Netherlands Brain Bank

Progress Report

2011 - 2012

Editors

Inge Huitinga
Bonnie van Huik
Michiel Kooreman
Marleen C. Rademaker
Wilma T.P. Verweij

Correspondence

Netherlands Brain Bank
Meibergdreef 47
1105 BA Amsterdam
The Netherlands

T (+31) 20 566 5499
F (+31) 20 691 8466
secretariaatnhb@nin.knaw.nl
www.brainbank.nl
www.hersenbank.nl
www.nin.knaw.nl

Contents

Introduction

It is with great pleasure that I present the 2011-2012 progress report of the Netherlands Brain Bank, two event-filled years for the NBB.

In 2011 we celebrated our 25th anniversary with a symposium on "Depression and Happiness". The well-attended event, an evening filled with a variety of lectures, a Q and A session, and interspersed with musical intervals, was concluded with a stand-up routine by Freek de Jonge.

In April 2012, Dr. Wouter Kamphorst was made Officer in the Royal Dutch "Order of Orange-Nassau", presented by the Mayor of Amsterdam. The honour was awarded to Dr. Kamphorst in recognition of more than 25 years of dedicated neuropathological work for the NBB.

June 2012 saw the start of NBB-Psy, our new national program for psychiatric diseases. The Netherlands Organisation for Scientific Research (NWO) awarded a grant of € 3,450,000 from the NWO Large Investment Subsidy program to the NBB-Psy project, an initiative of the NBB and five Dutch university medical centers: UMC Utrecht, Radboudumc (Nijmegen), Academic Medical Center (Amsterdam), VU University Medical Center (Amsterdam) and Erasmus MC (Rotterdam). This grant will be used to actively approach 70,000 psychiatric patients and their family members through patient cohorts and patient societies, in addition to a more general national call for psychiatric brain donor registrations. The program is anticipated to result in autopsies of more than 500 donors with a psychiatric disease over the next 10 years and is expected to force breakthroughs in worldwide post mortem psychiatric research.

The start of NBB-Psy perfectly fits the NBB's new strategy to shift its focus from general neurological donor programs to focused recruitment of clinically well-characterized neurological and psychiatric patients. For example, patients with Alzheimer's disease are currently only registered as brain donors if they were diagnosed in the VUmc Alzheimer Center. As a consequence of this shift in focus, the NBB now only welcomes registrations from a limited selection of diseases, which are listed on the

NBB website. This new donor recruitment policy is flexible and will be adjusted as and when necessary.

I am greatly indebted to the Netherlands Institute for Neuroscience, the Royal Netherlands Academy for Arts and Sciences, Stichting MS Research, Internationaal Parkinson Fonds, Internationale Stichting Alzheimer Onderzoek and Hersenstichting Nederland, as well as to private backers, for their financial support, which is indispensible for the continuation of the NBB.

I also thank the members of the autopsy team for their guidance and help with the autopsies, day or night. Many of them are PhD students and technicians who have volunteered to help us out despite their own busy programs and work commitments. Also indispensible are the autopsy assistants and pathologists at VUmc, to whom I would like to express my gratitude for their unstinting willingness to perform the autopsies.

Last but not least, a heartfelt thank you to all our donors and their families, without whom worldwide scientific research of the brain and brain disease would not be possible.

Inge Huitinga
Director Netherlands Brain Bank

Objectives NBB

The Netherlands Brain Bank was founded in 1985 by Dick Swaab, initially with the sole purpose of obtaining brain tissue for his Alzheimer research. However, he soon realized that a facility where people could register as brain donors for research purposes would greatly benefit other researchers in neuroscience as well. From the very start, the NBB has thus been accepting applications for brain tissue from researchers from all over the world.

It is still the primary objective of the NBB to collect, characterize and disseminate tissue of the human brain and spinal cord for the benefit of scientific research worldwide, with the ultimate goal of increasing the knowledge of the human brain and finding cures for neurological and psychiatric brain diseases.

The NBB organization chart can be found in the Appendix (Figure 15).

Donor Program

The NBB is one of the few brain banks in the world with an active donor program, which means that the NBB actively tries to motivate people with neurological and psychiatric disorders, as well as healthy individuals, to register as brain donors at the NBB. With this registration, donors give informed consent to the NBB to perform a rapid autopsy after death and to donate their brain tissue to reviewed research projects around the world. The donors also give permission to the NBB for the release of their medical information after they have passed away. The registration forms and accompanying informational brochures (informed consent forms) are in line with regulations and guidelines issued by international key organizations, such as the Council of Europe, the European Commission, the World Medical Association and the World Health Organization[1]. The informational brochures and registration forms, reviewed by the Medical Ethics Committee of the VUmc, were approved on October 30, 2009.

On December 31, 2012, 2702 living donors with a variety of disorders were registered at the NBB.

25th anniversary of the Netherlands Brain Bank

The Netherlands Brain Bank has been performing autopsies and disseminating tissue to researchers for over 25 years now, ever since its inception in 1985. The NBB's 25th anniversary in 2011 attracted a considerable amount of media attention, culminating in a symposium on "Depression and Happiness" on 15 September 2011 (see figure 1). The public activities and increased media coverage are reflected in an increased number of donor registrations, especially in 2011.

Increased focus of the NBB donor program

In 2011 and 2012 the NBB adapted its registration policy to the demand of the research community by actively encouraging new registrations of control donors and donors with diseases for which brain tissue demand is high. For diseases for which the demand for brain tissue is lower and/or for which the NBB's current stock is sufficient, we have issued a (temporary) halt to registrations. In addition to

1 Samarasekera, N., R. Al-Shahi Salman, et al. Brain Banking for Neurological Disorders. Lancet Neurology (in press).

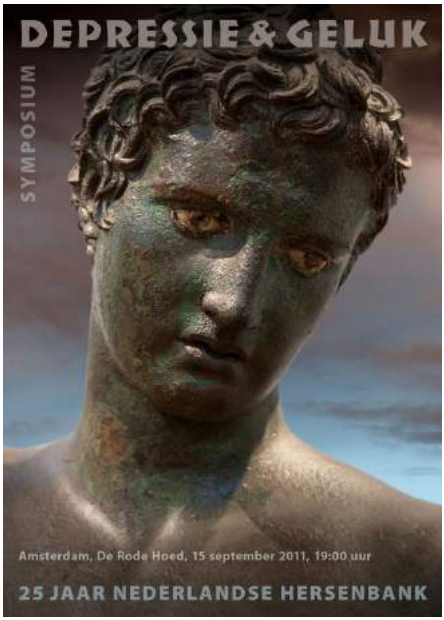


Figure 1 Announcement for the "Depression and Happiness" symposium

control donor registrations, we currently welcome registrations from donors with the following diagnoses: multiple sclerosis, Parkinson's disease, frontotemporal dementia, Alzheimer's disease (only for patients from the VUmc Alzheimer Center), narcolepsy, transsexualism, and psychiatric disorders (especially schizophrenia, depression, bipolar disorder, autism spectrum disorder, obsessive-compulsive disorder, attention deficit hyperactivity disorder, and post-traumatic stress disorder). This list will be adjusted when necessary. Applications of donors with rare diseases are assessed on an individual basis, because it is not feasible to provide an exhaustive list of diagnoses.

Another continuing effort is to increase the quality rather than the quantity of donor registrations through increased recruitment of donors from clinical cohorts, already reported in the progress report for 2009-2010. Many academic hospitals have clinical cohorts of patients with a specific neurological or psychiatric disorder to study disease course and the effect of experimental therapies. These patients are studied longitudinally and therefore many medical data are available in a standardized manner. This makes them a very interesting group for post mortem research. Moreover, it is our experience that people who participated in research during their lifetime also tend to be willing to donate tissue after their demise. In 2012, the NBB

continued with this approach by starting a collaboration with the Alzheimer Center of the VUmc (Prof. P. Scheltens, Dr. W. van der Flier and Dr. Y. Pijnenburg). Annually, the Alzheimer Center extensively diagnoses 600 new patients from all over the Netherlands. Because of the relatively large existing collection of Alzheimer brain tissue at the NBB, we currently do not accept registrations of donors with Alzheimer's disease who are not registered at the VUmc Alzheimer Center. On December 31, 2012, 7 registered donors were part of the Alzheimer Center cohort, 19 donors were part of CARPA cohort (Parkinson's disease) of the Academic Medical Center Amsterdam, and 28 donors were part of the SCOPA cohort (Parkinson's disease) of the Leiden University Medical Center.

Lastly, we intend to improve the accuracy of the NBB's information about living donors, an ambition inspired by the fact that a clinical diagnosis at the time of registration often differs from the diagnosis at the time of a donor's demise, for example when a control donor develops dementia. In many cases, the NBB does not receive timely notification. We aim to address this by regularly sending questionnaires to registered donors (every five years for control donors and every year for donors with a brain disorder), starting in 2013.

Newsletter

In order to keep our donors up to date about the progress made within the NBB and about the scientific output achieved with material provided by the NBB, we started a newsletter for all our registered donors in 2009. The second edition was issued in March 2012 and featured, among other things, the NBB's 25th anniversary celebrations and an overview of the anniversary symposium, the adjustments made to the donor registration policy, and interviews with a donor and a researcher.

Registrations

Figure 2 shows the number of registrations in 2011 and 2012, compared to the registrations in the period 2009-2010. The total number of registrations in 2011-2012 increased considerably in comparison to 2009-2010 (742 vs. 545). This rise can be attributed almost completely to the increase of control donors (almost twofold) and donors with psychiatric diseases, especially depression and bipolar disorder (more than threefold) and clearly illustrates the efficacy of the new donor program strategies of the NBB.

As figure 4 shows, the number of 288 new donor registrations in 2012 is comparable to the numbers of previous years, whereas the number of new donor registrations in 2011 soared to 454, probably as a result of the ample amount of media attention

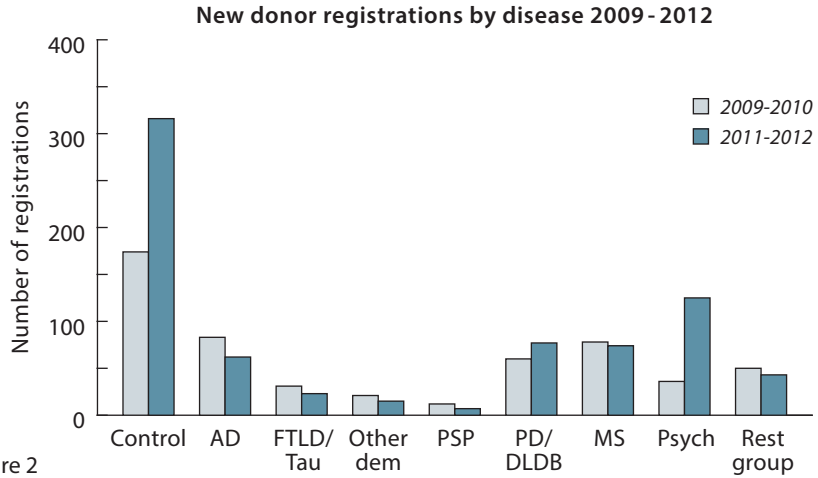


Figure 2

related to the NBB's 25th anniversary. Also, generally, media coverage of the NBB has remained relatively high over 2012. In June 2012, the NBB was awarded a large grant from NWO (The Netherlands Organisation for Scientific Research) for the development of a psychiatric brain tissue donor program. In the public media, several articles appeared about our program which emphasized the importance of control donors as well as our main focus: psychiatric diseases. Table 1 specifies the diagnoses of donors with psychiatric diseases who registered at the NBB in 2011 and 2012.

Table 1 New registrations of donors with a psychiatric disease in 2011-2012

Diagnosis	New registrations
Attention deficit hyperactivity disorder	7
Autism spectrum disorders	3
Bipolar disorder	23
Depression	76
Obsessive-compulsive disorder	2
Post-traumatic stress disorder	6
Schizophrenia	8
Total	*125*

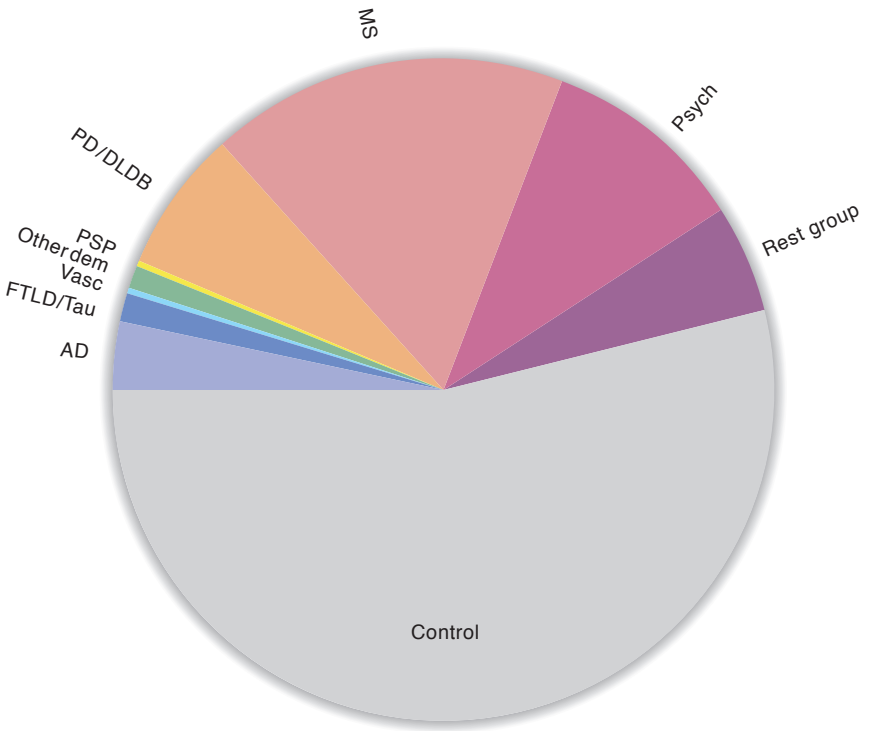


Figure 3 Breakdown of all registered donors by disease (total = 2702 on December 31, 2012)

Figure 3 shows how the 2702 donors registered with the NBB on December 31, 2012, are distributed across the different diagnoses. A large number of control donors is necessary, because a considerable number of these donors turn out to have a different diagnosis at the time of death.

In total, the NBB received 454 new registrations in 2011 and 288 new registrations in 2012 (figure 4). After the increase in annual registrations from 2006 onward, the number of registrations per year has stabilized since 2008, with a peak in 2011, likely due to the press activities in 2011 on the occasion of our 25th anniversary. The number of new female registrations remains higher than that of new male registrations. This is mostly due to the relatively higher number of female MS and non-demented control registrations. The two-fold higher prevalence of MS in females probably explains this for MS. We are as yet unable to offer an explanation for the disproportionate increase of female control donors. Such a difference is not seen in organ donation for transplantation purposes (source: **www.donorvoorlichting.nl**).

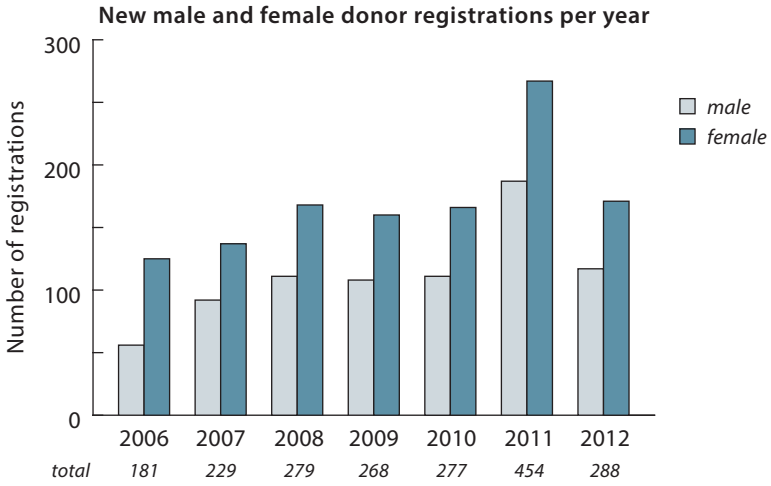


Figure 4

The increase and subsequent stabilization of the total numbers of annual registrations is reflected in the annual numbers of autopsies, which have also increased from 82 in 2006 to stable numbers of 120 to 130 in 2010-2012.

Presentations and articles

In the past two years the NBB has continued to invest time and effort into raising awareness of the importance of research with human brain tissue and the possibility of brain donation. In addition to the media coverage described above, the NBB has visited patient meetings to give presentations on the work of the NBB and on the possibility to become a donor. Being able to show what kind of research is performed on tissue donated to the NBB - research that might help find a cure - evokes many positive reactions and has led to many new donor registrations. Table 2 gives an overview of the articles published about the work of the NBB in 2011 and 2012. We always make sure to mention that patients with neurological or psychiatric diseases and healthy control donors are equally crucial for good scientific research, hoping to persuade family members to register as brain donors as well (see chapter Autopsies).

Websites of the NBB

The internet has become a very popular source for patients trying to learn more about their illness. By making sure that the NBB is mentioned on the websites of the various patient organizations, we try to boost public awareness of the importance of brain donation. We update our donor website (**www.hersenbank.nl**) regularly as

Table 2 Public relations activities and articles on the NBB in 2011 and 2012

Date	Title / description	Medium / additional information
2011/01/03	Interview with Inge Huitinga: 'Brain donor shortage'	Alzheimer Actueel (magazine patient organization)
2011/01/05	Interview with Inge Huitinga: 'Fellowship has been the base of my career'	Rondom MS (magazine patient organization)
2011/07/23	Interview with Dick Swaab: 'Living once is enough'	De Telegraaf (newspaper)
2011/07/31	Extensive interview with Dick Swaab including self-selected television fragments illustrating his life	Zomergasten (TV show)
Aug 2011	Articles about the 25th anniversary of the NBB, announcements and reviews of the 2011/09/15 symposium	*(websites of) newspapers:* De Telegraaf, Spits, De Gooi- en Eemlander, Noordhollands Dagblad, Trouw, De Gelderlander, Het Parool, de Volkskrant. *Other websites:* NOS News, Medisch Contact artsennet, Dutch Button Works, MSweb. Magazines: Medisch Contact, magazine of KNAW
2011/09/15	Symposium "Depressie & Geluk" (depression and happiness)	On the occasion of the 25th anniversary of the NBB
2011/02/09	Presentation: The role of Vitamin D in Multiple Sclerosis	MS organization Gouda
2011/11/24-25	MS research days	Event of patient organization
Nov 2011 and Oct-Nov 2012	Radio commercial with Inge Huitinga	MS Research (patient organization)
2011/12/01	'Meaningful numbers behind 30 years of MS research'	Brochure MS research Rondom MS (magazine patient organization)
2011/12/01	'Huge shortage of brain tissue for Parkinson research'	Papaver (Magazine of Parkinson patient organization)
2012/01/02	NBB featured in TV show	Labyrint (TV show), episode "Kopzorgen" (head worries).
June-Oct 2012	Press release and articles announcing the grant for developing a psychiatric tissue program (NBB-Psy)	NWO, KNAW, De Telegraaf, nu.nl, nd.nl (news website Nederlands Dagblad), scienceguide.nl, magazine of Rudolf Magnus Institute of Neuroscience
2012/04/10	Prize nomination for using alternatives for laboratory animals	Lef in 't Lab ('courage in the lab') award ceremony; Dutch Society for the Protection of Animals and NKCA
Oct 2012	'Biobanks, defrosting lurks'	Medicines (magazine)
Oct 2012	'NBB expands collection'	AMC Magazine
2012/12/01	'The NBB already has 3500 brains'	Experiment NL, issued by Quest and NOW (magazine)

well, so that our (potential) donors are kept informed about the work of the NBB. The English website of the NBB (**www.brainbank.nl**) provides researchers with detailed information regarding our procedures, diagnostics and the availability of tissue. In 2012, the NBB set up e-NBB (**www.e-nbb.org**), an online tissue database where researchers may view the available tissue and make their own preliminary tissue selection. More information about e-NBB can be found in the chapter on Tissue Supply.

New logo

In 2011 and 2012 the NBB has been making continuous efforts to further professionalize its communications. The Netherlands Institute for Neuroscience, of which the NBB is a department, initiated the development of a new logo and changed its Dutch name from "Nederlands Instituut voor Neurowetenschappen" to "Nederlands Herseninstituut", a name which reflects its activities more clearly. A new logo was developed for the NBB in the same style as the institute's logo. The NBB's latest initiative, NBB-Psy, a separate donor program geared towards specific psychiatric disorders, uses two distinct logos, one for communications aimed at donors (red / purple) and one for researchers (blue / green).

At the end of 2012, the NBB began using this new house style in all communication materials: the informed consent packages were changed to match the new look, and new websites and a general informational brochure for donor recruitment were created. The transition was completed in 2013. Figure 5 clearly shows the matching new logos.

The NBB wishes to acknowledge and thank all donors and their families for their generosity and the invaluable gift they are giving to future generations.

Figure 5 New logos of the Netherlands Institute for Neuroscience and the Netherlands Brain Bank

NBB-Psy The Netherlands Brain Bank for Psychiatry

Aim

NBB-Psy is a program of the NBB. Its aim is to establish a resource of human brain tissue of 7 major psychiatric disorders:

- Major depression disorder
- Schizophrenia
- Bipolar disorder
- Obsessive-compulsive disorder
- Post-traumatic stress disorder
- Autism spectrum disorder
- Attention-deficit hyperactivity disorder

Background

The personal, social and economic burden of psychiatric disorders is high and demands better treatment strategies. In order to be able to develop such strategies, an understanding of the underlying etiology and pathophysiology of psychiatric disorders is essential. The use of human brain tissue provides the most direct strategy for developing and testing hypotheses about the molecular and cellular basis of psychiatric disorders. The current availability of human brain tissue from patients with psychiatric disorders is nowhere near sufficient and it is therefore our mission to develop a qualitatively unique tissue program to extend the number of post-mortem brains of extensively phenotyped patients: NBB-Psy (NHB-Psy in Dutch). The resource will be made available to the national and international research community via the application procedures of the NBB. The NBB-Psy program is mainly funded by the Netherlands Organisation for Scientific Research (NWO).

Approach

Together with research groups from 5 Dutch universities (Utrecht, Nijmegen, University of Amsterdam, Vrije Universiteit Amsterdam, Rotterdam), the NBB has developed a strategy based on two strong assets in the Netherlands:

1. The NBB is one of the world's leading brain banks and is well-known for its unique rapid fresh dissection protocols (4-10h after death);
2. The availability of several large and extensively phenotyped cohorts of psychiatric patients.

We will appeal to patients and family members of these cohorts to register as brain

donors at the NBB. Table 3 shows the clinical cohorts that are involved in the program and their number of participants.

To further increase the number of registered donors, we will approach patient and family associations and (long-stay) clinics. We will give oral presentations at patient meetings organized by these associations and at these clinics, and we will hand out brochures to those who are interested in brain donation. Furthermore, we will publish articles on NBB-Psy in the magazines of patient and family associations (Figure 6). Controls will be obtained from the cohorts, from patient organizations and from the regular NBB donor program.

In order to optimize our donor recruitment strategy, we will actively monitor the number of prospective donors approached and the registration rate. Every year we will evaluate what strategy works best for what disorder and determine whether we need to adjust our recruitment strategy.

Table 3 Participating clinical cohorts, (long-stay) clinics and patient and family associations

Disorders	Clinical cohorts		Associations	(Long-stay) clinics
	Name cohort	Nr of participants		
Schizophrenia	GROUP	1057	Ypsilon	GGZ Centraal
			Anoiksis	GGZ Venray
Bipolar disorder	BiG	2500	Vereniging voor Manisch Depressieven en Betrokkenen (VMDB)	
Major depression disorder	NESDA	2056	Depressie Vereniging	
	NESDO	400		
Obsessive compulsive disorder	AMC OCD cohort	1158	Angst-, Dwang en Fobie-stichting (ADF stichting)	
	NOCDA	419		
Attention deficit hyperactivity disorder	NeurIMAGE	519	Balans	PsyQ
	IMpACT	250		Parnassia-BAVO
	Clinical adult ADHD cohort	4600		
Autism spectrum disorder	UMC ASD	998	Nederlandse Vereniging voor Autisme (NvA)	Leo Kannerhuis
	TRAILS	300		
	BOA cohort	158		
Posttraumatic stress disorder	TraumaTIPS	852	Veteranen Instituut	Stichting Arq
	AMC cohort PTSD	800		
	Police officers cohort	1000		

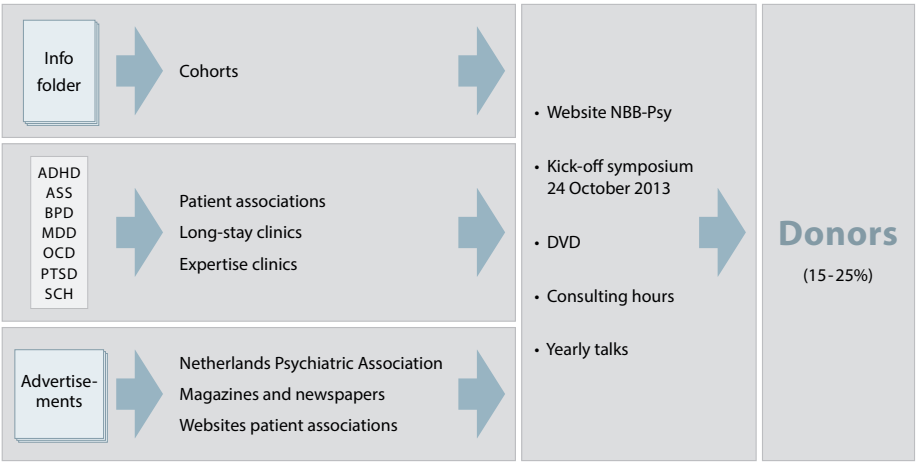


Figure 6 NBB-Psy Donor recruitment activities

Taken together, more than 50,000 people will be approached via the cohorts, associations and clinical settings. With a registration rate of 15-25%, we anticipate collecting at least 230-300 psychiatric and 125-200 control brains in the first 5 years and at least 450-600 psychiatric and 250-500 control brains in the coming 10 years.

Phenotyping

Phenotyping of participants of the psychiatric research cohorts has already been done extensively. When participants of the cohorts register as brain donors, they will be asked to give consent for the use of *all data* collected during the cohort study for research purposes.

Phenotyping of those potential donors who are not included in one of the clinical cohorts will be done with the MINI-Plus interview to confirm the psychiatric diagnosis, or, in the case of healthy controls, to rule out psychiatric symptoms.

For purposes of efficiency and to cause as little burden for the donors as possible, registration is done by means of a medical questionnaire. In addition, we will use web-based questionnaires to generate annual updates on relevant clinical parameters. For these web-based questionnaires, we will build a user-friendly, restricted-access website.

Healthy controls will be requested to fill in the questionnaire every five years (as fewer medical changes are expected to take place compared to patients with psychiatric disorders). The advantage of the regular update is that the health status of the control donors can be monitored during life.

Autopsies and post mortem phenotyping

The rapid autopsies will be performed within the framework of the NBB. Dissection protocols will be developed for each of the seven disorders. These dissection protocols have to comply with the current dissection protocols of the NBB, so that the control tissue can be supplied together with the NBB-Psy tissue. Also, the protocols of the seven disorders should mutually match, in order to be able to compare differences between the psychiatric disorders.

State-of-the-art neuropathological diagnoses will be performed and all data collected during life will be transferred to the NBB, where they will be anonymized, summarized and put into the NBB database. This information will be made available to the researcher. All personal data are fully protected according to national laws and European directives.

The NBB-Psy program is unique, because it uses novel methods to enrich the brain material by:

1. Isolation of primary microglia and astrocytes;
2. Generation of glial cell lines (microglia and astrocytes) from pure glial cells;
3. cDNA and DNA bank of the seven disorders;
4. Human induced pluripotent stem cells (iPSCs).

All the brain material will be made available to researchers via the application procedure of the NBB.

www.nhb-psy.nl
www.nbb-psy.nl
@NHBPsy

Autopsies & Diagnostics

Stabilization of annual number of autopsies

Since 1985 the NBB has obtained tissue from more than 3700 brain donors. The NBB performed 124 autopsies in 2011 and 129 autopsies in 2012. The number of autopsies has increased considerably since 2006 but has been stable since 2010. Table 4 shows the number of autopsies specified by diagnosis over the last 2 years. The numbers for 2012 are preliminary due to the delay in performing the post mortem diagnostic procedures, which means that the final diagnosis for 31% of the autopsies performed in 2012 is still pending.

Table 4 Annual numbers of autopsies by disease

	2006		2007		2008		2009		2010		2011		2012	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Contr	11	13%	12	13%	16	15%	17	15%	17	13%	25	20%	22	17%
AD	30	37%	28	31%	29	26%	28	25%	33	26%	39	31%	26	20%
FTLD/tau	3	4%	8	9%	16	15%	15	14%	13	10%	9	7%	5	4%
Other dem	9	11%	7	8%	13	12%	6	5%	13	10%	8	6%	7	5%
PSP	2	2%	3	3%	5	5%	9	8%	7	6%	4	3%	5	4%
MS	8	10%	14	16%	9	8%	11	10%	14	11%	12	10%	1	1%
PD/DLBD	6	7%	7	8%	12	11%	17	15%	18	14%	17	14%	11	9%
Psych	4	5 %	2	2%	4	4%	2	2%	3	2%	3	2%	3	2%
Other	9	11%	9	10%	5	5%	5	5%	9	7%	7	6%	9	7%
PANR					1	1%							40*	31%
Total	*82*		*90*		*110*		*110*		*127*		*124*		*129*	

* 15 of the autopsies listed as "PANR" are likely to be diagnosed as MS (these patients were clinically diagnosed with MS, a diagnosis that only rarely turns out to be false once the final neuropathological diagnosis has been determined). This brings the likely number of MS autopsies in 2012 to 16. However, PANR-autopsies with a different clinical diagnosis may also turn out to be MS cases.

Total number of brain donors

Figure 7 shows the total number of brain donors (3710) from which the NBB has obtained brain tissue since its start in 1985. Most of the tissue was obtained by means of autopsies performed by the NBB itself, but some was acquired from other sources (approximately 1472 donors).

During the last couple of years there have been relatively few autopsies of control donors and donors with Alzheimer's disease, and more of donors with other diagnoses, especially multiple sclerosis and Parkinson's disease / diffuse Lewy body dementia. This can be explained by the notion that the NBB started out as a brain bank focused on Alzheimer's disease. During the course of the NBB's existence the focus has shifted to include other diseases.

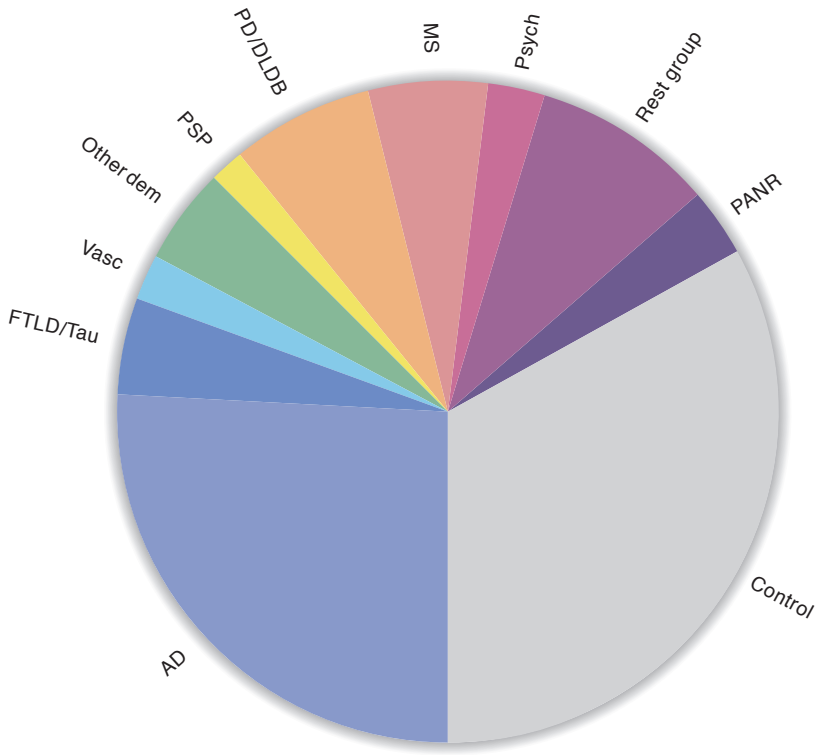


Figure 7 Total number of brain donations since 1985 (3710 on December 31, 2012)

Post mortem delay

Due to autolytic processes, tissue of the central nervous system quickly decays after death and there is thus only a small window of opportunity for brain autopsy. The post mortem delay (PMD: time elapsed from a person's demise to completion of the brain autopsy) depends on several factors: time of notification of the donor's death, distance and time for transportation of the body and the availability of brain bank staff to perform the autopsy. Because PMD has a strong impact on the quality of the tissue (i.e. RNA, DNA and proteins), several brain banks have established rapid autopsy protocols relying on 24/7 availability of staff. The NBB achieves short PMDs, with 65 % of all autopsies having a PMD between 4 to 8 hours, whereas the average PMD of other European brain banks is more than 12 hours in most cases, even when they work with a 24/7 availability of staff. Over the last 5 years the average PMD of the NBB autopsies has been extremely stable (figure 8).

Post mortem diagnostics

After completion of an autopsy the brain tissue is fixed in formalin for four weeks. After fixation, approximately eighteen standard regions of tissue are embedded in paraffin, cut and (immuno)histochemically stained. These sections are evaluated by one of our neuropathologists according to the latest international diagnostic criteria. Together with the clinical diagnosis this provides the definitive diagnosis. The fact that the final neuropathological diagnosis is often different from the initial clinical diagnosis emphasizes the importance of performing post mortem diagnostic pro-

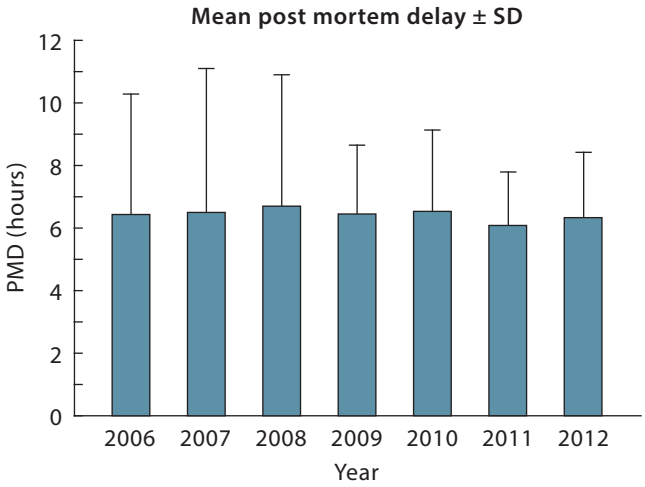


Figure 8

cedures. This is illustrated in table 5, which shows the neuropathological diagnoses for the recent autopsies of donors who were clinically diagnosed with Parkinson's disease. Clinical diagnoses of Parkinson's disease are followed by a different post mortem pathological diagnosis in a strikingly large number of cases, approximately 30% according to the NBB database. This general estimate is reflected in the numbers for 2010-2012 as well, although the numbers vary strongly per year.

Any possible discrepancies between the clinical and pathological diagnosis must be kept in mind when adjusting the registration policy. Donors recruited with a particular disease in mind may not all be suitable for use in research projects focused on that disease.

Table 5 Final neuropathological diagnoses for Parkinson protocol autopsies

Year	PD protocol autopsy	Final diagnosis	n	Diagnosis in PD spectrum
2010	12	PD	1	67%
		PD with dementia	6	
		Alzheimer's disease / Lewy body variant	1	
		Multi-system atrophy	2	
		Cortico-basal degeneration	1	
		Other	1	
2011	10	PD	7	100%
		PD with dementia	3	
2012	19	PD	5	53% (of the finished cases)
		PD with dementia	3	
		Multi-system atrophy	3	
		Progressive supranuclear palsy	1	
		Control with vascular encephalopathy	1	
		Non-Alzheimer dementia	1	
		Other	1	
		PA-report not ready	4	

Characterization of MS lesions

The NBB has an impressive collection of tissue blocks (4000 fixed or frozen) of more than 175 multiple sclerosis (MS) donors. The NBB dissects approximately 10-30 MS lesions per donor, a number of which is based on post mortem MRI guidance (in collaboration with VUmc) and a number on macroscopical appearance. In addition, tissue is dissected from standard locations for diagnostic purposes. Many tissue samples are mirror blocks of which one side is frozen in liquid nitrogen and the other fixed in formalin and embedded in paraffin. Most mirror blocks and diagnostic blocks have been analyzed using HE, Bodian and Klüver stains, and their histological appearances have been described by the neuropathologist of the NBB. However, standardized information about demyelinating activity, inflammation and neurodegeneration of the tissue blocks is not available, which hampers the effective dissemination of these MS tissues by the NBB.

We have cut and stained all tissue blocks of MS lesions and MS tissue blocks dissected for diagnosis for demyelination and inflammation using PLP-HLA double staining and characterized these for inflammation and demyelination. Scores are indicated in photographs of the lesions and provided to the researchers who apply for MS tissue (figure 9). This considerably facilitates dissemination of MS tissue. In addition, we have analyzed SNPs of the glucorticoid receptor, HLA, IL-1, IL-1RA and TNFalpha and measure markers of neurodegeneration (glutamate, neurofilament) and inflammation (s-CD163) in the CSF and extract clinical information from the clinical files of the MS brain donors. This project is a double-edged sword, since it also provides the unique opportunity to study the occurrence, incidence and distribution of the various types and stages of MS pathology, which enables correlation of this information with clinical and genetic characteristics in a large cohort of MS brain donors.

So far we have characterized all tissue blocks of 182 MS patients (until 2012) whose tissue was available at the NBB, and many of these have already been disseminated to research groups upon their official request. The DNA and CFS have been analyzed of a number of the MS brain donors and all clinical reports have been finalized. All data have been transferred to a MS post mortem database. Currently pathological, genetic and clinical data are being analyzed, to investigate whether specific pathology of MS relates to a specific clinical course and genetic background.

This project is financed by the VRIENDEN LOTERIJ

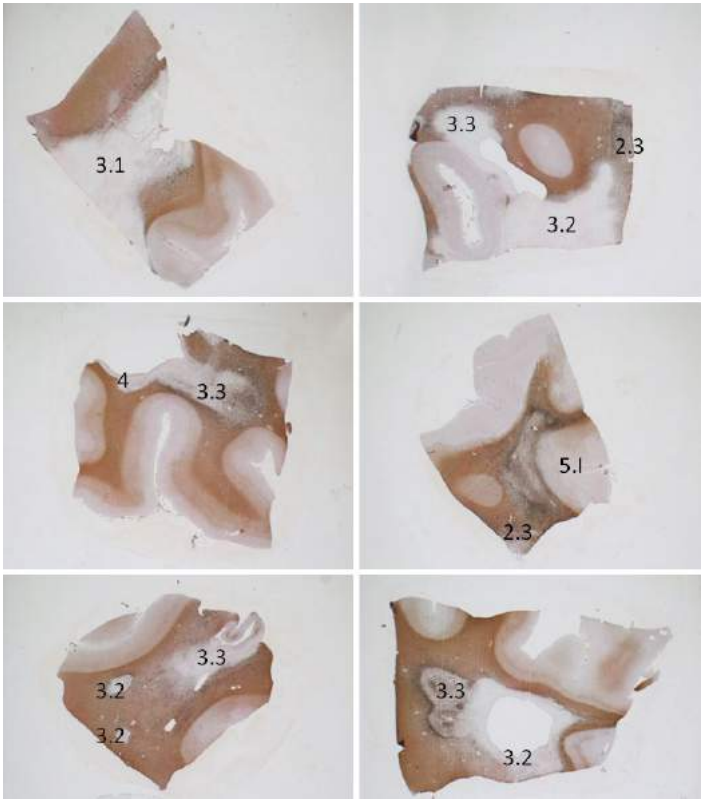


Figure 9 Photographs of MS lesion characterization.
Overview of 0.006 mm thick sections of MS lesions of a MS brain donor of the NBB. Myelin is brown, inflammatory activity (macrophages) is black. The number in the MS lesions indicates the type of MS lesion: no lesion (0), active lesion (2 or 3), inactive lesion (4), remyelination (6), in grey matter (5). Subscores indicate the degree of inflammatory activity.

Future plans

The tighter focus of the registration policy the NBB began in 2012 has been supplemented by a new autopsy policy as well. As of the beginning of 2013, the NBB reserves the right to decide not to perform autopsies under certain circumstances, for instance for diagnoses for which current tissue supplies are sufficient and/or for which research demand is low. This is particularly useful for cases in which donors with such a diagnosis are presumed to have become less suitable for research, for example when they have developed brain metastases of peripheral tumours. By March 2013 all donors had been informed in writing of this policy change.

Tissue Supply

Online tissue database: e-NBB

To professionalize the NBB's tissue sample dissemination procedure, the NBB has made its tissue database available online. As of late 2011 researchers may browse the database of this online application called the e-NBB (**www.e-nbb.org**) and make a tissue selection which they can send in along with the tissue application form. In 2012 the e-NBB was used to make a tissue selection for 27 applications. We expect the proportion of applications that make use of the e-NBB to increase as the e-NBB's existence becomes better known. We encourage this by informing researchers of the e-NBB when we receive a tissue availability inquiry.

Although this presumption is not yet supported by hard data, the first year of e-NBB use has given us the impression that the e-NBB has improved the efficiency of the tissue application procedure by decreasing the number of inquiries about tissue availability and the number of applications for tissue that the NBB does not have.

Number of research institutes that receive NBB tissue

In 2006, the NBB undertook to review all its procedures, which led to new informed consent forms and to professionalization of the application and tissue dissemination procedures. A Material Transfer Agreement (MTA) was drafted and put into use, to ensure the rights and obligations of the recipients of the tissue as well as those of the NBB. The first MTA was signed in June 2007. As of 31 December 2012, the NBB has entered into agreement with 101 universities / research institutes and 22 pharmaceutical companies worldwide. Once both parties have signed the MTA, which is valid for an indefinite period of time unless specified otherwise, any researcher within the institute can apply for tissue.

Number of tissue applications

The number of tissue applications has increased since the introduction of the new procedures, but has stabilized since 2009 (figure 10). Of the 96 applications received in 2011, 19 were from for-profit organizations (pharmaceuticals). Of the 98 applications received in 2012, 8 were from for-profit organizations.

Researchers may inquire about the availability of samples, which in most cases leads to an application. When it concerns a new research project, the application is re-

viewed by the NBB's scientific committee. If approved, a new project number is assigned and the necessary paperwork is done, after which the tissue is supplied. The review process takes approximately four weeks. For approved new applications in 2011 and 2012, the tissue was delivered on average 101 days after the tissue application date, with a standard deviation of 81 days. The large variance is mostly due to differences in the time needed after approval of the application, i.e. to agree on a suitable tissue selection and to finalize the necessary paperwork.

When the application concerns an existing research project that has already been reviewed, this is called a supplementary application. The option of filing a supplementary application was introduced in 2007, together with the MTA. With the original research project already approved, the requested tissue can be supplied even more quickly, provided that the tissue is available and the type and number of samples are reasonable compared to the original application. For approved supplementary applications in 2011 and 2012, the tissue was delivered on average 37 days after the supplementary application date, with a standard deviation of 57 days.

In 2011 and 2012 there were 15 cases (out of 194) where tissue inquiries led to applications that could not be approved, where approved applications could not be completed, or where the inquiry did not lead to an actual application. The main reasons why tissue inquiries or applications foundered are:

1. an application form was sent to the researcher, but the researcher never actually applied for tissue;

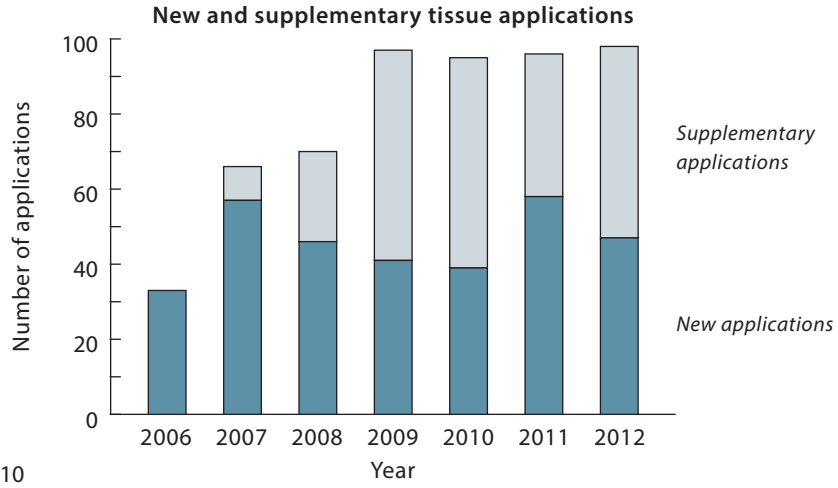


Figure 10

2. the researcher had to cancel the application due to financial problems (rejected grant applications).

There were also a number of applications that could only be partly approved due to tissue scarcity, which shows the need to increase the number of donors with a specific neurological or psychiatric disorder. This was one of the reasons to start donor recruitment efforts among clinical cohorts and to start NBB-Psy, the separate donor program for psychiatric diseases. In addition to tissue from donors with psychiatric diseases, other examples of scarce tissue types are frozen hippocampus samples from control donors and substantia nigra samples from donors with Parkinson's disease.

Tissues disseminated for research projects

Figure 11 shows the specification of supplied samples by diagnosis in 2011 and 2012, compared to the tissue supply in 2007-2010. Since 2008 the number of supplied tissue units has increased. This increase is equally distributed across the different diagnoses. As the NBB aims to increase the number of disseminated tissue samples, we will begin to explore possibilities to do so in 2013.

In line with the tissue applications, the majority of tissue samples were supplied to researchers affiliated to universities or other non-profit organizations. In 2011, 470 units out of a total of 4664 tissue units were supplied to for-profit organizations (pharmaceutical companies). In 2012, 4678 tissue units were supplied, of which 59 to for-profit organizations.

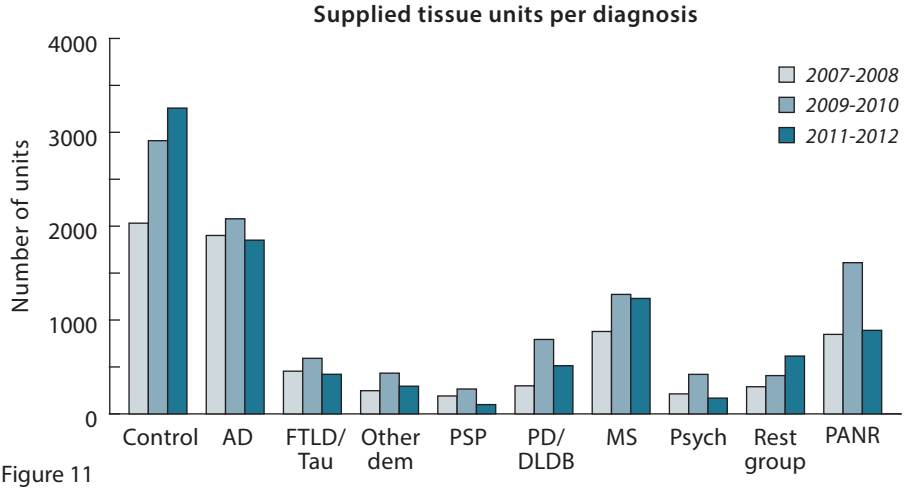


Figure 11

Figure 12 shows the specification of the samples by type of storage. The NBB not only provides frozen or formalin-fixed paraffin-embedded (FFPE) samples, but also fresh tissue and formalin-fixed tissue. The different treatments of the tissue allow different kinds of research approaches. In general, FFPE samples are especially suitable for performing immunohistochemistry and morphometric studies. For RNA and protein analysis studies mainly frozen tissue is used. The proportion of supplied fresh tissue has been increasing since 2009, which reflects the increased usage of tissue cultures and glial cell isolations in research.

Database mining

In addition to the NBB's collection of brain tissue samples, our database contains a wealth of clinical and neuropathological information about the donors. When researchers receive tissue, this is accompanied by the information files for all donors of whom they have received samples. However, the information in the database also provides ample research opportunities on its own. Over the last few years, there has been an increase in the number of requests from researchers who would like to analyze data from the NBB database without the actual use of brain tissue. In 2012, the NBB has formulated standardized guidelines for these situations in terms of financial contribution and corporate authorship, which can be found on the **website**. In the future, we intend to increase awareness regarding this possibility among researchers in order to enable more studies to make use of the valuable data.

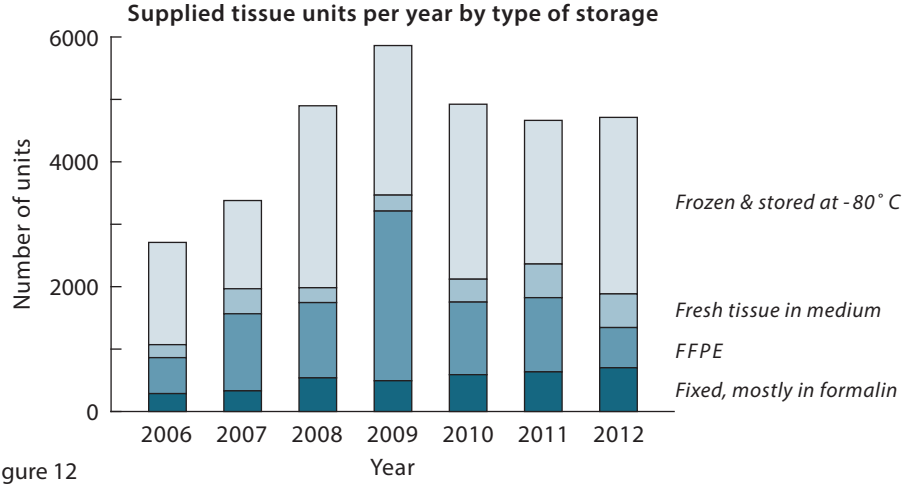


Figure 12

The possibilities of analysis of brain banks databases were an important topic of the MacBrain symposium (December 6th, 2012), organized by the Institute for Computing and Information Sciences (Radboud University Nijmegen) in cooperation with the NBB. The goal of the symposium was to provide researchers and practitioners with an overview of the cutting edge research on core aspects of integrative research of brain diseases, combining data management, machine learning and clinical and pathological studies.

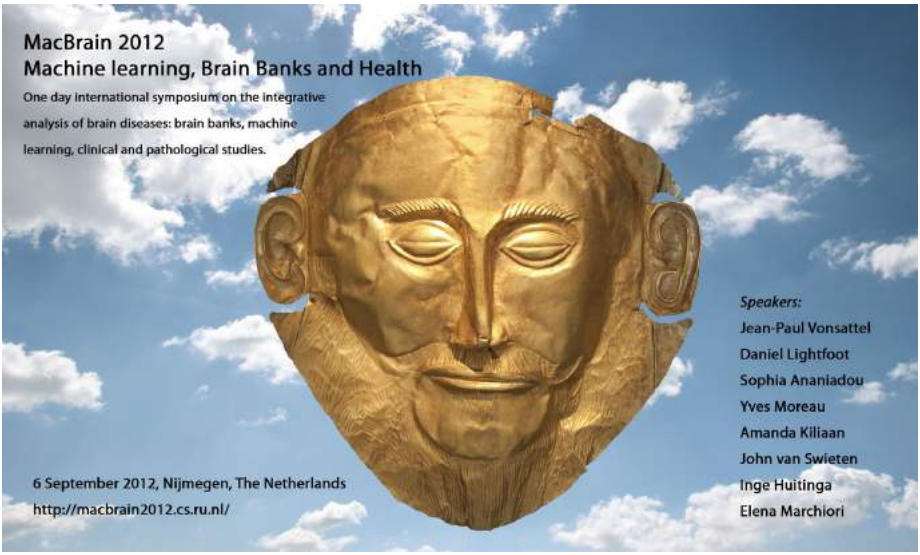


Figure 13 Announcement for the MacBrain symposium

Finances

The NBB receives structural financial support from the Royal Netherlands Academy of Arts and Sciences (KNAW) and the Netherlands Institute for Neuroscience (NIN), but apart from that it is almost completely dependent upon grants, donations and the financial contributions from researchers who use NBB material.

Grants	**2011**	**2012**
Structural contribution from the KNAW	€ 224,144	€ 224,144
Structural contribution from the NIN	€ 100,000	€ 100,000
Stichting MS Research	€ 106,254	€ 106,254
Internationale Stichting Alzheimer Onderzoek	€ 29,648	€ 24,706
Internationaal Parkinson Fonds	€ 25,000	€ 25,000
Hersenstichting Nederland	€ 12,000	€ 12,000

Grant for NBB-Psy	**2012-2017**
NWO (Netherlands Organisation for Scientific Research)	€ 3,450,000*

* This funding was jointly granted to the NBB and the five participating Dutch university medical centers for setting up the separate NBB-Psy program, as described in the chapter on Registrations. The project and its budget are coordinated by the NBB under the responsibility of the KNAW.

The necessity of grants

The costs to make tissue available for research are approximately € 800,000 per year. Without the help of patient organizations the NBB would not be able to maintain its high standards, and it is only thanks to the funding the NBB receives that it is able to continue brain banking.

The **Stichting MS Research** (MS Research Foundation; **www.msresearch.nl**) has been funding the NBB for many years, resulting in an increase of the number of MS donors and in the availability of MS tissue. Due to the special MRI-guided dissection protocol, the autopsy costs for MS are higher than for other autopsies. Moreover, the clinical files of people with MS are often more extensive and the summarization of their medical information requires a greater effort. Lastly, in-depth neuropathological diagnostics of the MS plaques is time-consuming, but indispensible for good

tissue dissemination. MS Research covers the costs of all MS autopsies and of some control autopsies.

The funding by the **Internationale Stichting Alzheimer Onderzoek** (International Foundation for Alzheimer Research; **www.alzheimer.nl**) has made it possible for the NBB to start up and maintain a DNA bank to keep up with the latest developments in research, where genotyping is becoming the important bridge between clinical and neuropathological characteristics. Previous support from the ISAO (11-01-2007 to 10-31-2009) allowed us, among other things, to produce a promotional DVD on the work of the NBB. € 17,000 of this grant was allocated to the production of the DVD. In 2011 and 2012 many copies of the DVD were distributed among (potential) donors.

The grants of the **Internationaal Parkinson Fonds** (International Parkinson Fund; **www.parkinsonfonds.nl**) cover the costs of a part of the Parkinson autopsies and some donor recruitment activities, which would not be possible without this extra funding.

Funding by the **Hersenstichting Nederland** (Netherlands Brain Foundation; **www.hersenstichting.nl**) is used to cover donor recruitment, autopsy and administration costs.

Donations

The "Stichting tot Ondersteuning van de Hersenbank" (Foundation for the Support of the NBB) was founded in 1986 and helps to realize the objectives of the NBB by giving financial support. In 2011 and 2012, we received € 17,700 in donations through this foundation.

Since January 2008, the foundation has been deemed an "Algemeen Nut Beogende Instelling" (Institution for Public Advancement) by the Dutch Tax Authorities. The assets of this Foundation are made up of donations, testamentary dispositions and legacies (Dutch Chamber of Commerce; registration no. 41205869). In the last quarter of 2013, the current Foundation for the Support of the NBB will be converted into the "Stichting Vrienden van het Herseninstituut" (Friends of the Brain Institute). Financial donations remain vital to the continued existence of the NBB and are thus very welcome. If you wish to help, please make your donation to: Stichting Vrienden van het Herseninstituut, account number 2167378 (IBAN: NL76INGB0002167378; BIC: INGBNL2A), mentioning "NBB". Because the foundation also raises money for

the Netherlands Institute for Neuroscience in general, mentioning "NBB" ensures that your donation reaches us.

We are very grateful for all grants and donations. The work of the NBB would not be possible without the support of numerous foundations, patient organizations, and the enthusiastic dedication of private individuals.

Research Projects 2011-2012

The abstracts can be downloaded from our website by clicking on the names below or by visiting **http://www.brainbank.nl/research/projects**

National

Anink, J. and Aronica, E. Department of (Neuro)Pathology, Academic Medical Center, Amsterdam. Adenosine hypothesis of Parkinson's disease.

Bonifati, V. Department of Clinical Genetics, Erasmus MC, Rotterdam. Characterization of the FBXO7 (PARK15) protein.

Bonifati, V. Department of Clinical Genetics, Erasmus MC, Rotterdam. Hereditary Parkinsonism and Dystonia with Hypermanganesemia, Polycythemia and Chronic Liver Disease caused by mutations in the *SLC30A10* gene.

Bossers, K. et al. Netherlands Institute for Neuroscience, Amsterdam. Alteration of the microRNA network during the progression of Alzheimer's disease.

Brouwer, N., Boddeke, H.W. et al. Medical Physiology, Department of Neuroscience, University Medical Center Groningen, Groningen. Analysis of age-related changes in gene expression in human microglia.

Bruinsma, I. and De Jong, B. Department of Neurology, Radboud University Medical Center, Nijmegen. Role of miRNAs in the pathology of multiple sclerosis.

Bsibsi, M., Amor. S. et al. Department of Pathology, VU University Medical Center, Amsterdam. Alpha B-crystallin activates an immune-regulatory response of microglia in preactive multiple sclerosis lesions.

Creyghton, M.P. Hubrecht Institute for Developmental Biology and Stem Cell Research, Utrecht. Epigenetic profiling of cis regulatory elements in the brain.

Dijkstra, A.A. and Van de Berg, W.D.J. et al. Department of Anatomy and Neurosciences, section Functional Neuroanatomy and Department of Pathology and Department of Medical genomics, VU University Medical Center, Amsterdam. Identifying molecular mechanism underlying the progression of sporadic Parkinson's disease using advanced genomic and proteomic techniques.

Doorn, K.J. et al. Department of Anatomy and Neurosciences, VU University Medical Center, Amsterdam. Microglial activation beyond the substantia nigra in Parkinson's disease.

Drukarch, B. and Wilhelmus, M.M.M. Department of Anatomy and Neurosciences, VU University Medical Center, Amsterdam. Association of tissue transglutaminase and lysyl oxidase with cerebral amyloid angiopathy.

Espitia Pinzón, N. and Van Dam, A.M. Department of Anatomy and Neurosciences, VU University Medical Center, Amsterdam. Tissue Transglutaminase in astrogliosis: towards improved remyelination.

Forstmann, B.U. and Alkemade, A. Cognitive Science Center Amsterdam, University of Amsterdam, Amsterdam. Subdivisions of the Subthalamic Nucleus.

Gao, S.F. et al. Netherlands Institute for Neuroscience, Amsterdam. Decreased NOS1 Expression in the Anterior Cingulate Cortex in Depression.

Gao, S.F. et al. Netherlands Institute for Neuroscience, Amsterdam. Reduced GAD65/67 immunoreactivity in the hypothalamic paraventricular nucleus in depression: A postmortem study.

Hendrickx, D. et al. Neuroimmunology Research Group, Netherlands Institute for Neuroscience, Amsterdam. Mechanisms of myelin phagocytosis in multiple sclerosis.

Hepp, D, Van de Berg, W.D.J. et al. Department of Anatomy and Neurosciences, Department of Neurology and Department of Pathology, VU Medical Center, Amsterdam. The pathological substrate of visual hallucinations in Parkinson's disease patients.

Hondius, D.C., Smit, A.B. et al. Department of Pathology, VU University Medical Center, Amsterdam and Department of Molecular and Cellular Neurobiology, Center for Neurogenomics and Cognitive Research, VU University Amsterdam, Neuroscience Campus Amsterdam. Changes in the human hippocampal proteome during Alzheimer's disease.

Hoozemans, J., Rozemuller, J.M. and Van der Vies, S. Department of Pathology, VU University Medical Center, Amsterdam. Detection of kinase activity in post mortem cerebrospinal fluid.

Huitinga, I. et al. Department of Neuroimmunology, Netherlands Institute for Neuroscience, Amsterdam. Identification of a key role for complement in neurodegeneration in multiple sclerosis.

Ingrassia, A. and Van de Berg, W.D.J. et al. Department of Anatomy and Neurosciences and Department of Pathology, VU University Medical Center, Amsterdam. Neuroprotection and degeneration in the olfactory bulb of Parkinson patients.

Iyer, A.M. et al. Department of (Neuro)Pathology, Academic Medical Center, Amsterdam. Developmental patterns of DR6 in normal human hippocampus and in Down syndrome.

Iyer, A.M., Aronica, E. et al. Department of (Neuro)Pathology, Academic Medical Center, Amsterdam. Pathways common to brain development and aging.

Kan, A.A., De Graan, P.N.E. et al. Department of Neuroscience & Pharmacology, Brain Division, UMC Utrecht. Towards unravelling the activated immune system in refractory temporal lobe epilepsy patients.

Kamphuis, W. et al. Department of Astrocyte Biology and Neurodegeneration, Netherlands Institute for Neuroscience, Amsterdam. Characterization of Glial Fibrillary Acidic Protein (GFAP) isoform expression in plaque related astrogliosis in human Alzheimer Disease.

Klaver, R., Geurts, J.J.G. et al. Department of Anatomy & Neuroscience, VU University Medical Center, Amsterdam. Grey matter atrophy in MS.

Kondova, I. Division of Pathology and Microbiology, Department of Animal Science, Biomedical Primate Research Center, Rijswijk. Age-related neurological disorders: comparison of brain tissues from humans, chimpanzees and rhesus macaques and exploring the role of miRNAs and small non-coding RNAs (ncRNAs) in the pathogenesis of neurodegeneration.

Kooij, G. et al. MS Center Amsterdam, Department of Molecular Cell Biology and Immunology, VU University Medical Center, Amsterdam. The blood-cerebrospinal fluid barrier: the primary site for inflammation in multiple sclerosis.

Kooijmans, R. et al. Vision and Cognition group, Netherlands Institute for Neuroscience, Amsterdam. Comparing calcium binding protein distribution in mouse, macaque and human.

Kreft, K.L., Hintzen, R.Q. et al. Department of Neurology and MS Center ErasMS, Erasmus MC, Rotterdam. Genetic determinants of kinesin expression in multiple sclerosis patients.

Kuiperij, H.B. and Verbeek, M.M. Department of Neurology, Radboud University Medical Center, Nijmegen. TDP-43 and tau as cerebrospinal fluid biomarkers to discriminate frontotemporal dementia subtypes.

Lopes Soriano, A., Geurts, J.J.G. et al. MS Center Amsterdam, Department of Anatomy & Neuroscience, VU University Medical Center, Amsterdam. Use of quantitative magnetic resonance imaging techniques to stage white matter lesions in multiple sclerosis. MRI-pathology correlation study (pilot).

Lucassen, P.J. SILS-Center for Neuroscience, University of Amsterdam, Amsterdam. Changes in glucocorticoid receptor expression and regulation in human hippocampus, amygdala during depression and dementia.

Luchetti, S., Huitinga, I. et al. Neuroimmunology Research group and Netherlands Brain Bank, Netherlands Institute for Neuroscience, Amsterdam. Characterization of MS lesions of the Netherlands Brain Bank.

Luchetti, S., Huitinga, I. et al. Department of Neuroimmunology, Netherlands Institute for Neuroscience, Amsterdam. Sex differences in neurosteroidogenesis in multiple sclerosis pathology.

Luchetti, S., Swaab, D.F. et al. Laboratory for Neuropsychiatric Disorders and Laboratory of Neuroimmunology, Netherlands Institute for Neuroscience, Amsterdam. Neurosteroids as potential neuroprotective agents in Parkinson's disease: implications for new therapeutic strategies.

Melief, J., Huitinga, I. et al. Netherlands Institute for Neuroscience, Amsterdam. Phenotyping primary microglia from the normal and multiple sclerosis brain.

Müller, M. et al. Department of Neurology, Donders Institute for Brain, Cognition and Behaviour, Radboud University Medical Center, Nijmegen. Differential expression of microRNAs in the hippocampus of Alzheimer's disease patients.

Nabuurs, R. et al. Departments of Radiology, Pathology and Anatomy, Leiden University Medical Center, Leiden. Histological basis of MRI visualization of AD/CAA in ex vivo human brain tissue.

Nijholt, D.A.T., Scheper, W. et al. Department of Genome Analysis and Department of Neurology, Academic Medical Center and Department of Neuropathology VU University Medical Center, Amsterdam. Activation of the unfolded protein response in neurodegenerative tauopathies.

Peferoen, L.A.N., Vogel, D.A.S. et al. Department of Pathology and Department of Molecular Cell Biology and Immunology, VU University Medical Center, Amsterdam. Do stressed oligodendrocytes trigger microglia activation in pre-active MS lesions?

Prins, M., Van Dam, A. et al. VU University Medical Center, Neuroscience Campus Amsterdam, Amsterdam. Chemokine signalling in the hippocampus of multiple sclerosis patients.

Qi, X.R. et al. Netherlands Institute for Neuroscience, Amsterdam. Aberrant stress hormone receptor balance in the human prefrontal cortex and hypothalamic paraventricular nucleus of depressed patients.

Qi, X.R. et al. Netherlands Institute for Neuroscience, Amsterdam. Abnormal retinoid and TrkB signaling in the prefrontal cortex in mood disorders.

Qi, X.R. et al. Netherlands Institute for Neuroscience, Amsterdam. Alterations in the neurosteroid biosynthetic pathways in the human prefrontal cortex in mood disorders: a postmortem study.

Ramaglia, V. et al. Department of Neuroimmunology, Netherlands Institute for Neuroscience, Amsterdam. A novel C3-dependent mechanism of microglial priming relevant to multiple sclerosis.

Reijerkerk, A. et al. Blood-Brain Barrier Research Group, Molecular Cell Biology and Immunology, VU University Medical Center, Amsterdam. MicroRNAs regulate human brain endothelial cell barrier function in inflammation: implications for multiple sclerosis.

Riese, H., Niezen-Koning, K. et al. University Medical Center Groningen, Groningen. Comparison of methylation and expression of the serotonin reuptake transporter gene in amygdala tissue, cerebrospinal fluid and peripheral blood.

Rizzu, P. et al. Medical Genomics, VU University Medical Center, Amsterdam. Regional differences in gene expression and promoter usage in aged human brains.

Rozemuller, J.M. et al. Department of Neuropathology, VU University Medical Center, Amsterdam. Neurovascular dysfunction in Alzheimer's disease.

Rozemuller, J.M. et al. Department of Neuropathology, VU University Medical Center, Amsterdam. The pathology distribution of the non-memory phenotype of Alzheimer disease.

Schenk, G.J., Geurts, J.J.G. et al. Department of Anatomy & Neuroscience, VU University Medical Center, Amsterdam. Pathobiology of MS: complex interplay between degeneration and inflammation.

Schwab, B.C. et al. Applied Analysis and Mathematical Physics and Biomedical Signals and Systems, University of Twente, Enschede. A Possible Role of Neural Gap Junctions in Parkinson's Disease Pathology.

Shan, L. et al. Netherlands Institute for Neuroscience, Amsterdam. The human histaminegic system in health and neuropsychiatric disorders.

Smolders, J., Hamann, J. et al. Neuroimmunology Research Group, Netherlands Institute for Neuroscience, Amsterdam and Department of Experimental Immunology, Academic Medical Center, Amsterdam. Characteristics of differentiated CD8+ and CD4+ T cells present in the human brain.

Smolders, J., Huitinga, I. et al. Neuroimmunology Research Group and Astrocyte Biology and Neurodegeneration Group, Netherlands Institute for Neuroscience, Amsterdam. Expression of vitamin D receptor and metabolizing enzymes in multiple sclerosis-affected brain tissue.

Van de Berge, S., Van Strien, M. and Hol, E. Astrocyte Biology & Neurodegeneration, Netherlands Institute for Neuroscience, Amsterdam. The proliferative capacity of subventricular neural stem cells is maintained in the Parkinsonian brain.

Van Dijk, K.D. and Van de Berg, W.D.J. Department of Anatomy and Neurosciences, section Functional Neuroanatomy and Department of Neurology, Neuroscience Campus Amsterdam, VU University Medical Center, Amsterdam. The expression of clusterin in the entorhinal cortex in Parkinson's disease: a pilot study.

Van Luijn, M.M., Hintzen, R.Q. et al. Departments of Immunology and Neurology and MS Center ErasMS, Erasmus MC, Rotterdam. Clusterin and chromogranin A expression and localization in white and grey matter brain tissue of multiple sclerosis patients.

Van Nierop, G.P. and Verjans, G.M.G.M. et al. Departments of Viroscience and Neurology and ErasMS center Neurology, Erasmus MC, University Medical Center, Rotterdam. Specificity and phenotype of T cells in MS lesions.

Van Riel, D. et al. Department of Viroscience, Erasmus MC, Rotterdam. The olfactory nerve: A shortcut for influenza viruses into the CNS?

Van der Star, B. and Amor, S. Department of Pathology, VU University Medical Center, Amsterdam.

Van Swieten, J.C. Department of Neurology, Erasmus MC, Rotterdam. Immunohistochemistry and biochemical characterisation of frontotemporal dementia.

Van Velzen, M, Verjans, G.M.G.M. et al. Department of Virology, Erasmus MC, Rotterdam. Latent herpes simplex virus and varicella zoster virus infections of sensory neurons of the peripheral nervous system.

Veerhuis, R. and Hoozemans, J. Department of Clinical chemistry and Alzheimer center and department of Pathology, VU University Medical Center, Amsterdam. Analysis of mediators of inflammation in Alzheimer's disease.

Verwer, R.W.H. and Swaab, D.F. Netherlands Institute for Neuroscience, Amsterdam. Reactivation and functional activity of neurons in cultured postmortem brain tissue slices.

Willems, J. et al. Department of Neuroimmunology, Netherlands Institute for Neuroscience, Amsterdam. Immunopathology of the hippocampus in multiple sclerosis.

International

Alberio, T. and Fasano, M. Department of Theoretical and Applied Sciences; Laboratory of Biochemistry and functional Proteomics; University of Insubria, Varese, Italy. Analysis of proteasomal and autophagic function in post-mortem Parkinson's disease tissues.

Andreyeva, A. and Gottmann, K. Institute for Neuro- and Sensory Physiology, University Düsseldorf, Düsseldorf, Germany. Role of N-cadherin in the aging human brain and in Alzheimer´s disease.

Bayer, T.A. Department of Psychiatry, University Medicine Goettingen, Goettingen, Germany. Intraneuronal Abeta accumulation in Alzheimer's disease.

Berson, A., Soreq, H. et al. Department of Biological Chemistry and the Edmond and Lily Safra Center of Brain Science, Hebrew University of Jerusalem, Jerusalem, Israel. Cholinergic-associated loss of hnRNP-A/B in Alzheimer's disease impairs cortical splicing and cognitive function in mice.

Chung, S. et al. Department of Psychiatry, Asan Medical Center, University of Ulsan College of Medicine. Seoul, South Korea. Relationships between intracellular Na levels and circadian rhythms in SCN2.2 cell.

Cranley, D. And Brophy, P. Centre for Neuroregeneration, University of Edinburgh, Edinburgh, Scotland. Aberrant ion channel expression and nodal disruption within dorsal root ganglia in multiple sclerosis.

Csiba, L. and Farkas, S. Department of Neurology, University of Debrecen, Debrecen, Hungary. A comparative analysis of expressed and functionally active dopamine receptors in the human brain obtained from Parkinson's disease patients and age matched controls.

Curtis, M.A. and Graham, S. Centre for Brain Research, Auckland University, Auckland, New Zealand. Cannabinoid receptor expression in MS lesions.

Darreh-Shori T. and Unger Lithner, C. Alzheimer Neurobilogy Center, Karolinska Institute, Stockholm, Sweden. The pathological mechanisms of β-amyloid in the brain of Alzheimer's disease and controls.

Delalle, I. et al. Boston University School of Medicine and Harvard NeuroDiscovery Center, USA. Exosomal and cell-class specific miRNA-profiles in bipolar disorder.

Deleersnijder, A., Baekelandt, V. et al. Laboratory of Neurobiology and Gene Therapy, K. U. Leuven, Belgium. Comparative analysis of different peptidyl-prolyl isomerases reveals FK506-binding protein 12 as the most potent enhancer of α-synuclein aggregation.

Duan, S. et al. Department of Neurobiology, Zhejiang University School of Medicine, Hangzhou, China. The protective role of purinergic receptors against the pathogenesis of Alzheimer's disease.

Fernandez-Ruiz, J. et al. Instituto Universitario de Investigación en Neuroquímica, Department of Biochemistry and Molecular Biology, Faculty of Medicine, Complutense University, Madrid, Spain. Evaluation of the endocannabinoid receptors and enzymes in the postmortem cerebellum of different SCA patients.

Francescatto, M. et al. Genome Biology of neurodegenerative diseases, DZNE, Tubingen, Germany. Profiling the promoterome of the human brain.

Fraussen, J. and Somers, V. Hasselt University, Biomedical Research Institute, and Transnationale Universiteit Limburg, School of Life Sciences, Diepenbeek, Belgium. Antibody-independent effects of B cells in multiple sclerosis (MS).

Galter, D. Department of Neuroscience, Karolinska Institutet, Stockholm, Sweden. Expression and quantification of candidate genes of Alzheimer's disease in brain regions primarily affected by disease.

Grosser, C., Van de Nes, J.A.P. et al. Institute of Human Genetics and Institute of Pathology and Neuropathology, Faculty of Medicine, University Duisburg-Essen, Essen, Germany. Methylation analysis of *SST* and *SSTR4* promoters in the neocortex of Alzheimer's disease patients.

Hemelsoet, D. et al. Department of Neurology/LCEN3, Ghent University Hospital, Ghent, Belgium. The role of matrix metalloproteinases in excitotoxicity and neuroinflammation in temporal lobe epilepsy.

Hökfelt, T. and Ceccatelli, S. Department of Neuroscience, Karolinska Institute, Stockholm, Sweden. To characterize the expression levels of galanin and its receptors in human pituitary tumors.

Houlden, H. UCL Institute of Neurology, London, United Kingdom. Genetic analysis of inherited leukodystrophies: genotype-phenotype correlations in the CSF1R gene.

Ikemoto, K. et al. Department of Neuropsychiatry, Fukushima Medical University School of Medicine, Fukushima, Japan. DNA methylation status of MAOA and MAOB genes in post-mortem brains of patients with schizophrenia.

Ishunina, T., and Swaab, D.F. Department of Histology, Embryology, Cytology, Kursk State Medical University, Kursk, Russia and Netherlands Institute for Neuroscience, Amsterdam, the Netherlands. Estrogen receptor α splice variants in the human brain.

Jones, E. et al. Wolfson Centre for Age-Related Diseases, King's College London, London, United Kingdom. Functional Genetic Analysis: Mechanisms of Dementia in People with Down syndrome.

Kalmar, B., Greensmith, L. and Fisher, E. UCL Institute of Neurology, Queen Square London, United Kingdom. Lower motor neuron pathology in Down's syndrome.

Kaut, O. et al. Department of Neurology, University of Bonn, Bonn, Germany. Genome-wide DNA methylation analysis of depression in human brain samples.

Klafki, H.W. et al. Department of Psychiatry and Psychotherapy, University of Duisburg-Essen, LVR-Klinikum, Essen, Germany. Identification of post-translational modifications of the β-amyloid peptides in amyloid plaques.

Ko, E.A. et al. Yonsei University College of Medicine, Seoul, South Korea. Interaction of HMGB1 with α-synuclein and the effect on α-synuclein aggregation.

Köse, M. et al. PharmaCenter Bonn, Pharmaceutical Institute, Pharmaceutical Chemistry I, University of Bonn, Bonn, Germany. GPCR distribution in human brain from patients with Alzheimer's and Parkinson's disease.

Krol, A. et al. Architecture et Réactivité de l'ARN, Institut de Biologie Moléculaire et cellulaire, Strasbourg, France. Structure-function of the long non-coding HAR1 RNA in the human brain.

Leuze, C.W.U., et al. Department of Neurophysics, Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany. 3D cortical profiles of diffusion MRI data in primary motor (M1) and somatosensory (S1) cortex.

Mikkelsen, J.D. Neurobiology Research Unit, University Hospital Rigshospitalet, Copenhagen, Denmark. Detection of nicotinergic receptors in normal and diseased brain.

Mott, N. and Pak, T.R. Loyola University Chicago, Chicago, USA. Mapping human estrogen receptor beta splice variants in the aged brain.

Nichterwitz, S. et al. Department of Neuroscience, Karolinska Institutet, Stockholm, Sweden. Characterization of human protein expression in motor neuron populations that are resistant or vulnerable to degeneration in Amyotrophic Lateral Sclerosis.

Nielsen, H.M., Wennström, M. et al. Lund University, Dept of Clinical Sciences, Molecular Memory Research Unit, Skåne University Hospital, Malmö, Sweden. Analyses of distribution and activation of NG2-cells in the Alzheimer´s brain.

Niola, F. and De Pietri Tonelli, D. Neuroscience and Brain Technologies department, Fondazione Istituto Italiano di Tecnologia (IIT), Genova, Italy. Somatic 22q11 loss of heterozygosity in psychiatric disorders.

Notter, T. et al. Institute of Pharmacology and Toxicology, University of Zurich, Switzerland. Characterization of Reelin-positive deposits in the human postmortem brain of non-demented subjects and patients with AD.

O'Neill, C. et al. Neurobiology and Alzheimer's Disease Laboratory, Department of Biochemistry, BioSciences Institute, University College Cork, Cork, Ireland. Examination of the Akt/PTEN signalling system in Alzheimer's disease and related disorders.

Papanikolopoulou, K. and Skoulakis, E.M.C. Division of Neuroscience, Biomedical Sciences Research Centre "Alexander Fleming", Vari, Greece. Use of phosphorylated Tau as a biomarker.

Picardi, E. and Eisenberg, E. Istituto di Biomembrane e Bioenergetica del CNR, Bari, Italy. Assessment of global A-to-I RNA editing patterns in Alzheimer's disease by parallel DNA capturing and sequencing.

Preisner, A. et al. Institute of Neuropathology, University Hospital Münster, Münster, Germany. Functional role for Wnt/β-Catenin for remyelination (failure) in MS.

Pressey, S. et al. Institute of Neurology, University College London, Queen Square Brain Bank, London, United Kingdom. Proteomic investigation into pathological progression in Parkinson's disease.

Qiao, J.P., Zhou, J.N. et al. CAS Key Laboratory of Brain Function and Disease School of Life Sciences, University of Science and Technology of China, Anhui, China. Novel Indanone Derivatives as Potential Imaging Probes for β-Amyloid Plaques in the brain.

Rak, M. et al. Department of Chemistry, University of Manitoba, Manitoba, Canada. Synchrotron infrared imaging applied to the analysis of molecular changes accompanying Alzheimer's disease.

Riederer, P. et al. Clinic and Policlinic of Psychiatry, Psychosomatics and Psychotherapy, University of Wuerzburg, Germany. Neuromelanin of Substantia nigra-pathological aspects of Parkinson's disease.

Rizzu, P. et al. Applied Genomics of neurodegenerative diseases, DZNE, Tubingen, Germany. Expression profiling of FTD patients with mutations in *MAPT*, *GRN* and *c9orf72*.

Soreq, H. et al. The Edmond & Lily Safra Center for Brain Sciences and the Department of Biological Chemistry, Hebrew University of Jerusalem, Israel. Profiling of alternative splicing in early stages of Alzheimer's disease.

Sriram, S. and Seely, E. Vanderbilt Medical Center, Multiple Sclerosis Laboratory, Nashville, USA. MALDI analysis of spinal cord tissue of MS patients and controls.

Stüber, C. et al. Department of Neurophysics, Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany. Iron and myelin in human brain tissue: histology, PIXE, and ultra-high-field MRI susceptibility maps.

Szodorai, A. and Nitsch, R.M. Molecular Psychiatry, University of Zurich, Zürich, Switzerland. Analysis of the putative presence of dendritic cells in demented brains.

Szodorai, A. and Nitsch, R.M. Molecular Psychiatry, University of Zurich, Zürich, Switzerland. Reduction of pyroglutamate-Abeta (pEAbeta) containing HC brain cells in AD.

Tiepolt, S. et al. Department of Nuclear Medicine, University of Leipzig, Leipzig, Germany. Validation of 7 Tesla MRI to image β-amyloid plaque associated iron in Alzheimer's disease.

Wang, Y. Institute of Neuroscience, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, Shanghai, China. Neuroprotection of TRPC6 channels in Alzheimer's disease.

Wanker, E.E. Max Delbrück Centrum für Molekulare Medizin (MDC), Berlin, Germany. Small molecule AMC3.1 for AD therapy by converting monomeric and oligomeric ABeta to protofibrils.

Wähnert, M. et al. Department of Neurophysics, Max Planck Institute for Human Cognitive and Brain Sciences, Leipzig, Germany. Do cortical layers conform to the Laplace equation?

Webb, S. et al. Institute of Neurosciences, Department of Neurology, Southern General Hospital, Glasgow, Scotland, United Kingdom. A comparison of viral infections in lymph nodes of patients with Multiple Sclerosis and normal controls.

Wennström, M. and Nielsen, H.M. Department of Clinical Sciences, Lund university, Malmö, Sweden. In vitro studies on glial targets in neurodegenerative dementia.

Yoon, S-Y. and Kim, D-H. Department of Anatomy and Cell Biology, University of Ulsan College of Medicine, Seoul, Korea. Search for the key pathogenic molecules in Alzheimer's disease brain.

Zhou, J. Institute of Neuroscience, Shanghai Institutes for Biological Sciences, Chinese Academy of Sciences, Shanghai, China. Verification of αB-crystallin and Hapln2 expression in brain of patients with Parkinson's disease.

Pharmaceutical companies

Asterand UK Ltd.

Analysis of protein expression in normal and Alzheimer's Disease human brain.

Examination of autoradiographic binding of a proprietary radioligand in frozen sections of different brain regions from normal donors and those with Parkinson's Disease.

Expression of therapeutic candidate gene in cerebellum from Type 2 Diabetic and control donors.

AstraZeneca

Identification and validation of transcripts and proteins in brain tissue derived from Alzheimer's patients with special emphasis on pharmacogenetics.

Bayer Schering Pharma AG

Interrelationship of activated microglia & reactive astrocytes in AD.

Characterization of alpha-synuclein binding molecules.

Characterization of small molecules binding to dementia related pathological targets.

BioFocus DPI BV

Collaborative research program with CHDI Foundation (Cure Huntington's Disease Initiative).

Evotec AG

Identification of small molecules binding to aggregated huntingtin for the development of a PET-ligand.

GlaxoSmithKline

Identification of potential therapeutic targets for amyotrophic lateral sclerosis and Huntington's disease.

Identification and validation of potential therapeutic targets for multiple sclerosis.

London Genetics Limited

Immunohistochemisty studies in cadaver control (normal) human dorsal root ganglia.

Merck Serono SA

Inflammation drives axonal pathology in the grey matter of neurodegenerative diseases.

Neurimmune Therapeutics AG

Characterization of therapeutic antibody candidates with respect to binding of pathological protein deposits, in Alzheimer's disease, Huntington's disease and amyotrophic lateral sclerosis.

Neusentis (a Pfizer Ltd. research unit)

Determining expression of pain-related ion channels in human lumbar spinal cord.

Novartis AG

Autoradiographic examination of extra- and intracellular markers in Huntington's disease, with special emphasis on visualization of huntingtin aggregates.

Autoradiography studies on orexin receptors in the human brain.

Pfizer Ltd.

Target characterisation and safety de-risking.

Prosensa Theurapeutics BV

Polyclonal antibody screening for the detection of the brain dystrophin isoform in muscle of patients with Duchenne Muscular Dystrophy.

Publications 2008-2012

In recent years many articles have been published that report the results of research projects realized with the use of NBB tissue. Table 6 shows the number of publications (2004- September 2012) for which NBB-tissue was used, for different journal impact factors. The impact factor of a scientific journal indicates how often its recently published articles have been cited, on average.

Table 6 Number of publications resulting from the use of NBB tissue, by impact factor

Impact factor	Publications	Publications by NIN research groups
Over 9	56	27 (48%)
7 - 9	52	12 (19%)
Under 7	362	128 (35%)
All journals	*470*	*167 (36%)*

The following publications were realized through the use of NBB tissue

't Hart, B.A., R.Q. Hintzen, and J.D. Laman. Multiple Sclerosis - a Response-to-damage Model. Trends Mol.Med. 15.6 (2009): 235–244.

Abildayeva, K. et al. Human Apolipoprotein CI Expression in Mice Impairs Learning and Memory Functions. Journal of lipid research 49.4 (2008): 856–869.

Alafuzoff, I., L. Parkkinen, et al. Assessment of Alpha-synuclein Pathology: a Study of the BrainNet Europe Consortium. J.Neuropathol.Exp.Neurol. 67.2 (2008): 125–143.

Alafuzoff, I., D.R. Thal, et al. Assessment of Beta-amyloid Deposits in Human Brain: a Study of the BrainNet Europe Consortium. Acta Neuropathol. 117.3 (2009): 309–320.

Alafuzoff, I., M. Pikkarainen, et al. Inter-laboratory Comparison of Neuropathological Assessments of Beta-amyloid Protein: a Study of the BrainNet Europe Consortium. Acta Neuropathol. 115.5 (2008): 533–546.

Alafuzoff, I., T. Arzberger, et al. Staging of Neurofibrillary Pathology in Alzheimer's Disease: a Study of the BrainNet Europe Consortium. Brain Pathol. 18.4 (2008): 484–496.

Alafuzoff, I., P.G. Ince, et al. Staging/typing of Lewy Body Related Alpha-synuclein Pathology: a Study of the BrainNet Europe Consortium. Acta Neuropathol. 117.6 (2009): 635–652.

Alberio, T. et al. Proteomic Analysis of Dopamine and A-synuclein Interplay in a Cellular Model of Parkinson's Disease Pathogenesis. FEBS Journal 277.23 (2010): 4909–4919.

Alkemade, A., C.X. Yi, et al. AgRP and NPY Expression in the Human Hypothalamic Infundibular Nucleus Correlate with Body Mass Index, Whereas Changes in alphaMSH Are Related to Type 2 Diabetes. J.Clin.Endocrinol.Metab 97.6 (2012): E925–E933.

Alkemade, A., E.C. Friesema, et al. Expression of Thyroid Hormone Transporters in the Human Hypothalamus. J.Clin.Endocrinol.Metab 96.6 (2011): E967–E971.

Alkemade, A., U.A. Unmehopa, et al. Suppressor of Cytokine Signaling 3 in the Human Hypothalamus. Peptides 35.1 (2012): 139–142.

Alt, S.R. et al. Differential Expression of Glucocorticoid Receptor Transcripts in Major Depressive Disorder Is Not Epigenetically Programmed. Psychoneuroendocrinology 35.4 (2010): 544–556.

Amadoro, G., V. Corsetti, A. Stringaro, et al. A NH2 Tau Fragment Targets Neuronal Mitochondria at AD Synapses: Possible Implications for Neurodegeneration. J.Alzheimers. Dis. 21.2 (2010): 445–470.

Amadoro, G., V. Corsetti, A. Atlante, et al. Interaction Between NH(2)-tau Fragment and Abeta in Alzheimer's Disease Mitochondria Contributes to the Synaptic Deterioration. Neurobiol.Aging 33.4 (2012): 833.e1 – e25.

Amor, S. et al. Inflammation in Neurodegenerative Diseases. Immunology 129.2 (2010): 154–169.

Anand, U., W.R. Otto, D. Sanchez-Herrera, et al. Cannabinoid Receptor CB2 Localisation and Agonist-mediated Inhibition of Capsaicin Responses in Human Sensory Neurons. Pain 138.3 (2008): 667–680.

Anand, U., W.R. Otto, P. Facer, et al. TRPA1 Receptor Localisation in the Human Peripheral Nervous System and Functional Studies in Cultured Human and Rat Sensory Neurons. Neurosci.Lett. 438.2 (2008): 221–227.

Andreyeva, A. et al. C-terminal Fragment of N-cadherin Accelerates Synapse Destabilization by Amyloid-beta. Brain 135.7 (2012): 2140–2154.

Aziz, A. et al. Hypocretin and Melanin-concentrating Hormone in Patients with Huntington Disease. Brain Pathol. 18.4 (2008): 474–483.

Baillie, J.K. et al. Somatic Retrotransposition Alters the Genetic Landscape of the Human Brain. Nature 479.7374 (2011): 534–537.

Bao, F. et al. Different Beta-amyloid Oligomer Assemblies in Alzheimer Brains Correlate with Age of Disease Onset and Impaired Cholinergic Activity. Neurobiol.Aging 33.4 (2012): 825.e1–e13.

Basso, M. et al. Characterization of Detergent-insoluble Proteins in ALS Indicates a Causal Link Between Nitrative Stress and Aggregation in Pathogenesis. PLoS.One. 4.12 (2009): e8130.

Bauer, N.M. et al. Myelin Basic Protein Synthesis Is Regulated by Small Non-coding RNA 715. EMBO Rep. 13.9 (2012): 827–834.

Bell, J.E. et al. Management of a Twenty-first Century Brain Bank: Experience in the BrainNet Europe Consortium. Acta Neuropathol. 115.5 (2008): 497–507.

Berrocal, M. et al. Altered Ca2+ Dependence of Synaptosomal Plasma Membrane Ca2+-ATPase in Human Brain Affected by Alzheimer's Disease. FASEB J. 23.6 (2009): 1826–1834.

Berson, A. et al. Changes in Readthrough Acetylcholinesterase Expression Modulate Amyloid-beta Pathology. Brain 131.1 (2008): 109–119.

Berson, Amit et al. Cholinergic-associated Loss of hnRNP-A/B in Alzheimer's Disease Impairs Cortical Splicing and Cognitive Function in Mice. EMBO Molecular Medicine 4.8 (2012): 730–742.

Beyer, N., D.T. Coulson, S. Heggarty, R. Ravid, J. Hellemans, et al. Zinc Transporter mRNA Levels in Alzheimer's Disease Postmortem Brain. J.Alzheimers.Dis. 29.4 (2012): 863–873.

Beyer, N., D.T. Coulson, S. Heggarty, R. Ravid, G.B. Irvine, et al. ZnT3 mRNA Levels Are Reduced in Alzheimer's Disease Post-mortem Brain. Mol.Neurodegener. 4.1 (2009): 53.

Bharathi, and K.S. Rao. Molecular Understanding of Copper and Iron Interaction with Alpha-synuclein by Fluorescence Analysis. J.Mol.Neurosci. 35.3 (2008): 273–281.

Bien, J. et al. The Metalloprotease Meprin Beta Generates Amino Terminal-truncated Amyloid Beta Peptide Species. J.Biol.Chem. 287.40 (2012): 33304–33313.

Booij, J.C. et al. The Dynamic Nature of Bruch's Membrane. Prog.Retin.Eye Res. 29.1 (2010): 1–18.

Borgers, A.J. et al. Distribution of Serotonin Transporters in the Human Hypothalamus. Endocrine Abstracts. Vol. 26 OC2.3. Rotterdam, The Netherlands, 2011.

Bosman, Sjanne et al. Clinicopathological Correlations of the Frontal Lobe Syndrome: Results of a Large Brain Bank Study. Alzheimer's Association International Conference 2012 8.4, Supplement (2012): P623.

Bossers, K., G. Meerhoff, et al. Analysis of Gene Expression in Parkinson's Disease: Possible Involvement of Neurotrophic Support and Axon Guidance in Dopaminergic Cell Death. Brain Pathol. 19.1 (2009): 91–107.

Bossers, K., K.T. Wirz, et al. Concerted Changes in Transcripts in the Prefrontal Cortex Precede Neuropathology in Alzheimer's Disease. Brain 133.12 (2010): 3699–3723.

Bossers, K., B. Ylstra, et al. Intensity-based Analysis of Dual-color Gene Expression Data as an Alternative to Ratio-based Analysis to Enhance Reproducibility. BMC.Genomics 11.1 (2010): 112.

Bradbury, Margaret et al. False Reassurance Following Genetic Susceptibility Testing for Alzheimer's Disease: Evidence from the REVEAL Study. Alzheimer's & dementia : the journal of the Alzheimer's Association 7.4 (2011): S179–S180.

Breij, E.C. et al. Homogeneity of Active Demyelinating Lesions in Established Multiple Sclerosis. Ann.Neurol. 63.1 (2008): 16–25.

Brockschnieder, D. et al. Preclinical Characterization of a Novel Class of 18F-Labeled PET Tracers for Amyloid-beta. J.Nucl.Med. 53.11 (2012): 1794–1801.

Bronner, I.F. et al. Comprehensive mRNA Expression Profiling Distinguishes Tauopathies and Identifies Shared Molecular Pathways. PLoS.One. 4.8 (2009): e6826.

Broux, B. et al. CX(3)CR1 Drives Cytotoxic CD4(+)CD28(-) T Cells into the Brain of Multiple Sclerosis Patients. J.Autoimmun. 38.1 (2012): 10–19.

Bruno, M.A. et al. Amyloid Beta-induced Nerve Growth Factor Dysmetabolism in Alzheimer Disease. J.Neuropathol.Exp.Neurol. 68.8 (2009): 857–869.

Bsibsi, M. et al. The Microtubule Regulator Stathmin Is an Endogenous Protein Agonist for TLR3. J.Immunol. 184.12 (2010): 6929–6937.

Bugiani, M. et al. Defective Glial Maturation in Vanishing White Matter Disease. J.Neuropathol. Exp.Neurol. 70.1 (2011): 69–82.

Buijs, R.M. et al. Spleen Vagal Denervation Inhibits the Production of Antibodies to Circulating Antigens. PLoS.One. 3.9 (2008): e3152.

Caglayan, S., A. Bauerfeind, et al. Identification of Alzheimer Disease Risk Genotype That Predicts Efficiency of SORL1 Expression in the Brain. Archives of neurology 69.3 (2012): 373.

Caglayan, Safak, Vanessa Schmidt, et al. Identification of Alzheimer's Disease Haplotype That Predicts Efficiency of SORL1/SORLA Expression in the Brain. Alzheimer's & Dementia 7.4, Supplement (2011): S180.

Cao, Y. et al. Changed Clathrin Regulatory Proteins in the Brains of Alzheimer's Disease Patients and Animal Models. J.Alzheimers.Dis. 22.1 (2010): 329–342.

Carrano, A. et al. Neuroinflammation and Blood-brain Barrier Changes in Capillary Amyloid Angiopathy. Neurodegener.Dis. 10.1-4 (2012): 329–331.

Carrano, A. et al. Amyloid Beta Induces Oxidative Stress-mediated Blood–brain Barrier Changes in Capillary Amyloid Angiopathy. Antioxidants & redox signaling 15.5 (2011): 1167–1178.

Chen, X.N. et al. The Involvement of Retinoic Acid Receptor-alpha in Corticotropin-releasing Hormone Gene Expression and Affective Disorders. Biol.Psychiatry 66.9 (2009): 832–839.

Chen-Plotkin, A.S. et al. Genetic and Clinical Features of Progranulin-associated Frontotemporal Lobar Degeneration. Archives of neurology 68.4 (2011): 488.

Christensen, D.Z. et al. Accumulation of Intraneuronal Aβ Correlates with ApoE4 Genotype. Acta neuropathologica 119.5 (2010): 555–566.

Clarner, T. et al. Myelin Debris Regulates Inflammatory Responses in an Experimental Demyelination Animal Model and Multiple Sclerosis Lesions. Glia 60.10 (2012): 1468–1480.

Cogswell, J.P. et al. Identification of miRNA Changes in Alzheimer's Disease Brain and CSF Yields Putative Biomarkers and Insights into Disease Pathways. J.Alzheimers.Dis. 14.1 (2008): 27–41.

Colsch, B. et al. Sulfogalactosylceramides in Motor and Psycho-cognitive Adult Metachromatic Leukodystrophy: Relations Between Clinical, Biochemical Analysis and Molecular Aspects. Biochim.Biophys.Acta 1780.3 (2008): 434–440.

Corneveaux, J.J. et al. Association of CR1, CLU and PICALM with Alzheimer's Disease in a Cohort of Clinically Characterized and Neuropathologically Verified Individuals. Hum. Mol.Genet. 19.16 (2010): 3295–3301.

Coulson, D.T., N. Beyer, et al. BACE1 mRNA Expression in Alzheimer's Disease Postmortem Brain Tissue. J.Alzheimers.Dis. 22.4 (2010): 1111–1122.

Coulson, D.T., S. Brockbank, et al. Identification of Valid Reference Genes for the Normalization of RT qPCR Gene Expression Data in Human Brain Tissue. BMC.Mol.Biol. 9.1 (2008): 46.

Couturier, N. et al. Mast Cell Transcripts Are Increased Within and Outside Multiple Sclerosis Lesions. J.Neuroimmunol. 195.1 (2008): 176–185.

Cuello, A.C., M.T. Ferretti, and M.F. Iulita. Preplaque ('preclinical') Abeta-induced Inflammation and Nerve Growth Factor Deregulation in Transgenic Models of Alzheimer's Disease-like Amyloid Pathology. Neurodegener.Dis. 10.1-4 (2012): 104–107.

De Jager, M. et al. Tissue Transglutaminase Colocalizes with Extracellular Matrix Proteins in Cerebral Amyloid Angiopathy. Neurobiol.Aging 1558-1497 (Electronic) (2012): n. pag.

De Kimpe, L., A. Bennis, et al. Disturbed Ca2+ Homeostasis Increases Glutaminyl Cyclase Expression; Connecting Two Early Pathogenic Events in Alzheimer's Disease In Vitro. PloS one 7.9 (2012): e44674.

De Kimpe, L., E.S. Van Haastert, et al. Intracellular Accumulation of Aggregated Pyroglutamate Amyloid Beta: Convergence of Aging and Aβ Pathology at the Lysosome. Age (2012): 1–15.

Deleersnijder, A. et al. Comparative Analysis of Different Peptidyl-prolyl Isomerases Reveals FK506-binding Protein 12 as the Most Potent Enhancer of A-synuclein Aggregation. Journal of Biological Chemistry 286.30 (2011): 26687–26701.

Dennissen, F.J. et al. Mutant Ubiquitin (UBB+1) Associated with Neurodegenerative Disorders Is Hydrolyzed by Ubiquitin C-terminal Hydrolase L3 (UCH-L3). FEBS Lett. 585.16 (2011): 2568–2574.

Dennissen, F.J., N. Kholod, and F.W. van Leeuwen. The Ubiquitin Proteasome System in Neurodegenerative Diseases: Culprit, Accomplice or Victim? Prog.Neurobiol. 96.2 (2012): 190–207.

Diaz-Hernandez, M. et al. Tissue-nonspecific Alkaline Phosphatase Promotes the Neurotoxicity Effect of Extracellular Tau. J.Biol.Chem. 285.42 (2010): 32539–32548.

Domercq, M. et al. Dual-specific Phosphatase-6 (Dusp6) and ERK Mediate AMPA Receptor-induced Oligodendrocyte Death. Journal of Biological Chemistry 286.13 (2011): 11825–11836.

Doorn, K.J. et al. Emerging Roles of Microglial Activation and Non-motor Symptoms in Parkinson's Disease. Progress in Neurobiology 98.2 (2012): 222–238.

Doorn, R. et al. Fingolimod Attenuates Ceramide-induced Blood–brain Barrier Dysfunction in Multiple Sclerosis by Targeting Reactive Astrocytes. Acta Neuropathologica 124.3 (2012): 397–410.

Dopper, E.G.P. et al. Symmetrical Corticobasal Syndrome Caused by a Novel C. 314dup Progranulin Mutation. Journal of Molecular Neuroscience 45.3 (2011): 354–358.

Duan, X.H. et al. Novel Anilinophthalimide Derivatives as Potential Probes for Beta-amyloid Plaque in the Brain. Bioorg.Med.Chem. 18.3 (2010): 1337–1343.

Durrenberger, P.F. et al. Effects of Antemortem and Postmortem Variables on Human Brain mRNA Quality: a BrainNet Europe Study. J.Neuropathol.Exp.Neurol. 69.1 (2010): 70–81.

Echavarri, C. et al. Neuropsychiatric Symptoms in Alzheimer's Disease and Vascular Dementia. J.Alzheimers.Dis. 33.3 (2012): 715–721.

Eikelenboom, P. et al. Neuroinflammation in Plaque and Vascular Beta-amyloid Disorders: Clinical and Therapeutic Implications. Neurodegener.Dis. 5.3-4 (2008): 190–193.

Eisele, S. et al. Prospects of Transcript Profiling for mRNAs and MicroRNAs Using Formalin-Fixed and Paraffin-Embedded Dissected Autoptic Multiple Sclerosis Lesions. Brain Pathol. 22.5 (2012): 607–618.

Elliott, E., O. Laufer, and I. Ginzburg. BAG-1M Is Up-regulated in Hippocampus of Alzheimer's Disease Patients and Associates with Tau and APP Proteins. J.Neurochem. 109.4 (2009): 1168–1178.

Farkas, S., K. Nagy, Z. Jia, T. Hortobagyi, et al. Signal Transduction Pathway Activity Compensates Dopamine D(2)/D(3) Receptor Density Changes in Parkinson's Disease: a Preliminary Comparative Human Brain Receptor Autoradiography Study with [(3)H]raclopride and [(35)S]GTPgammaS. Brain Res. 1453.1872-6240 (Electronic) (2012): 56–63.

Farkas, S., K. Nagy, Z. Jia, T. Harkany, et al. The Decrease of Dopamine D(2)/D(3) Receptor Densities in the Putamen and Nucleus Caudatus Goes Parallel with Maintained Levels of CB(1) Cannabinoid Receptors in Parkinson's Disease: a Preliminary Autoradiographic Study with the Selective Dopamine D(2)/D(3) Antagonist [(3)H]raclopride and the Novel CB(1) Inverse Agonist [(1)(2)(5)I]SD7015. Brain Res.Bull. 87.6 (2012): 504–510.

Fazio, F. et al. Switch in the Expression of mGlu1 and mGlu5 Metabotropic Glutamate Receptors in the Cerebellum of Mice Developing Experimental Autoimmune Encephalomyelitis and in Autoptic Cerebellar Samples from Patients with Multiple Sclerosis. Neuropharmacology 55.4 (2008): 491–499.

Friesema, E.C. et al. Thyroid Hormone Transporters and Deiodinases in the Developing Human Hypothalamus. Eur.J.Endocrinol. 167.3 (2012): 379–386.

Fronczek, R. et al. Hypocretin (orexin) Loss in Alzheimer's Disease. Neurobiol.Aging 33.8 (2011): 1642–1650.

Fu, Xing et al. Rapid Metabolic Evolution in Human Prefrontal Cortex. Proceedings of the National Academy of Sciences 108.15 (2011): 6181–6186.

Gahete, M.D., A. Rubio, M. Duran-Prado, et al. Expression of Somatostatin, Cortistatin, and Their Receptors, as Well as Dopamine Receptors, but Not of Neprilysin, Are Reduced in the Temporal Lobe of Alzheimer's Disease Patients. J.Alzheimers.Dis. 20.2 (2010): 465–475.

Gahete, M.D., A. Rubio, J. Cordoba-Chacon, et al. Expression of the Ghrelin and Neurotensin Systems Is Altered in the Temporal Lobe of Alzheimer's Disease Patients. J.Alzheimers. Dis. 22.3 (2010): 819–828.

Gao, S.F. et al. Decreased NOS1 Expression in the Anterior Cingulate Cortex in Depression. Cereb.Cortex 1460-2199 (Electronic) (2012): n. pag.

Garcia-Falgueras, A. et al. Galanin Neurons in the Intermediate Nucleus (InM) of the Human Hypothalamus in Relation to Sex, Age, and Gender Identity. J.Comp Neurol. 519.15 (2011): 3061–3084.

Garcia-Falgueras, A., and D.F. Swaab. A Sex Difference in the Hypothalamic Uncinate Nucleus: Relationship to Gender Identity. Brain 131.12 (2008): 3132–3146.

Garcia-Falgueras, A., and D.F. Swaab. Sexual Hormones and the Brain: An Essential Alliance for Sexual Identity and Sexual Orientation. Endocr.Dev. 17.1662-2979 (Electronic) (2010): 22–35.

Ge, J.F. et al. The Binding of Resveratrol to Monomer and Fibril Amyloid Beta. Neurochem. Int. 1872-9754 (Electronic) (2012): n. pag.

Geurts, J.J., E.L. Blezer, et al. Does High-field MR Imaging Improve Cortical Lesion Detection in Multiple Sclerosis? J.Neurol. 255.2 (2008): 183–191.

Geurts, J.J., E.J. Kooi, et al. Multiple Sclerosis as an 'Inside-out' Disease. Ann.Neurol. 68.5 (2010): 767–768.

Geurts, J.J., and F. Barkhof. Grey Matter Pathology in Multiple Sclerosis. Lancet Neurol. 7.9 (2008): 841–851.

Giaccone, G. et al. New Lexicon and Criteria for the Diagnosis of Alzheimer's Disease. Lancet Neurol. 10.4 (2011): 298–299.

Goto-Inoue, N. et al. A New Lipidomics Approach by Thin-layer Chromatography-blot-matrix-assisted Laser Desorption/ionization Imaging Mass Spectrometry for Analyzing Detailed Patterns of Phospholipid Molecular Species. J.Chromatogr.A 1216.42 (2009): 7096–7101.

Gouw, A.A. et al. Heterogeneity of White Matter Hyperintensities in Alzheimer's Disease: Post-mortem Quantitative MRI and Neuropathology. Brain 131.12 (2008): 3286–3298.

Govarts, C. et al. Analysis of Antibody Reactivity in Paired Cerebrospinal Fluid and Serum of a Relapsing Remitting Multiple Sclerosis Patient. Autoimmunity 42.8 (2009): 699–704.

Gulyas, B. et al. The Norepinephrine Transporter (NET) Radioligand (S,S)-[18F]FMeNER-D2 Shows Significant Decreases in NET Density in the Human Brain in Alzheimer's Disease: a Post-mortem Autoradiographic Study. Neurochem.Int. 56.6 (2010): 789–798.

Gupta, V.B., S.S. Indi, and K.S. Rao. Studies on the Role of Amino Acid Stereospecificity in Amyloid Beta Aggregation. J.Mol.Neurosci. 34.1 (2008): 35–43.

Ha, T.Y. et al. S100a9 Knockdown Decreases the Memory Impairment and the Neuropathology in Tg2576 Mice, AD Animal Model. PLoS.One. 5.1 (2010): e8840.

Harold, D. et al. Genome-wide Association Study Identifies Variants at CLU and PICALM Associated with Alzheimer's Disease. Nat.Genet. 41.10 (2009): 1088–1093.

Hashimoto, T., and M. Nakai. Increased Hippocampal Quinone Reductase 2 in Alzheimer's Disease. Neurosci.Lett. 502.1 (2011): 10–12.

Heidbrink, C. et al. Reduced Cortisol Levels in Cerebrospinal Fluid and Differential Distribution of 11β-hydroxysteroid Dehydrogenases in Multiple Sclerosis: Implications for Lesion Pathogenesis. Brain, behavior, and immunity 24.6 (2010): 975–984.

Hellstrom-Lindahl, E., R. Ravid, and A. Nordberg. Age-dependent Decline of Neprilysin in Alzheimer's Disease and Normal Brain: Inverse Correlation with A Beta Levels. Neurobiol.Aging 29.2 (2008): 210–221.

Hellstrom-Lindahl, E., M. Viitanen, and A. Marutle. Comparison of Abeta Levels in the Brain of Familial and Sporadic Alzheimer's Disease. Neurochem.Int. 55.4 (2009): 243–252.

Hendrickx, D et al. Myelin phagocytosis in multiple sclerosis. Glia 59.S1 (2011): S60–S61.

Hendrickx, D. et al. Myelin Phagocytosis in Multiple Sclerosis. Multiple Sclerosis Journal 17.10S (2011): S354–S354.

Hoogerhout, P. et al. A Cyclic Undecamer Peptide Mimics a Turn in Folded Alzheimer Amyloid Beta and Elicits Antibodies Against Oligomeric and Fibrillar Amyloid and Plaques. PLoS.One. 6.4 (2011): e19110.

Hoozemans, J.J., J.M. Rozemuller, et al. Cyclooxygenase-1 and -2 in the Different Stages of Alzheimer's Disease Pathology. Curr.Pharm.Des 14.14 (2008): 1419–1427.

Hoozemans, J.J., A.J. Rozemuller, et al. Neuroinflammation in Alzheimer's Disease Wanes with Age. J.Neuroinflammation. 8.1742-2094 (Electronic) (2011): 171.

Hoozemans, J.J., E.S. van Haastert, et al. The Unfolded Protein Response Is Activated in Pretangle Neurons in Alzheimer's Disease Hippocampus. Am.J.Pathol. 174.4 (2009): 1241–1251.

Hortobágyi, T. et al. Optineurin Inclusions Occur in a Minority of TDP-43 Positive ALS and FTLD-TDP Cases and Are Rarely Observed in Other Neurodegenerative Disorders. Acta neuropathologica 121.4 (2011): 519–527.

Hu, H.Y., L. He, et al. Evolution of the Human-specific microRNA miR-941. Nat Commun 3 (2012): 1145.

Hu, H.Y., S. Guo, et al. MicroRNA Expression and Regulation in Human, Chimpanzee, and Macaque Brains. PLoS genetics 7.10 (2011): e1002327.

Huisman, E., H.B. Uylings, and P.V. Hoogland. Gender-related Changes in Increase of Dopaminergic Neurons in the Olfactory Bulb of Parkinson's Disease Patients. Mov Disord. 23.10 (2008): 1407–1413.

Huizinga, R., K. Kreft, et al. Endotoxin- and ATP-neutralizing Activity of Alkaline Phosphatase as a Strategy to Limit Neuroinflammation. Journal of Neuroinflammation 9.1 (2012): 266.

Huizinga, R., B.J. van der Star, et al. Phagocytosis of Neuronal Debris by Microglia Is Associated with Neuronal Damage in Multiple Sclerosis. Glia 60.3 (2012): 422–431.

Ikemoto, K. Are D-Neurons and Trace Amine-Associated Receptor, Type 1 Involved in Mesolimbic Dopamine Hyperactivity of Schizophrenia? Medicinal Chemistry (2012).

Ikemoto, K. Why D-neuron? Direction from Psychiatric Research. J Neurol Neurophysiol 10.S11 (2012): 2155–9562.

Ikemoto, K. Striatal D-neurons: In New Viewpoints for Neuropsychiatric Research Using Post-mortem Brains. Fukushima J.Med.Sci. 54.1 (2008): 1–3.

Ikemoto, K. Why D-neuron? Importance in Schizophrenia Research. Open Journal of Psychiatry 2.4 (2012): 393–398.

Ishunina, T.A., and D.F. Swaab. Age-dependent ERalpha MB1 Splice Variant Expression in Discrete Areas of the Human Brain. Neurobiol.Aging 29.8 (2008): 1177–1189.

Ishunina, T.A., and D.F. Swaab. Estrogen Receptor-alpha Splice Variants in the Human Brain. Gynecol.Endocrinol. 24.2 (2008): 93–98.

Ishunina, T.A., and D.F. Swaab. Hippocampal Estrogen Receptor-alpha Splice Variant TADDI in the Human Brain in Aging and Alzheimer's Disease. Neuroendocrinology 89.2 (2009): 187–199.

Ishunina, T.A., and D.F. Swaab. Decreased Alternative Splicing of Estrogen Receptor-α mRNA in the Alzheimer's Disease Brain. Neurobiology of Aging 33.2 (2012): 286–296.e3.

Jansen, C., P. Parchi, S. Capellari, R. Strammiello, et al. A Second Case of Gerstmann-Straussler-Scheinker Disease Linked to the G131V Mutation in the Prion Protein Gene in a Dutch Patient. J.Neuropathol.Exp.Neurol. 70.8 (2011): 698–702.

Jansen, C., P. Parchi, S. Capellari, A.J. Vermeij, et al. Prion Protein Amyloidosis with Divergent Phenotype Associated with Two Novel Nonsense Mutations in PRNP. Acta Neuropathol. 119.2 (2010): 189–197.

Jefferson, T. et al. Metalloprotease Meprin Beta Generates Nontoxic N-terminal Amyloid Precursor Protein Fragments in Vivo. J.Biol.Chem. 286.31 (2011): 27741–27750.

Johnson, A.E. et al. AZD2184: a Radioligand for Sensitive Detection of B-amyloid Deposits. Journal of Neurochemistry 108.5 (2009): 1177–1186.

Jun, G. et al. Comprehensive Search for Alzheimer Disease Susceptibility Loci in the APOE Region. Archives of neurology 69.10 (2012): 1270.

Junker, A. et al. MicroRNA Profiling of Multiple Sclerosis Lesions Identifies Modulators of the Regulatory Protein CD47. Brain 132.12 (2009): 3342–3352.

Juréus, A. et al. Characterization of AZD4694, a Novel Fluorinated Aβ Plaque Neuroimaging PET Radioligand. Journal of Neurochemistry 114.3 (2010): 784–794.

Kalsbeek, A. et al. Vasopressin and the Output of the Hypothalamic Biological Clock. J.Neuroendocrinol. 22.5 (2010): 362–372.

Kan, A.A, S. van Erp, et al. Genome-wide microRNA Profiling of Human Temporal Lobe Epilepsy Identifies Modulators of the Immune Response. Cellular and Molecular Life Sciences 69.18 (2012): 3127–3145.

Kan, A.A, W. de Jager, et al. Protein Expression Profiling of Inflammatory Mediators in Human Temporal Lobe Epilepsy Reveals Co-activation of Multiple Chemokines and Cytokines. Journal of neuroinflammation 9.1 (2012): 1–22.

Karlsen, A.S., and B. Pakkenberg. Total Numbers of Neurons and Glial Cells in Cortex and Basal Ganglia of Aged Brains with Down Syndrome--a Stereological Study. Cereb.Cortex 21.11 (2011): 2519–2524.

Kipp, M. et al. BLBP-expression in Astrocytes During Experimental Demyelination and in Human Multiple Sclerosis Lesions. Brain, Behavior, and Immunity 25.8 (2011): 1554–1568.

Klok, M.D. et al. Decreased Expression of Mineralocorticoid Receptor mRNA and Its Splice Variants in Postmortem Brain Regions of Patients with Major Depressive Disorder. J.Psychiatr.Res. 45.7 (2011): 871–878.

Koning, N., D.F. Swaab, et al. Distribution of the Immune Inhibitory Molecules CD200 and CD200R in the Normal Central Nervous System and Multiple Sclerosis Lesions Suggests Neuron-glia and Glia-glia Interactions. J.Neuropathol.Exp.Neurol. 68.2 (2009): 159–167.

Koning, N., Eijk M. van, et al. Expression of the Inhibitory CD200 Receptor Is Associated with Alternative Macrophage Activation. J.Innate.Immun. 2.2 (2010): 195–200.

Koning, N., B.M. Uitdehaag, et al. Restoring Immune Suppression in the Multiple Sclerosis Brain. Prog.Neurobiol. 89.4 (2009): 359–368.

Kooi, E.J., Horssen J. van, et al. Abundant Extracellular Myelin in the Meninges of Patients with Multiple Sclerosis. Neuropathol.Appl.Neurobiol. 35.3 (2009): 283–295.

Kooi, E.J., E.M. Strijbis, et al. Heterogeneity of Cortical Lesions in Multiple Sclerosis: Clinical and Pathologic Implications. Neurology 79.13 (2012): 1369–1376.

Kooi, E.J., J.J. Geurts, et al. Meningeal Inflammation Is Not Associated with Cortical Demyelination in Chronic Multiple Sclerosis. J.Neuropathol.Exp.Neurol. 68.9 (2009): 1021–1028.

Kooij, G., M.R. Mizee, et al. Adenosine Triphosphate-binding Cassette Transporters Mediate Chemokine (CC Motif) Ligand 2 Secretion from Reactive Astrocytes: Relevance to Multiple Sclerosis Pathogenesis. Brain 134.2 (2011): 555–570.

Kooij, G., J. van Horssen, et al. T Lymphocytes Impair P-glycoprotein Function During Neuroinflammation. Journal of autoimmunity 34.4 (2010): 416–425.

Kreft, K.L. et al. The IL-7Ralpha Pathway Is Quantitatively and Functionally Altered in CD8 T Cells in Multiple Sclerosis. J.Immunol. 188.4 (2012): 1874–1883.

Kretzschmar, H. Brain Banking: Opportunities, Challenges and Meaning for the Future. Nat. Rev.Neurosci. 10.1 (2008): 70–78.

Kuipers, H.F. et al. CC Chemokine Receptor 5 Gene Promoter Activation by the Cyclic AMP Response Element Binding Transcription Factor. Blood 112.5 (2008): 1610–1619.

Lewis, S. Neurodegenerative Disorders: Microglia Get Ready, Set... Nature Reviews Neuroscience 13.3 (2012): 154–155.

Lilja, A.M. et al. Functional Interactions of Fibrillar and Oligomeric Amyloid-beta with Alpha7 Nicotinic Receptors in Alzheimer's Disease. J.Alzheimers.Dis. 23.2 (2011): 335–347.

Liu, C-Q. et al. A Quantitative in Situ Hybridization Protocol for Formalin-fixed Paraffin-embedded Archival Post-mortem Human Brain Tissue. Methods 52.4 (2010): 359–366.

Liu, X. et al. Extension of Cortical Synaptic Development Distinguishes Humans from Chimpanzees and Macaques. Genome research 22.4 (2012): 611–622.

Lucassen, P.J. The Origin and Development of Plaques and Phosphorylated Tau Are Associated with Axonopathy in Alzheimer's Disease. Neuroscience bulletin 27.5 (2011): 287–299.

Lucassen, P.J. et al. Decreased Numbers of Progenitor Cells but No Response to Antidepressant Drugs in the Hippocampus of Elderly Depressed Patients. Drugs and Stem Cells in Brain Repair 58.6 (2010): 940–949.

Luchetti, S., K. Bossers, G.V. Frajese, et al. Neurosteroid Biosynthetic Pathway Changes in Substantia Nigra and Caudate Nucleus in Parkinson's Disease. Brain Pathol. 20.5 (2010): 945–951.

Luchetti, S., K. Bossers, S. Van de Bilt, et al. Neurosteroid Biosynthetic Pathways Changes in Prefrontal Cortex in Alzheimer's Disease. Neurobiol.Aging 32.11 (2009): 1964–1976.

Luchetti, S., I. Huitinga, and D.F. Swaab. Neurosteroid and GABA-A Receptor Alterations in Alzheimer's Disease, Parkinson's Disease and Multiple Sclerosis. Neuroscience 191.1873-7544 (Electronic) (2011): 6–21.

Lund, H. et al. Tau-tubulin Kinase 1 Expression, Phosphorylation and Co-localization with phospho-Ser422 Tau in the Alzheimer's Disease Brain. Brain Pathol. 1750-3639 (Electronic) (2012): n. pag.

Lutz, M.W. et al. Genetic Variation at a Single Locus and Age of Onset for Alzheimer's Disease. Alzheimers.Dement. 6.2 (2010): 125–131.

Mackenzie, I.R. et al. Nomenclature and Nosology for Neuropathologic Subtypes of Frontotemporal Lobar Degeneration: An Update. Acta Neuropathol. 119.1 (2010): 1–4.

Mackenzie, I.R. et al. Nomenclature for Neuropathologic Subtypes of Frontotemporal Lobar Degeneration: Consensus Recommendations. Acta Neuropathol. 117.1 (2009): 15–18.

Makris, Nikos et al. Volumetric Parcellation Methodology of the Human Hypothalamus in Neuroimaging: Normative Data and Sex Differences. NeuroImage (2012): n. pag.

Mali, Y., and N. Zisapels. Gain of Interaction of ALS-linked G93A Superoxide Dismutase with Cytosolic Malate Dehydrogenase. Neurobiol.Dis. 32.1 (2008): 133–141.

Marcello, E. et al. SAP97-mediated Local Trafficking Is Altered in Alzheimer Disease Patients' Hippocampus. Neurobiol.Aging 33.2 (2010): 422.e1–e10.

Matute, C. P2X7 Receptors in Oligodendrocytes: a Novel Target for Neuroprotection. Mol. Neurobiol. 38.2 (2008): 123–128.

Maubach, K.A. et al. BGC20-1531, a Novel, Potent and Selective Prostanoid EP Receptor Antagonist: a Putative New Treatment for Migraine Headache. Br.J.Pharmacol. 156.2 (2009): 316–327.

Medhurst, A.D. et al. Characterization of Histamine H3 Receptors in Alzheimer's Disease Brain and Amyloid Over-expressing TASTPM Mice. Br.J.Pharmacol. 157.1 (2009): 130–138.

Meli, G. et al. Direct in Vivo Intracellular Selection of Conformation-sensitive Antibody Domains Targeting Alzheimer's Amyloid-beta Oligomers. J.Mol.Biol. 387.3 (2009): 584–606.

Melief, Jeroen et al. Phenotyping Primary Human Microglia: Tight Regulation of LPS Responsiveness. Glia 60.10 (2012): 1506–1517.

Mencarelli, C. et al. Goodpasture Antigen-binding Protein/ceramide Transporter Binds to Human Serum Amyloid P-component and Is Present in Brain Amyloid Plaques. Journal of Biological Chemistry 287.18 (2012): 14897–14911.

Meng, Q.Y. et al. Distribution of Retinoic Acid Receptor-alpha Immunoreactivity in the Human Hypothalamus. Neuroscience 174.1873-7544 (Electronic) (2011): 132–142.

Meynen, G. et al. Hypothalamic Vasopressin and Oxytocin mRNA Expression in Relation to Depressive State in Alzheimer's Disease: a Difference with Major Depressive Disorder. J.Neuroendocrinol. 21.8 (2009): 722–729.

Middeldorp, J. et al. Specific Human Astrocyte Subtype Revealed by Affinity Purified GFAP Antibody; Unpurified Serum Cross-reacts with neurofilament-L in Alzheimer. PLoS. One. 4.11 (2009): e7663.

Mizrak, S.C. et al. Embryonic Stem Cell-like Cells Derived from Adult Human Testis. Hum. Reprod. 25.1 (2010): 158–167.

Mohan, H. et al. Extracellular Matrix in Multiple Sclerosis Lesions: Fibrillar Collagens, Biglycan and Decorin Are Upregulated and Associated with Infiltrating Immune Cells. Brain Pathol. 20.5 (2010): 966–975.

Moloney, A.M. et al. Defects in IGF-1 Receptor, Insulin Receptor and IRS-1/2 in Alzheimer's Disease Indicate Possible Resistance to IGF-1 and Insulin Signalling. Neurobiology of Aging 31.2 (2010): 224–243.

Monoranu, C.M. et al. pH Measurement as Quality Control on Human Post Mortem Brain Tissue: a Study of the BrainNet Europe Consortium. Neuropathol.Appl.Neurobiol. 35.3 (2009): 329–337.

Mulder, S.D. et al. CSF Levels of PSA and PSA-ACT Complexes in Alzheimer's Disease. Ann. Clin.Biochem. 46.4 (2009): 477–483.

Mulder, S.D. et al. The Effect of Amyloid Associated Proteins on the Expression of Genes Involved in Amyloid-β Clearance by Adult Human Astrocytes. Special Issue: Stress and neurological disease 233.1 (2012): 373–379.

Mulder, S., and R. Veerhuis. Differences in Pathways Used for Beta-amyloid Uptake by Adult Human Astrocytes and Microglia? Alzheimer's Association International Conference 2012 Alzheimer's Association International Conference 2012 8.4, Supplement (2012): P642–P643.

Nabuurs, R.J.A. et al. High-field MRI of Single Histological Slices Using an Inductively Coupled, Self-resonant Microcoil: Application to Ex Vivo Samples of Patients with Alzheimer's Disease. NMR in Biomedicine 24.4 (2011): 351–357.

Nielsen, H.M., S.D. Mulder, et al. Astrocytic A Beta 1-42 Uptake Is Determined by A Beta-aggregation State and the Presence of Amyloid-associated Proteins. Glia 58.10 (2010): 1235–1246.

Nielsen, H.M., R. Veerhuis, et al. Binding and Uptake of A Beta1-42 by Primary Human Astrocytes in Vitro. Glia 57.9 (2009): 978–988.

Nijholt, D.A., T.R. de Graaf, et al. Endoplasmic Reticulum Stress Activates Autophagy but Not the Proteasome in Neuronal Cells: Implications for Alzheimer's Disease. Cell Death. Differ. 18.6 (2011): 1071–1081.

Nijholt, D.A., E.S. van Haastert, et al. The Unfolded Protein Response Is Associated with Early Tau Pathology in the Hippocampus of Tauopathies. J.Pathol. 226.5 (2012): 693–702.

O'Callaghan, P. et al. Heparan Sulfate Accumulation with Abeta Deposits in Alzheimer's Disease and Tg2576 Mice Is Contributed by Glial Cells. Brain Pathol. 18.4 (2008): 548–561.

O'Neill, C. et al. Insulin and IGF-1 Signalling: Longevity, Protein Homoeostasis and Alzheimer's Disease. Biochemical Society Transactions 40.4 (2012): 721.

Olah, M. et al. An Optimized Protocol for the Acute Isolation of Human Microglia from Autopsy Brain Samples. Glia 60.1 (2012): 96–111.

Ouwendijk, W.J., S.E. Flowerdew, et al. Immunohistochemical Detection of Intra-neuronal VZV Proteins in Snap-frozen Human Ganglia Is Confounded by Antibodies Directed Against Blood Group A1-associated Antigens. J.Neurovirol. 18.3 (2012): 172–180.

Ouwendijk, W.J., A. Choe, et al. Restricted Varicella-zoster Virus Transcription in Human Trigeminal Ganglia Obtained Soon after Death. J.Virol. 86.18 (2012): 10203–10206.

Pampliega, O. et al. Increased Expression of Cystine/glutamate Antiporter in Multiple Sclerosis. J.Neuroinflammation. 8.1742-2094 (Electronic) (2011): 63.

Pavlakis, P.P. et al. Peripheral Neuropathies in Sjogren Syndrome: a New Reappraisal. J.Neurol. Neurosurg.Psychiatry 82.7 (2011): 798–802.

Peferoen, L.A. et al. Epstein Barr Virus Is Not a Characteristic Feature in the Central Nervous System in Established Multiple Sclerosis. Brain 133.5 (2010): e137.

Pereira, S. et al. Nuclear Localization of a Novel Human Syntaxin 1B Isoform. Gene 423.2 (2008): 160–171.

Perng, M.D. et al. Glial Fibrillary Acidic Protein Filaments Can Tolerate the Incorporation of Assembly-compromised GFAP-delta, but with Consequences for Filament Organization and alphaB-crystallin Association. Mol.Biol.Cell 19.10 (2008): 4521–4533.

Persengiev, S. et al. Genome-wide Analysis of miRNA Expression Reveals a Potential Role for miR-144 in Brain Aging and Spinocerebellar Ataxia Pathogenesis. Neurobiology of Aging 32.12 (2011): 2316–e17.

Persengiev, S., I. Kondova, and R.E. Bontrop. Functional Annotation of Small Noncoding RNAs Target Genes Provides Evidence for a Deregulated Ubiquitin-Proteasome Pathway in Spinocerebellar Ataxia Type 1. Journal of nucleic acids 2012 (2012): n. pag.

Persengiev, S.P., I.I. Kondova, and R.E. Bontrop. The Impact of microRNAs on Brain Aging and Neurodegeneration. Current gerontology and geriatrics research 2012 (2012): n. pag.

Petanjek, Z. et al. Lifespan Alterations of Basal Dendritic Trees of Pyramidal Neurons in the Human Prefrontal Cortex: a Layer-specific Pattern. Cereb.Cortex 18.4 (2008): 915–929.

Pollio, G. et al. Increased Expression of the Oligopeptidase THOP1 Is a Neuroprotective Response to Abeta Toxicity. Neurobiol.Dis. 31.1 (2008): 145–158.

Qiao, J-P. et al. Novel Indanone Derivatives as Potential Imaging Probes for β-Amyloid Plaques in the Brain. ChemBioChem 13.11 (2012): 1652–1662.

Quadri, M. et al. Mutations in SLC30A10 Cause Parkinsonism and Dystonia with Hypermanganesemia, Polycythemia, and Chronic Liver Disease. The American Journal of Human Genetics 90.3 (2012): 467–477.

Quinn, J.G. et al. alpha-Synuclein mRNA and Soluble Alpha-synuclein Protein Levels in Postmortem Brain from Patients with Parkinson's Disease, Dementia with Lewy Bodies, and Alzheimer's Disease. Brain Res. 1459.1872-6240 (Electronic) (2012): 71–80.

Ramaglia, V. et al. C3-dependent Mechanism of Microglial Priming Relevant to Multiple Sclerosis. Proceedings of the National Academy of Sciences 109.3 (2012): 965–970.

Rascovsky, K. et al. Sensitivity of Revised Diagnostic Criteria for the Behavioural Variant of Frontotemporal Dementia. Brain 134.9 (2011): 2456–2477.

Reuwer, A.Q. et al. The Prolactin Receptor Is Expressed in Macrophages Within Human Carotid Atherosclerotic Plaques: a Role for Prolactin in Atherogenesis? J.Endocrinol. 208.2 (2011): 107–117.

Richard, E., A. Carrano, et al. Characteristics of Dyshoric Capillary Cerebral Amyloid Angiopathy. J.Neuropathol.Exp.Neurol. 69.11 (2010): 1158–1167.

Richard, E., W.A. van Gool, et al. Morphometric Changes in the Cortical Microvascular Network in Alzheimer's Disease. J.Alzheimers.Dis. 22.3 (2010): 811–818.

Roberts, J.C. et al. Autoradiographical Imaging of PPARgamma Agonist Effects on PBR/TSPO Binding in TASTPM Mice. Exp.Neurol. 216.2 (2009): 459–470.

Roses, A.D. et al. A TOMM40 Variable-length Polymorphism Predicts the Age of Late-onset Alzheimer's Disease. Pharmacogenomics.J. 10.5 (2009): 375–384.

Roses, A.D. et al. An Inherited Variable poly-T Repeat Genotype in TOMM40 in Alzheimer Disease. Arch.Neurol. 67.5 (2010): 536–541.

Royer-Zemmour, B. et al. Epileptic and Developmental Disorders of the Speech Cortex: Ligand/receptor Interaction of Wild-type and Mutant SRPX2 with the Plasminogen Activator Receptor uPAR. Hum.Mol.Genet. 17.23 (2008): 3617–3630.

Rozemuller, A.J.M. et al. Neuroinflammation and Common Mechanism in Alzheimer's Disease and Prion Amyloidosis: Amyloid-Associated Proteins, Neuroinflammation and Neurofibrillary Degeneration. Neurodegenerative Diseases 10.1-4 (2012): 301–304.

Santa-Mara, I. et al. Coenzyme q Induces Tau Aggregation, Tau Filaments, and Hirano Bodies. J.Neuropathol.Exp.Neurol. 67.5 (2008): 428–434.

Schieb, H. et al. β-Amyloid Peptide Variants in Brains and Cerebrospinal Fluid from Amyloid Precursor Protein (APP) Transgenic Mice COMPARISON WITH HUMAN ALZHEIMER AMYLOID. Journal of Biological Chemistry 286.39 (2011): 33747–33758.

Schreibelt, G. et al. Protective Effects of Peroxiredoxin-1 at the Injured Blood-brain Barrier. Free Radic.Biol.Med. 45.3 (2008): 256–264.

Seabrook, T.J. et al. Angiogenesis Is Present in Experimental Autoimmune Encephalomyelitis and Pro-angiogenic Factors Are Increased in Multiple Sclerosis Lesions. J.Neuroinflammation. 7.1 (2010): 95.

Seelaar, H, JM Papma, et al. Brain Perfusion Patterns in Familial Frontotemporal Lobar Degeneration. Neurology 77.4 (2011): 384–392.

Seelaar, H, W Kamphorst, et al. Distinct Genetic Forms of Frontotemporal Dementia. Neurology 71.16 (2008): 1220–1226.

Seelaar, H. et al. Frequency of Ubiquitin and FUS-positive, TDP-43-negative Frontotemporal Lobar Degeneration. J.Neurol. 257.5 (2010): 747–753.

Seewann, A., H. Vrenken, et al. Diffusely Abnormal White Matter in Chronic Multiple Sclerosis: Imaging and Histopathologic Analysis. Arch.Neurol. 66.5 (2009): 601–609.

Seewann, A., E.J. Kooi, S.D. Roosendaal, P.J. Pouwels, et al. Postmortem Verification of MS Cortical Lesion Detection with 3D DIR. Neurology 78.5 (2012): 302–308.

Seewann, A., E.J. Kooi, S.D. Roosendaal, F. Barkhof, et al. Translating Pathology in Multiple Sclerosis: The Combination of Postmortem Imaging, Histopathology and Clinical Findings. Acta Neurol.Scand. 119.6 (2009): 349–355.

Shan, L., K. Bossers, U. Unmehopa, et al. Alterations in the Histaminergic System in Alzheimer's Disease: a Postmortem Study. Neurobiology of Aging 33.11 (2012): 2585–2598.

Shan, L., K. Bossers, S. Luchetti, et al. Alterations in the Histaminergic System in the Substantia Nigra and Striatum of Parkinson's Patients: a Postmortem Study. Neurobiology of Aging 33.7 (2012): 1488.e1–1488.e13.

Shan, L., M.A. Hofman, D.J. van Wamelen, E.J.W. Van Someren, et al. Diurnal Fluctuation in Histidine Decarboxylase Expression, the Rate Limiting Enzyme for Histamine Production, and Its Disorder in Neurodegenerative Diseases. Sleep 35.5 (2012): 713.

Shan, L., C-Q. Liu, R. Balesar, M.A. Hofman, et al. Neuronal Histamine Production Remains Unaltered in Parkinson's Disease Despite the Accumulation of Lewy Bodies and Lewy Neurites in the Tuberomamillary Nucleus. Neurobiology of Aging 33.7 (2012): 1343–1344.

Shen, C. et al. Hydrogen Peroxide Promotes Abeta Production through JNK-dependent Activation of Gamma-secretase. J.Biol.Chem. 283.25 (2008): 17721–17730.

Shen, Y.X. et al. Hrd1 Facilitates Tau Degradation and Promotes Neuron Survival. Current Molecular Medicine 12.2 (2012): 138–152.

Simón-Sánchez, J. et al. The Clinical and Pathological Phenotype of C9ORF72 Hexanucleotide Repeat Expansions. Brain 135.3 (2012): 723–735.

Smolders, J. et al. Vitamin D in the Healthy and Inflamed Central Nervous System: Access and Function. European Charcot Foundation Symposium A Reappraisal of Nutrition and Environment in Multiple Sclerosis European Charcot Foundation Symposium 311.1–2 (2011): 37–43.

Somel, M. et al. MicroRNA-driven Developmental Remodeling in the Brain Distinguishes Humans from Other Primates. PLoS biology 9.12 (2011): e1001214.

Steen, C. et al. Reduced Creatine Kinase B Activity in Multiple Sclerosis Normal Appearing White Matter. PLoS ONE 5.5 (2010): e10811.

Stockley, J.H., and C. O'Neill. Understanding BACE1: Essential Protease for Amyloid-beta Production in Alzheimer's Disease. Cell Mol.Life Sci. 65.20 (2008): 3265–3289.

Sun, Y. et al. Loss-of-function Mutations in IGSF1 Cause an X-linked Syndrome of Central Hypothyroidism and Testicular Enlargement. Nat Genet 44.12 (2012): 1375–1381.

Svedberg, M.M. et al. [(11)C]PIB-amyloid Binding and Levels of Abeta40 and Abeta42 in Post-mortem Brain Tissue from Alzheimer Patients. Neurochem.Int. 54.5 (2009): 347–357.

Swaab, D.F., and A.M. Bao. (Re-)activation of Neurons in Aging and Dementia: Lessons from the Hypothalamus. Exp.Gerontol. 46.2 (2011): 178–184.

Swahn, B.M. et al. Synthesis and Evaluation of 2-pyridylbenzothiazole, 2-pyridylbenzoxazole and 2-pyridylbenzofuran Derivatives as 11C-PET Imaging Agents for Beta-amyloid Plaques. Bioorg.Med.Chem.Lett. 20.6 (2010): 1976–1980.

Swaminathan, S. et al. Analysis of Copy Number Variation in Alzheimer's Disease in a Cohort of Clinically Characterized and Neuropathologically Verified Individuals. PLoS ONE 7.12 (2012): 1–12.

Tallantyre, E.C. et al. Greater Loss of Axons in Primary Progressive Multiple Sclerosis Plaques Compared to Secondary Progressive Disease. Brain 132.5 (2009): 1190–1199.

Tallantyre, E.C. et al. Clinico-pathological Evidence That Axonal Loss Underlies Disability in Progressive Multiple Sclerosis. Multiple Sclerosis 16.4 (2010): 406–411.

Taziaux, M., D.F. Swaab, and J.Bakker. Sex Differences in the Neurokinin B System in the Human Infundibular Nucleus. Journal of Clinical Endocrinology & Metabolism 97.12 (2012): E2210–E2220.

Teunissen, C. E. et al. Brain-specific Fatty Acid-binding Protein Is Elevated in Serum of Patients with Dementia-related Diseases. European Journal of Neurology 18.6 (2011): 865–871.

Timmons, S. et al. Akt Signal Transduction Dysfunction in Parkinson's Disease. Neurosci. Lett. 467.1 (2009): 30–35.

Tofighi, R. et al. Galanin and Its Three Receptors in Human Pituitary Adenoma. Neuropeptides 46.5 (2012): 195–201.

Toiber, D. et al. N-acetylcholinesterase-induced Apoptosis in Alzheimer's Disease. PLoS.One. 3.9 (2008): e3108.

Tong, Z., C. Han, et al. Accumulated Hippocampal Formaldehyde Induces Age-dependent Memory Decline. Age (Dordr.) 1574-4647 (Electronic) (2012): n. pag.

Tong, Z., J. Zhang, et al. Urine Formaldehyde Level Is Inversely Correlated to Mini Mental State Examination Scores in Senile Dementia. Neurobiol.Aging 32.1 (2011): 31–41.

Torkildsen, O. et al. Upregulation of Immunoglobulin-related Genes in Cortical Sections from Multiple Sclerosis Patients. Brain Pathol. 20.4 (2010): 720–729.

Trouw, L.A. et al. C4b-binding Protein in Alzheimer's Disease: Binding to Abeta1-42 and to Dead Cells. Mol.Immunol. 45.13 (2008): 3649–3660.

Tsvetkov, Peter et al. E3 Ligase STUB1/CHIP Regulates NAD(P)H:Quinone Oxidoreductase 1 (NQO1) Accumulation in Aged Brain, a Process Impaired in Certain Alzheimer Disease Patients. Journal of Biological Chemistry 286.11 (2011): 8839–8845.

Ulfman, L.H. et al. Homeostatic Intracellular-free Ca2+ Is Permissive for Rap1-mediated Constitutive Activation of Alpha4 Integrins on Eosinophils. J.Immunol. 180.8 (2008): 5512–5519.

Valdes-Gonzalez, T. et al. New Approach for Glyco- and Lipidomics--molecular Scanning of Human Brain Gangliosides by TLC-Blot and MALDI-QIT-TOF MS. J.Neurochem. 116.5 (2011): 678–683.

Van Abel, D. et al. Direct Downregulation of CNTNAP2 by STOX1A Is Associated with Alzheimer's Disease. Journal of Alzheimer's Disease 31.4 (2012): 793–800.

Van de Berg, W.D.J. et al. 1.7.1 NEUROPATHOLOGY IN THE PREMOTOR STAGES AND SUBTYPES OF PARKINSON'S DISEASE. Abstracts of WFN XIX World Congress on Parkinson's Disease and Related Disorders 18, Supplement 2.0 (2012): S4.

Van de Berg, W.D.J. et al. Patterns of Alpha-synuclein Pathology in Incidental Cases and Clinical Subtypes of Parkinson's Disease. Proceedings of WFN XIX World Congress on Parkinson's Disease and Related Disorders 18, Supplement 1.0 (2012): S28–S30.

Van den Berge, S.A., J.T. Kevenaar, et al. Dementia in Parkinson's Disease Correlates with alpha-Synuclein Pathology but Not with Cortical Astrogliosis. Parkinsons.Dis. 2012.2042-0080 (Electronic) (2012): 420957.

Van den Berge, S.A., J. Middeldorp, et al. Longterm Quiescent Cells in the Aged Human Subventricular Neurogenic System Specifically Express GFAP-delta. Aging Cell 9.1474-9726 (Electronic) (2010): 313–326.

Van den Berge, S.A., M.E. van Strien, et al. Reply: Quantitative Evaluation of the Human Subventricular Zone. Brain 135.8 (2012): e222–e222.

Van den Berge, S.A., M.E. van Strien, et al. The Proliferative Capacity of the Subventricular Zone Is Maintained in the Parkinsonian Brain. Brain 134.11 (2011): 3249–3263.

Van der Star, B.J. et al. In Vitro and In Vivo Models of Multiple Sclerosis. CNS & Neurological Disorders - Drug Targets 11.5 (2012): 570–588.

Van der Valk, P., and S. Amor. Preactive Lesions in Multiple Sclerosis. Current opinion in neurology 22.3 (2009): 207–213.

Van Dijk, Karin D. et al. The Proteome of the Locus Ceruleus in Parkinson's Disease: Relevance to Pathogenesis. Brain Pathology 22.4 (2012): 485–498.

Van Dijk, M. et al. The Pre-eclampsia Gene STOX1 Controls a Conserved Pathway in Placenta and Brain Upregulated in Late-onset Alzheimer's Disease. J.Alzheimers.Dis. 19.2 (2010): 673–679.

Van Doorn, R. et al. Sphingosine 1-phosphate Receptor 1 and 3 Are Upregulated in Multiple Sclerosis Lesions. Glia 58.12 (2010): 1465–1476.

Van Doorn, R. et al. Sphingosine 1-phosphate Receptor 5 Mediates the Immune Quiescence of the Human Brain Endothelial Barrier. J Neuroinflammation 9.1 (2012): 133.

Van Eijk, M., G. Aust, M.S. Brouwer, Meurs M. van, et al. Differential Expression of the EGF-TM7 Family Members CD97 and EMR2 in Lipid-laden Macrophages in Atherosclerosis, Multiple Sclerosis and Gaucher Disease. Immunol.Lett. 129.1879-0542 (Electronic) (2010): 64–71.

Van Gassen, K.L. et al. Hippocampal Nabeta3 Expression in Patients with Temporal Lobe Epilepsy. Epilepsia 50.4 (2009): 957–962.

Van Horssen, J., J.A. Drexhage, T. Flor, W. Gerritsen, et al. Nrf2 and DJ1 Are Consistently Upregulated in Inflammatory Multiple Sclerosis Lesions. Free Radic.Biol.Med. 49.1873-4596 (Electronic) (2010): 1283–1289.

Van Horssen, J., G. Schreibelt, J. Drexhage, T. Hazes, et al. Severe Oxidative Damage in Multiple Sclerosis Lesions Coincides with Enhanced Antioxidant Enzyme Expression. Free Radic.Biol.Med. 45.0891-5849 (Print) (2008): 1729–1737.

Van Horssen, J. et al. Clusters of Activated Microglia in Normal-appearing White Matter Show Signs of Innate Immune Activation. J Neuroinflammation 9.1 (2012): 156.

Van Luijn, MM et al. Downregulation of C-type Lectin CLEC16A Causes Late Endosomal Dysfunction and Impairs Processing and Surface Expression of HLA Class II. 137.Supplement s1 (2012): 28–28.

Van Noort, J.M., D. Baker, and S. Amor. Mechanisms in the Development of Multiple Sclerosis Lesions: Reconciling Autoimmune and Neurodegenerative Factors. CNS & Neurological Disorders - Drug Targets 11.5 (2012): 556–569.

Van Noort, J.M., M. Bsibsi, et al. Alphab-crystallin Is a Target for Adaptive Immune Responses and a Trigger of Innate Responses in Preactive Multiple Sclerosis Lesions. J.Neuropathol. Exp.Neurol. 69.7 (2010): 694–703.

Van Noort, J.M., P.J. van den Elsen, et al. Preactive Multiple Sclerosis Lesions Offer Novel Clues for Neuroprotective Therapeutic Strategies. CNS.Neurol.Disord.Drug Targets. 10.1 (2011): 68–81.

Van Noort, J.M. Stress Proteins in CNS Inflammation. J.Pathol. 214.2 (2008): 267–275.

Van Noort, J.M., and M. Bsibsi. Toll-like Receptors in the CNS: Implications for Neurodegeneration and Repair. Prog.Brain Res. 175.1875-7855 (Electronic) (2009): 139–148.

Van Strien, M.E., S.A. van den Berge, and E.M. Hol. Migrating Neuroblasts in the Adult Human Brain: a Stream Reduced to a Trickle. Cell Research 21.11 (2011): 1523–1525.

Van Strien, M.E. et al. Appearance of Tissue Transglutaminase in Astrocytes in Multiple Sclerosis Lesions: A Role in Cell Adhesion and Migration? Brain Pathology 21.1 (2011): 44–54.

Van Swieten, J.C., and P. Heutink. Mutations in Progranulin (GRN) Within the Spectrum of Clinical and Pathological Phenotypes of Frontotemporal Dementia. Lancet Neurol. 7.10 (2008): 965–974.

Van Tijn, P., E.M. Hol, F.W. van Leeuwen, and D.F. Fischer. The Neuronal Ubiquitin-proteasome System: Murine Models and Their Neurological Phenotype. Prog.Neurobiol. 85.0301-0082 (Print) (2008): 176–193.

Van Tijn, P. et al. Presenilin Mouse and Zebrafish Models for Dementia: Focus on Neurogenesis. Progress in neurobiology 93.2 (2011): 149–164.

Van Velzen, M., J.D. Laman, A. Kleinjan, A. Poot, et al. Neuron-interacting Satellite Glial Cells in Human Trigeminal Ganglia Have an APC Phenotype. J.Immunol. 183.1550-6606 (Electronic) (2009): 2456–2461.

Van Velzen, M. et al. Latent Acyclovir-resistant Herpes Simplex Virus Type 1 in Trigeminal Ganglia of Immunocompetent Individuals. Journal of Infectious Diseases 205.10 (2012): 1539–1543.

Van Wamelen, D.J. et al. Paraventricular Nucleus Neuropeptide Expression in Huntington's Disease Patients. Brain Pathol. 22.5 (2012): 654–661.

Van Wamelen, D.J. et al. Functional Increase of Brain Histaminergic Signaling in Huntington's Disease. Brain Pathology 21.4 (2011): 419–427.

Van Zwam, M., R. Huizinga, M.J. Melief, et al. Brain Antigens in Functionally Distinct Antigen-presenting Cell Populations in Cervical Lymph Nodes in MS and EAE. J.Mol.Med. 87.1432-1440 (Electronic) (2009): 273–286.

Van Zwam, M., A.F. Wierenga-Wolf, et al. Myelin Ingestion by Macrophages Promotes Their Motility and Capacity to Recruit Myeloid Cells. J.Neuroimmunol. 225.1872-8421 (Electronic) (2010): 112–117.

Van Zwam, M., R. Huizinga, N. Heijmans, et al. Surgical Excision of CNS-draining Lymph Nodes Reduces Relapse Severity in Chronic-relapsing Experimental Autoimmune Encephalomyelitis. J.Pathol. 217.1096-9896 (Electronic) (2009): 543–551.

Varani, K. et al. A2A Adenosine Receptor Overexpression and Functionality, as Well as TNF-alpha Levels, Correlate with Motor Symptoms in Parkinson's Disease. FASEB J. 24.2 (2010): 587–598.

Venkataramani, V. et al. Antibody 9D5 Recognizes Oligomeric Pyroglutamate Amyloid-beta in a Fraction of Amyloid-beta Deposits in Alzheimer's Disease Without Cross-reactivity with Other Protein Aggregates. J.Alzheimers.Dis. 29.2 (2012): 361–371.

Verwey, N.A. et al. Quantification of Amyloid-beta 40 in Cerebrospinal Fluid. J.Immunol. Methods 348.1-2 (2009): 57–66.

Vitali, M. et al. Analysis of the Genes Coding for Subunit 10 and 15 of Cytochrome c Oxidase in Alzheimer's Disease. J.Neural Transm. 116.12 (2009): 1635–1641.

Wang, Q et al. Hippocampal GR Expression Is Increased in Elderly Depressed Females. Neuropharmacology 62.1 (2012): 527–533.

Wang, S.S. et al. Gene Expression Analysis in the Human Hypothalamus in Depression by Laser Microdissection and Real-time PCR: The Presence of Multiple Receptor Imbalances. Mol.Psychiatry 13.8 (2008): 786–99, 741.

Watanabe, T., Y. Hikichi, et al. FBL2 Regulates Amyloid Precursor Protein (APP) Metabolism by Promoting Ubiquitination-dependent APP Degradation and Inhibition of APP Endocytosis. J.Neurosci. 32.10 (2012): 3352–3365.

Watanabe, T., Kammer H. von der, et al. Neuronal Expression of F-Box and Leucine-Rich-Repeat Protein 2 Decreases over Braak Stages in the Brains of Alzheimer's Disease Patients. Neurodegener.Dis. 11.1 (2012): n. pag.

Wawrzik, M. et al. The C15orf2 Gene in the Prader-Willi Syndrome Region Is Subject to Genomic Imprinting and Positive Selection. Neurogenetics. 11.2 (2010): 153–161.

Westerlund, M. et al. Altered Enzymatic Activity and Allele Frequency of OMI/HTRA2 in Alzheimer's Disease. The FASEB Journal 25.4 (2011): 1345–1352.

White, J.H. et al. Identification of a Novel Asthma Susceptibility Gene on Chromosome 1qter and Its Functional Evaluation. Hum.Mol.Genet. 17.13 (2008): 1890–1903.

Wijte, D. et al. A Novel Peptidomics Approach to Detect Markers of Alzheimer's Disease in Cerebrospinal Fluid. Methods 56.4 (2012): 500–507.

Wilczak, N. et al. IGF Binding Protein Alterations on Periplaque Oligodendrocytes in Multiple Sclerosis: Implications for Remyelination. Neurochem.Int. 52.8 (2008): 1431–1435.

Wilhelmus, M.M., S.M. van der Pol, et al. Association of Parkinson Disease-related Protein PINK1 with Alzheimer Disease and Multiple Sclerosis Brain Lesions. Free Radic.Biol. Med. 50.3 (2010): 469–476.

Wilhelmus, M.M., R. Verhaar, et al. Presence of Tissue Transglutaminase in Granular Endoplasmic Reticulum Is Characteristic of Melanized Neurons in Parkinson's Disease Brain. Brain Pathol. 21.2 (2011): 130–139.

Wilhelmus, M.M., Jager M. de, et al. Transglutaminase 1 and Its Regulator Tazarotene-induced Gene 3 Localize to Neuronal Tau Inclusions in Tauopathies. J.Pathol. 226.1 (2012): 132–142.

Wilhelmus, M.M., S.C. Grunberg, et al. Transglutaminases and Transglutaminase-Catalyzed Cross-Links Colocalize with the Pathological Lesions in Alzheimer's Disease Brain. Brain Pathol. 19.4 (2009): 612–622.

Wilhelmus, M. M.M. et al. Novel Role of Transglutaminase 1 in Corpora Amylacea Formation? Neurobiology of Aging 32.5 (2011): 845–856.

Willems, J.G.P. et al. Immunopathology of the Hippocampus in Multiple Sclerosis. Multiple Sclerosis Journal 17.10S (2011): S325–S325.

Wirths, O. et al. Identification of Low Molecular Weight Pyroglutamate A{beta} Oligomers in Alzheimer Disease: a Novel Tool for Therapy and Diagnosis. J.Biol.Chem. 285.53 (2010): 41517–41524.

Wirths, O. et al. Pyroglutamate Abeta Pathology in APP/PS1KI Mice, Sporadic and Familial Alzheimer's Disease Cases. Journal of neural transmission 117.1 (2010): 85–96.

Witte, M.E., L. Bo, et al. Enhanced Number and Activity of Mitochondria in Multiple Sclerosis Lesions. J.Pathol. 219.2 (2009): 193–204.

Witte, M.E., J.J. Geurts, et al. Mitochondrial Dysfunction: a Potential Link Between Neuroinflammation and Neurodegeneration? Mitochondrion. 10.5 (2010): 411–418.

Witte, M.E., J.G. Bol, et al. Parkinson's Disease-associated Parkin Colocalizes with Alzheimer's Disease and Multiple Sclerosis Brain Lesions. Neurobiol.Dis. 36.3 (2009): 445–452.

Witte, M.E., P.G. Nijland, et al. Reduced Expression of PGC-1alpha Partly Underlies Mitochondrial Changes and Correlates with Neuronal Loss in Multiple Sclerosis Cortex. Acta Neuropathol. 125.2 (2012): 231–243.

Wu, L. et al. Neural Stem Cells Improve Neuronal Survival in Cultured Postmortem Brain Tissue from Aged and Alzheimer Patients. J.Cell Mol.Med. 12.5 (2008): 1611–1621.

Yang, Q. et al. DNA Methylation of the Monoamine Oxidases A and B Genes in Postmortem Brains of Subjects with Schizophrenia. Lung 3.77 (2012): 5–5.

Yi, C-X. et al. High Calorie Diet Triggers Hypothalamic Angiopathy. Molecular Metabolism 1.1–2 (2012): 95–100.

Zhao, J. et al. Gene Expression of GABA and Glutamate Pathway Markers in the Prefrontal Cortex of Non-suicidal Elderly Depressed Patients. J.Affect.Disord. 138.3 (2012): 494–502.

Zhao, T. et al. Loss of Nuclear Activity of the FBXO7 Protein in Patients with Parkinsonian-pyramidal Syndrome (PARK15). PloS one 6.2 (2011): e16983.

Zhou, T. et al. Dendritic Cell Nuclear Protein-1, a Novel Depression-related Protein, Upregulates Corticotropin-releasing Hormone Expression. Brain 133.10 (2010): 3069–3079.

Zou, F. et al. Brain Expression Genome-wide Association Study (eGWAS) Identifies Human Disease-associated Variants. PLoS.Genet. 8.6 (2012): e1002707.

Zouambia, M. et al. Proteasome Subunit Proteins and Neuropathology in Tauopathies and Synucleinopathies: Consequences for Proteomic Analyses. Proteomics. 8.6 (2008): 1221–1236.

Staff and Collaborations

Managing director NIN

R. van der Neut	r.van.der.neut@nin.knaw.nl

Director

I. Huitinga	i.huitinga@nin.knaw.nl

Technical coordinator

M. Kooreman	m.kooreman@nin.knaw.nl

Management assistant

B. van Huik (from December 2012)	b.van.huik@nin.knaw.nl
M.C. Rademaker (until December 2012)	m.rademaker@nin.knaw.nl

Secretariat

P. Brom	p.brom@nin.knaw.nl

Lab technicians

A. van den Berg	a.van.den.berg@nin.nl
P. Evers	p.evers@nin.knaw.nl

Medical writers

C. van Eden	c.van.eden@nin.knaw.nl
I. Ehmer (from January 2011)	i.ehmer@nin.knaw.nl

Project coordinator NBB-Psy

M.C. Rademaker	m.rademaker@nin.knaw.nl

Neuropathologists

J.M. Rozemuller Department of Pathology, VUmc	jm.rozemuller@vumc.nl
P. van der Valk Department of Pathology, VUmc	p.vandervalk@vumc.nl

M. Bugiani
Department of Pathology, VUmc
m.bugiani@vumc.nl

P. van der Voorn
Department of Pathology, VUmc
jp.vandervoorn@vumc.nl

W. Kamphorst
Department of Pathology, VUmc

Neurologist

S. Luchetti
(For evaluation of clinical files of MS donors)
s.luchetti@nin.knaw.nl

Autopsy team

J. Anink, A. van den Berg, S. Bosman (from March 2012), P. Evers, S. Hoyng, M. Kooreman, J. Korecka, C. Mamber, K. Roet, K. Schuurman, L. Shan, U. Unmehopa, Y. van der Werf.

We owe special thanks to the autopsy assistants of the Pathological Institute, VUmc, Amsterdam, A. Bakker, P. Kraaijeveld, T. Oldert and R. Vos, and to John and Thomas of Rouwservice Nederland and the undertakers of Uitvaartcentrum Zuid (Unigra) for their dedication to the Netherlands Brain Bank.

Tissue Advisory Board (scientific committee)

I. Huitinga (NBB)
J. Verhaagen (Netherlands Institute for Neuroscience)
J.M. Rozemuller (Department of Pathology, VUmc)
M. Kooreman (NBB)

Advisory Council

Prof. Dr. D.A.J.P. Denys (Psychiatry)
Prof. Mr. Dr. J.K.M. Gevers (Health Law)
Prof. Dr. P. Heutink (Genetics)
Drs. A.A. Keizer (Geriatrics)
Prof. Dr. H.P.H. Kremer (Neurology)
Dr. E. Marchiori (Informatics)
Prof. Dr. C.H. Polman (Neurology)
Prof. Dr. P. van der Valk (Pathology)

List of Abbreviations

Diagnoses

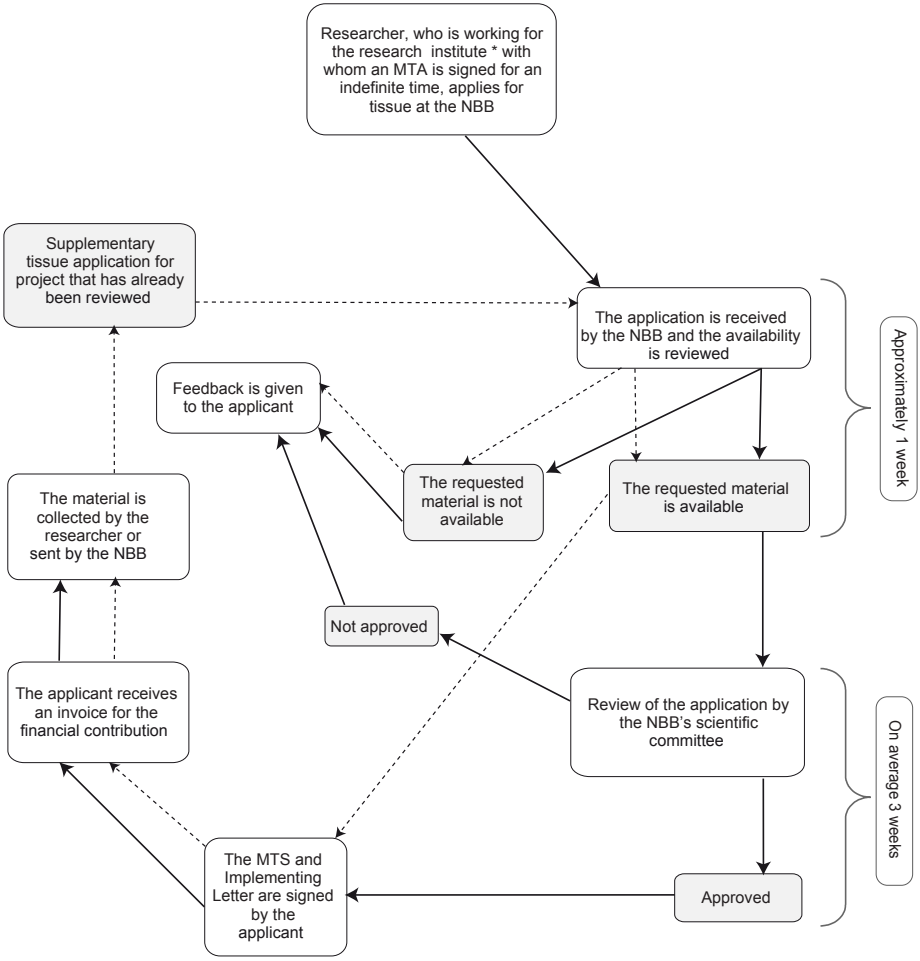
AD	Alzheimer's disease
Contr	Non-demented controls
FTLD/tau	Frontotemporal lobar degeneration/Tauopathy
MS	Multiple sclerosis
Other dem	Other dementia
PANR	Pathological report not ready
PD/DLBD	Parkinson's disease/Diffuse Lewy body dementia
PSP	Progressive supranuclear palsy
Psych	Psychiatric disorders
Rest group	Other diagnoses
Trans	Transsexuality
Vasc	Vascular dementia

Organizations

AMC	Academic Medical Center
KNAW	Koninklijke Nederlandse Akademie van Wetenschappen (Royal Netherlands Academy of Arts and Sciences)
NBB	Netherlands Brain Bank
NIN	Nederlands Herseninstituut (Netherlands Institute for Neuroscience)
NWO	Nederlandse Organisatie voor Wetenschappelijk Onderzoek (Netherlands Organisation for Scientific Research)
NKCA	Nationaal Kenniscentrum Alternatieven voor Dierproeven (Netherlands Knowledge Centre on Alternatives to Animal Use)
VUmc	VU University medical centre

Appendix

Figure 14 NBB's Procedure of Material Transfer



* The research institute is a legal entity with whom the MTA is signed. Legally, the research institute is thus a party of the agreement. The research institute is thus called "Recipient" of the Material in the MTA and *not* the researcher.
In case no MTA for indefinite time has been signed at the institute/organization where the researcher is working, the NBB will not supply any tissue. First, the authorized person (head manager or managing coordinator) needs to sign the MTA.

——— Application new project
- - - - - - - - - - Supplementary application within reviewed project

Figure 15 Non-hierarchic scheme of the organization of the Netherlands Brain Bank