important learning process.

All-important too to effective sexual interaction are the maintenance of confidence levels. The self is nourished on self-esteem or self-value – the belief in oneself that underlies how the individual asserts his/her presence and projects his/her personality. Without this basic belief in oneself it becomes difficult to even approach someone, let alone try to establish a sexual relationship with them.

A unique feature of ataxia is that, often, the shift from childhood to adulthood is paralleled by a transition from 'normal' to 'disabled' status. At this, still relatively early stage, the young person is caught between definitions. He/she exists within the 'normal' world of interaction with his/her peers. But, as the ataxia progresses, he/she will find it increasingly harder to maintain a 'normal' status, being pulled inexorably towards the 'disabled' category. This will also reinforce tendencies towards isolation and withdrawal. On a purely physical level the teenager with ataxia may find it harder to engage in normal adolescent activities: going to rock concerts, friend's houses, etc. This in turn may lead to increasing distancing from much of the daily social routine common to both sexes: going to pubs, clubs and dances. Opportunities for male-female interaction and potential sexual contact become less.

As the ataxia progresses marked differences in lifestyle, in common with other people with disabilities, will emerge. These will work to increase barriers to sexual interaction. Accommodation is a major problem for people with disabilities. Those who remain in their parental home often do so without privacy, without control, sometimes under conditions of asexuality. Residential homes are usually worse.

Lack of transport is another problem. Many young people with ataxia will remain unable to drive and must rely on lifts and taxis. Also there is the well-known problem of access: to pubs, restaurants, public buildings, theatres, etc.

Work is a very important area for sexual interaction – as well as being an important source of self-esteem. For many young people with disabilities, 'employment' can often mean home-based work, say on a computer keyboard, which is usually something a lot less than full immersion in the social and interpersonal environment of the workplace that makes 'work' such an important social activity in the first place.

As well, the sort of social contact that people with disabilities do participate in is not normally con-

ducive to sexual interaction. Day-centres are notorious for providing no sex education of any kind, and ignoring any mention of sexuality at all amongst their 'clients'. The sort of charity evening organised by Ataxia Groups at local or national level doesn't offer much scope for erotic negotiations either (or, if one does, I've yet to hear... ).

One exception, oddly enough, remains that of group holidays, particularly trips abroad. Perhaps it's the exotic location, or an admixture of sun, sea and sand, but many relationships have been formed on holiday and have proved lasting on return to colder climes. On this score I can personally recommend FAGs 'Holidays In The Sun' programme, organised by their EURO-ATAXIA representative, Peter Cordwell.

Sometime in late adolescence or early adulthood the young person with ataxia will face the most intense crisis of his/her life: the decision to use a wheelchair. Although taken exclusively for physical reasons, becoming a wheelchair user has important consequences for emotional and psychological well-being. Using a wheelchair means accepting a self-definition as 'disabled', and it's how the self incorporates this (unwelcome) personal identity that often leads to problems – acceptance is never easy.

Sexuality, moreover, is a form of interpersonal communication. A troubled psyche is hardly the best basis on which to engage in effective sexual interaction. Getting blind drunk at parties may help relieve inner *angst*, but is not a recommended method for making sexual contact.

To conclude, problems in sexuality faced by young people with ataxia are not different in kind from those faced by any young person. Adolescence, it can confidently be predicted, will continue to be a rough time for all teenagers whatever their physical status. Having ataxia *as well* will, however, magnify and give a much sharper edge to the 'normal' problems it must be admitted. Being disabled in an able-bodied world is never going to be a smooth ride. But the real dangers are internal – a collapse in confidence leading to psychological withdrawal and loneliness. Isolation is always negative and is opposed to any conception of healthy sexuality. Positive sexuality is about interaction, communication and personal engagement. It's about saying *yes!*

*Michael Morgan*

## BANGLADESH

The offer was too tempting. From friends Erik received an invitation to visit them in Dhaka during their stay in Bangladesh. This country has one thing in common with The Netherlands: it too is a crowded delta. Bangladesh is the part of former British India where the Ganges and the Brahmaputra divide into several separate streams, which subsequently flow into the Bay of Bengal. It is situated in the tropics, is hot and very humid, and is one of the poorest and disaster-ridden countries in the world.

When friends heard about the trip they said to Erik: "You can't be serious, to Bangladesh, you in your wheelchair?" His answer was always the same: "Why not? My father is joining me, he has prepared as much as he can and everywhere you'll find a helping hand; for the rest we'll cross the bridge when we come to it."

Although Bangladesh is a country with very wide rivers, it has no bridges to speak of and no dykes whatsoever. Written-off ferries from the western world – some say 'held together by paperclips' – are crossing rivers as wide as the widest ones in Holland. During a trip in a borrowed car we had to cross one of these rivers: the Meghna. There we found the same bustle as anywhere in the world at landing-places of ferries. Waiting trucks, busses, cars, one man on a bicycle and many foot-passengers. In our country there is usually a cafeteria or another place to have a seat and to eat or drink something: at this landing place people were peddling their food-stuff. One ferry had just left, another was on his way back. It gave us the opportunity to leave the car and give Erik a different seat.

We left the car and were immediately surrounded by countless peddlers, shoe-shiners and beggars. The peddlers were selling bananas, pieces of coconut, and cigarettes by piece. They were hoping to earn something from us, the white millionaires. (In their eyes every white man is a millionaire, which in fact we are: the money someone on social security in Holland receives per month is enough for a Bengali family to live for a whole year – by their standards even a rich life). We asked a peddler the price of a piece of coconut. The only word we understood was: "... poisha." (A *poisha* is a hundredth of a *taka*, which is the Bengali currency). How much poisha he wanted, we did not understand. When we asked it for the second time, another

Bengali quickly said something to the peddler. What it was we couldn't hear, we only understood "taka." The peddler put up two fingers and said: "Two taka." A hundredfold of what he was asking the first time. Two *taka* is in Dutch money only 20 cents: without arguing we paid him what he asked. The piece of coconut was a bit dry, but so what.

More and more people were surrounding us, we were almost squeezed. Some of them were travellers, most of them beggars. One traveller started questioning us in English. He was asking where we came from and where we were going. After answering his questions, we asked him one: "Why are people so amazed at the sight of Erik and his wheelchair?" (We thought it had something to do with the ingenuity of the wheelchair's construction: that kind of contraption you'll never see in Bangladesh). His answer was that he was amazed to see Erik sitting in a wheelchair. We explained as much of the reasons as we could. Again he had a question, he hesitated a bit before he asked: "Are your doctors not able to cure him?" We had to say that that was impossible. Dismayed he shook his head, our answer disappointed him. It was a blow to his image of the Western world. He always thought that white people can do everything, but also white people sometimes have to sit in a wheelchair.

We might have been generous to a couple of beggars or have given some of them a 'rich' day, but Erik's friends had argued with forceful arguments not to do so. If you give something to someone, you will be overrun by beggars. As an example they told the story of a member of the staff of an embassy whose car was in reparation for a week. To get to his work he went by riksha, a bicycle-taxi. At the time he had to pay the fare he gave the man instead of the usual three *taka* a note of 20 *taka*. He repeated his gesture the day after. The third day at 4 o'clock in the morning the whole street was so crowded with riksha's that nothing, no car or bicycle, could pass through: all of the riksha-drivers were hoping to be the one who earned a week's wages for just one ride.

Nevertheless we found it hard to follow the advice of Erik's friends. It was difficult to ignore the poor souls, because we were shocked by what we saw: maimed people who were limping with a crooked leg because the leg was not been treated when it was broken, people with half-rotten faces as a result of leprosy, blind people led by children (a young child was holding a stick at one end while the blind man was holding the other end; the child

accompanied him until they had begged their daily meal together). A man walked around of which we first thought he was also a white man; he had fair hair and a white skin. He proved to be an albino, someone who has no pigment and absolutely cannot stand the sun. He held his hand above his eyes to avoid the sunlight, the little we could see of his skin was inflamed. In that climate he surely will die of skin-cancer. A part of the peddlers and beggars went with the ferry, of course as unpaying passengers. With some regret we did not hand out any money (there were still too many beggars), but in the car we had some packets of cigarettes. Each beggar we gave a cigarette, until all the cigarettes were gone.

In the meantime the ferry had reached the other side of the wide river. There we met the same human misery. The peddlers, the misshapen and the beggars live off the ferry like scavenging seagulls live off a heap of garbage. These poor souls are human seagulls. We both – the one able-bodied, the other with his FA – were happy to be in the privileged position of being born and living in the western world.

*Lieuwe Koopmans*

### IMPORTANT ADDRESS CHANGE!

**EURO-ATAXIA Editor, Michael Morgan, has recently moved to the address below. Would all contributors please take a note of it and pass it on to whoever else might be interested in submitting material to EURO-ATAXIA now or in the future.**

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### FORTHCOMING CONFERENCES

**Ataxia researchers and others may be interested in a number of conferences and meetings occurring in the first half of 1994.**

**The 3rd International Workshop on Machado-Joseph Disease** will take place on the island of São Miguel, Azores, Portugal between 7-9 April 1994. The Workshop will be led by a number of international experts on MJD and other hereditary ataxias. For details, contact:

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June sees two major conferences on hereditary ataxia. **The Ataxia meeting** will be held in Tunis between 20-21 and the **European Society of Neurology** meeting will be held in Barcelona between 25-29.

In the UK the **Genetic Interest Group** continues to stimulate discussion of Human Genetics at all levels. On Saturday 23 April an open meeting on the teaching of Genetics in secondary schools will be held in London, entitled *Human Genetics: Science and Ethics in the Classroom*. (Contact Teryll Helm on +44 71 618521 if interested). The next in the excellent series of GIG **Interface** meetings will be held on 21 June, again in London. The subject will be *Late Onset Genetic Conditions* and the GIG AGM will also be held after the meeting. For more details, bookings etc., GIG can be reached on +44 865 744002.

**CLOSING DATE FOR THE  
NEXT ISSUE**

**1 SEPTEMBER 1994**

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