Issue No. 54
Winter 2014

Psychiatry Research Trust

EXPLORING NEW HORIZONS
in Mental Health
and Brain Disease Research

....To raise funds for research into mental illness and brain disorders in co-operation with the Institute of Psychiatry, Psychology and Neuroscience and Bethlem Royal and Maudsley Hospitals
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EDITORIAL

We do hope that you had an enjoyable summer. It only seems a short while ago that we were sending out our Spring newsletter and now, here we are, with our Winter issue. It seems the shops have had their Christmas stock on sale for ages and, not to be left out, please find enclosed our Christmas card flyer. We were rather late with this last year so, hopefully, this year there will be more time for you to order your cards from us.
Below you will find information about the newly created Leslie (Nicky) Paine Memorial Fund. As one of Nicky's constant appeals was for donations towards our general fund we thought a Memorial Fund for these funds would be an appropriate way of remembering him. The majority of donations received are asked to be used for specific areas of research and are of great importance for us to fund these particular research areas. General funds are necessary to support areas of research which do not fall within these designated areas, such as small projects and relatively modest requests by young researchers who have difficulty obtaining funding elsewhere.

One of Nicky's roles as our Director was donning his editor's hat and proofreading the newsletters. We are very fortunate in that his partner, Christine Lutman, has agreed to take on this role.

You might have noticed that the Institute of Psychiatry (IoP) has now changed its name to The Institute of Psychiatry, Psychology and Neuroscience (IoPPN). A long title but it reflects the ever growing remit of the Institute's research and teaching.

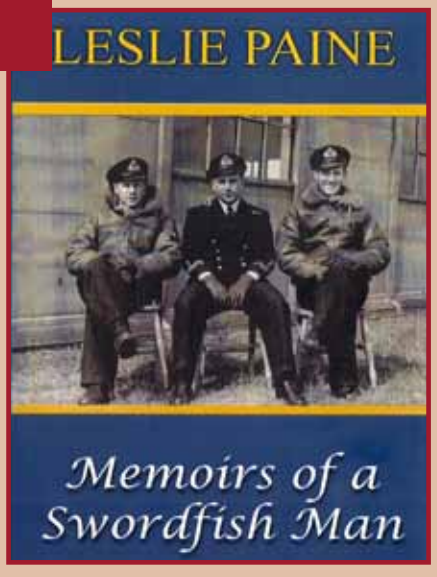
Finally, (though it does seem rather early!) we will take this opportunity to wish you all an enjoyable Christmas and New Year. Many thanks for your continuing support and don't forget, keep raising those funds for our researchers and do send us your fundraising event photos for inclusion in the newsletter and on our website.

Lesley Pease
Chief Administrator

LESLIE PAINE MEMORIAL FUND

In memory of our much missed Director, Leslie (Nicky) Paine, we are launching the Leslie Paine Memorial Fund. After reporting his death last December in our previous Newsletter, we have received many kind words and donations in his memory.

The Trustees now wish to establish a memorial fund in Leslie's name in recognition of his tireless work supporting the PRT As many of you will remember from his Trust Director's Reports in past Newsletters, Leslie constantly appealed for general funds to maintain the running of the Trust and his memorial fund will continue his work to raise general funds.



Shortly before his death, Leslie's last book, Memoirs of a Swordfish Man, was published and his partner, Christine, has generously donated a number of copies of the book, from a limited print run, as part of raising donations for the Memorial Fund. While recounting the serious subject of his experiences as a pilot in the Fleet Air Arm during the second world war (for which he received medals from both the British and Russian governments), these memoirs are subject to his usual modest and light touch. Quoting from the first chapter, Leslie writes "….events and experiences – amusing, sometimes sad, occasionally surprising, but always, I hope, entertaining and interesting, albeit just part of one ordinary naval airman's war". This is a truly charming and readable book which can be purchased from the PRT for £9.29 (£7.99 + £1.30 p&p). All proceeds will go toward the Leslie Paine Memorial Fund.

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ASHLEY JONES

GENETICS AND SPREAD IN AMYOTROPHIC LATERAL SCLEROSIS

PROJECT DESCRIPTION

The aim of this research is to characterise the spread of Amyotrophic Lateral Sclerosis (ALS) across anatomy and time. This will implement DNA genotyping and sequencing, calculating gene RNA levels, estimating ALS pathology and incorporating clinical information and models of disease spread. I will isolate DNA and RNA from tissue of spinal areas representing different disease states, compare and model them. Then, largely genetic discovery techniques will be employed using genome wide association analyses of genotypes for ALS cases and non-ALS controls, genome-wide gene expression analyses and an advanced statistical analysis will be used to identify aberrant changes in DNA or RNA. Also, genetic and clinical information will be used to model the disease as it progresses through patient and across time.

PROGRESS IN PAST YEAR

What this research has shown

There are three major publications that have arisen from this project. In summary, the findings are as follows:

(i) In 2011 the most important genetic discovery in ALS was identified in gene C9ORF72. We know now it is the prevalent mutation in ALS. My research highlighted additional disease-causing variance in C9ORF72; which means there is another unknown mutation in C9ORF72. This finding was published, and we now are closer to identify this mutation - it is likely to be another repeat but with alternative nucleotides.

(ii) Typically, projects that analyse RNA in ALS use isolated regions of the spinal cord. The problem with this method is that ALS can begin in almost any spinal cord segment and then spreads outwards. So, for example, if we analyse only lumbar samples in multiple patients, then there is a heterogeneity of disease (some samples will be worse than others). My project takes multiple segments from the spinal cord and measures how RNA changes as the disease spreads. I found significant changes in known ALS genes and new interesting candidates in which their functions are related with pathology.

(iii) In 2012 Professor Ammar Al-Chalabi's group published a staging system in ALS. This system allows one to calculate the speed of progression of the disease by using clinical milestones. This is important in ALS because the speed of the disease from onset to mortality is highly variable between patients. My research has replicated this finding but, furthermore, found that the staging system accurately predicts changes in health-related quality of life (health utility) scores, motor function, depression and anxiety. This is important in clinical trials where a patient's course of ALS varies significantly, making it difficult to calculate this aspects of the disease.

Implications

Here are the following implications, following the numerals from above:

(i) The C9ORF72 mutation in this gene was originally thought to account for approximately 10-15% of all Caucasian ALS patients. The implication of my finding in C9ORF72 calculates that this to be actually >20%.

(ii) Identifying changes in RNA levels in ALS has been problematic. In my project, because we are accounting for heterogeneity between tissue-types, we see for the first-time changes in RNA for major ALS genes, like C9ORF72 and MATR3, and major pathological pathways, like RNA processing and immune-response.

(iii) There are several implications from the research on ALS clinical stages. (a) It validates the clinical staging system in three clinical trials and a patient population register. (b) We can use health utility to estimate economic costs of therapies. (c) It can more accurately measure the efficacy of clinical trial drugs than previously possible.

PUBLICATIONS & CONFERENCES ATTENDED

Five publications:

Sept 2013. **Neurobiology of Aging**
Residual association at C9orf72 suggests an alternative amyotrophic lateral sclerosis-causing hexanucleotide repeat
Jan 2014. **Journal of Neurology, Neurosurgery and Psychiatry**
Use of clinical staging in amyotrophic lateral sclerosis for phase 3 clinical trials
Feb 2014. **Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration**
Health utility decreases with increasing clinical stage in amyotrophic lateral sclerosis.
April 2014. **Human Molecular Genetics**
A genome-wide association meta-analysis identifies a novel locus at 17q11.2 associated with sporadic amyotrophic lateral sclerosis
June 2014 **Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration.**
Estimating clinical stage of amyotrophic lateral sclerosis from the ALS Functional Rating Scale.
Two conferences:
MNDA International Symposium Milan 2013: Poster Presentation on Gene Expression in ALS across the spinal cord
ENCALS Leuven 2014: Young Investigator of the Year Award and Platform/Award Presentation on (multiple findings)

DR. ELAINE HUNTER

CLINICAL STUDIES AND NEUROMODULATION IN DEPERSONALISATION DISORDER

PROJECT DESCRIPTION

Depersonalisation disorder (DPD) is a distressing condition typified by a sense of unreality about the self and the outside world. The project aims to improve our clinical understanding of this problem and develop more effective treatments via two routes a) brain imaging and brain stimulation studies and b) clinical database and psychological interventions.

a. Brain imaging and stimulation studies

   a. The project has carried out structural brain imaging on a sample of patients to investigate the possible regional brain correlates of DPD symptoms.

   b. A pilot study of repetitive Transcranial Magnetic Stimulation (rTMS) in a sample of patients with Depersonalisation disorder has been conducted to see if this improves symptoms.

b. Clinical demographics and therapy

   a. Data has been collected on patients seen in the Depersonalisation Clinic. This database now numbers over 500 patients. This data will be used to write up a clinical case series to document patterns in symptoms.

   b. Since January 2014 patients have been offered cognitive behavioural therapy adapted for Depersonalisation disorder. These cases are being evaluated for outcome. We plan to use this data to apply for funding to run a larger trial to assess the effectiveness of this treatment for DPD.

PROGRESS IN PAST YEAR

Brain imaging and stimulation studies

1. Structural Imaging. We report the first structural MRI study of 20 patients with DPD and 21 controls. DPD patients showed significantly lower cortical thickness in the right middle temporal regions. Clinical severity scores negatively correlated with cortical thickness in middle and right inferior frontal regions. Further research is required to specify their functional significance and whether they are vulnerability or disease markers.

2. rTMS. We hypothesised that inhibition to right ventrolateral prefrontal cortex (VLPFC) using rTMS would lead to increased arousal and reduced symptoms of DPD. 17 patients and 20 controls were randomised to receive one session of right-sided rTMS to VLPFC or temporo-parietal junction (TPJ). Only those patients with DPD who had rTMS to VLPFC showed increased electrodermal capacity, while patients who had either VLPFC or TPJ rTMS showed a similar significant reduction in symptoms. We conclude that rTMS is a potential therapeutic option for DPD.

Depersonalisation Disorder Database

1. More data has been collected from patients referred to the specialist clinic on a range of measures. This database now numbers over 500 cases. An analysis of scores on self-report anxiety measures in patients with depersonalisation from this sample has been carried out and compared with anxiety scores from clinical samples of patients with anxiety disorders who have undergone the Beck Anxiety Inventory (BAI). This has shown interesting similarities and differences between the two conditions for example, the DPD group presented significantly lower scores on the panic subscale, marginally lower scores on the autonomic, and significantly higher scores on the neurophysiological subscale of the BAI.

2. A small open trial of CBT for depersonalisation disorder was carried out in the clinic (Hunter et al., 2005). CBT has been re-established in the clinic with an updated approach. A small number of cases have been completed. The effectiveness of this approach will be evaluated. It is planned that the results from this evaluation project will contribute to a grant application to seek funding for a larger scale study of CBT for Depersonalisation.

PUBLICATIONS & CONFERENCES ATTENDED

Seven Publications

2012 **Journal of Trauma & Dissociation**
Psychophysiological investigations in depersonalisation disorder and effects of electrodermal biofeedback.
2012 **Psychiatry**
Depersonalisation disorder and anxiety: A special relationship?
In press. **Applied Psychophysiology and Biofeedback**
Biofeedback for Psychiatric Disorders: A Systematic Review.
2012 **Journal of nervous and mental disease**
Conditional Reasoning in Asperger's Syndrome and Depersonalisation Disorder
2014 **Brain Simulation**
Testing a neurobiological model of depersonalisation disorder using repetitive transcranial magnetic stimulation.,
2013 **Neuroimaging**
Interoceptive–reflective regions differentiate alexithymia traits in depersonalisation disorder. (2013), **Psychiatry Res: Neuroimaging** Psychiatry Research:
2014
A structural MRI study of cortical thickness in depersonalisation disorder.

Two Conferences

Presentation: **AGM Royal College of Psychiatrist Edinburgh** July 2013. S Nessler. Structural Neuroimaging in Depersonalisation Disorder
Presentations on **CBT for Depersonalisation Disorder** given to SLaM Early Intervention in Psychosis clinical staff (February 2014) and at a Confer talk to clinicians in a Trauma and Dissociation series of Talks

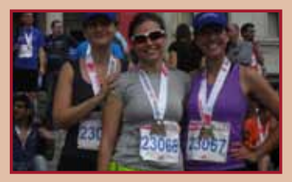
RESEARCH PROJECTS FUNDED FOR 2014 – 2015 FINANCIAL YEAR
as at 1s October 2014
Project funding includes salaries, fellowships, studentships, consumables, equipment and miscellaneous. Grants are made as a result of open competition

Area of Research	Project	Total Costs Funded	Award Duration	Purpose of Funding
Anxiety Disorders	Investigating the neurocognitive regulation of anxiety states using suggestions and fMRI	£50,000	1 ½ years	Salary & Support Costs
Anxiety Disorder	The role of controlled and automatic processes in Generalised Anxiety Disorder	£50,000	1 year	Salary & Support Costs
Bipolar Disorder	Predictors of Suicide & other causes of death in bipolar affective disorder	£10,000	3 years	Research Costs
Bipolar Disorder	Pinpointing resilience in Bipolar Disorder	£22,400	1 year	Support Costs
Depersonalisation	Clinical studies and neuromodulation in Depersonalisation Disorder	£50,000	2 years	Salary
Depression	Impact of Nutrition Adult Neurogenesis and Depression	£17,184	3 years	Laboratory Consumables
Depression	Brief IPT for depression among people newly diagnosed with HIV/AIDS: Adaptation and evaluation using mixed methods	£76,066	To be confirmed	Salary & Support Costs
Depression	How does childhood maltreatment lead to depression in adults?	£50,000	3 years	Salary + Consumables
Depression	Does inflammation cause depressed mood by inducing changes in brain structure?	£50,000	2 years	Consumables
Depressive Disorders, Bipolar Disease & Chronic Fatigue Syndrome	Assessment of hypothalamic-pituitary adrenal axis in atypical and non- atypical major depressive disorder, bipolar depression and chronic fatigue syndrome. (HPA HAIR STUDY)	£6,135	2 years	Participant payments + support costs
Prenatal Depression	A pilot study on the effect of maternal prenatal depression on infant brain development	£56,000	3 years	Salary
Motor Neuron Disease	Role of TDP-43 aggregation in initiating toxicity in human neuronal stem cells and neurons	£36,083	1 year	Salary & Consumables
Motor Neuron Disease	Modelling Motor Neuron Disease using induced pluripotent stem cells	£88,850	3 years	Student Stipend, Fees + support costs
Parkinson's Disease	Objective assessment of muscle rigidity in exploring aetio-pathogenesis of Parkinson's disease	£19,500	3 years	PhD Studentship
Motor Neuron Disease	Gene-hunting in familial amyotrophic lateral sclerosis using exon-capture and high-throughput sequencing and sibling pair analysis	£79,255	3 years	Student Stipend, Fees + support costs
Eating Disorders	An examination of the feasibility acceptability and effectiveness of a skills based intervention for adolescents with eating disorders	£80,000	3 years	PhD Scholarship
Eating Disorders	An fMRI pilot study of the effects of meal support in Eating Disorders	£15,000	2 years	Support Costs
Eating Disorders	New Brain Directed Treatments for Eating Disorders	£43,300	4 years	Student Stipend + Support Costs
Eating Disorders	A multimodal investigation of treatment enhancers to improve social functioning, self-evaluation, and emotional regulation in eating disorders	£38,300	3 years	Student Stipend + Support Costs
International Mental Health	Co-ordinating the Field Trials of ICD11-PHC at the Institute of Psychiatry (World Health Organisation project)	£20,456	1 year	Salary & Support Costs
Parkinsons	Objective assessment of muscle rigidity in exploring aetio-pathogenesis of Parkinson's disease	£19,500	3 years	PhD Studentship
Psychotic Disorders	Interaction between genes and psychosocial factors n the aetiology and outcome of psychotic disorders	£76,090	3 years	Student Stipend + Fees + Consumables
Psychotic Disorders	Investigating the effects of polygenetic risk on the outcome of psychosis	£67,100	3 years	Student Stipend + Fees
Psychiatric Disorders	Exploring the relationship between shortened telomere lengths and psychiatric disorders: dissecting the genetic and environmental contributions	£28,606	2 years	Support Costs
Sleep Disorders	Validation & Development of Overnight Learning Paradigms of Memory Consolidation in Sleep.	£114,000	3 years	Salary & Consumables
Sleep Disorders	The long-term clinical effectiveness of a community, one day, self-referral CBT workshop to improve insomnia: a 4 year follow-up	£73,682	1 year	Salary & Support Costs

LONDON 10K RUN

This year's London 10K Run took place on Sunday 13th July 2014. While last year we needed sun-shades and factor 50, this year we needed umbrellas and waterproofs! However, this was a much more comfortable day for running that the heat of the previous race and the sun did eventually come out and shine on the competitors once they had finished.

Altogether it was a fun morning with thousands of runners ranging from professional athletes to fun runners and, most importantly of all, people taking part to raise funds for a diverse array of charities. We filled our 24 place allocation easily this year with some first time runners and some returning for their second, third or more times and many have asked to run again next year.



Our brilliant runners raised over £2,700 and we send them a huge thank you and hope to see them again next year.

DOMINIC PLANT

BIOLOGICAL MARKERS OF SUSCEPTIBILITY TO DEPRESSION AMONG YOUNG ADULTS EXPOSED TO MATERNAL DEPRESSION IN UTERO

PROJECT DESCRIPTION

The main objective of this study is to explore the effects of adverse early life experiences, gathered prospectively, on young adult psychopathology, with the goal of understanding the processes and mechanisms that underpin this association from a biopsychosocial perspective.

Key aims:
1. To identify pathways from maternal psychopathology and childhood maltreatment to offspring young adult emotional, behavioural and physical health problems.

2. To examine the role of dysregulation in the stress system as a mechanism for the translation of psychosocial adversity into biological susceptibility for depression.

3. To investigate the relationship between inflammation and hypothalamic-pituitary-adrenal (HPA) axis function, and examine its contribution as a biological mechanism for the conversion of psychosocial adversity into disease risk.

A prospective longitudinal design will be employed. A random group of 151 pregnant women initially recruited into the study from two general practices in South London, between January and December 1986, took part in assessments at 20 and 36 weeks gestation, and 3 and 12 months postpartum. Three further follow-up phases have been carried out at the offspring's 4th, 11th and 16th birthdays in order to chart the development of the mother-child dyads. The 25-year-old young adult offspring will provide data through one-to-one clinical interview. We will assess their current mental state along with past psychiatric history retrospective to 16 years, along with information on employment, relationships, family life, health behaviour through a schedule childhood maltreatment history and interpersonal functioning. Participants will also provide a blood and saliva sample for the examination of neuroendocrine, inflammation and metabolic markers of disease.

PROGRESS IN PAST YEAR

What this research has shown

In the last year I have completed data collection and analysis. Our findings have shown that a mother's depression in pregnancy predicts her offspring being depressed in young adulthood (25 years). Moreover, we find that child maltreatment mediates this association, finding a significant indirect effect between maternal antenatal depression, offspring child maltreatment and offspring adulthood depression. We also observed that maternal depression during pregnancy significantly predicts elevated inflammation in non-depressed adulthood offspring. Furthermore, our results revealed that maltreatment non-depressed offspring showed an elevated cortisol awakening response, whilst maltreated and depressed adulthood offspring exhibited a blunted awakening cortisol profile. We did not find any significant impact of early life insults on metabolic parameters.

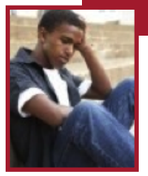
Implications

These findings show the pervasive and detrimental effects of offspring exposure to maternal depression in utero, drawing attention to both the long-term psychological and biological sequelae. Moreover, this data has direct implications for influencing treatment decisions. Treating maternal depression during pregnancy (when contact with health services is already established) could be a unique opportunity to reduce child maltreatment and later depression in the population.

PUBLICATIONS & CONFERENCES ATTENDED

Submitted for publication:
Journal of the American Academy of Child and Adolescent Psychiatry
Child maltreatment mediates the association between maternal depression during pregnancy and offspring depression in adulthood.
Four Conferences presentations
103rd Annual Meeting of the American Psychopathological Association, New York, 2013 - Early life adversity is associated with anxiety and depressive disorders in young adulthood: a prospective longitudinal investigation over 25 years.
20th Annual Psychoneuroimmunology Research Society Scientific Meeting, Stockholm 2013 - Exposure to depression in utero predicts adulthood inflammation. Oral presentation and poster)
Talk at **Perinatal Mental Health; The National Training Course 2013: Preventing Suffering in this Generation and the Next** 2013 - Linking poor mental health across generations: childhood trauma and abuse, London
British Association for Psychopharmacology 2013 Meeting - Effect of child maltreatment on cortisol levels in young adulthood.

EMMA DEMPSTER



DNA METHYLATION AND ADOLESCENT DEPRESSION: AN MZ DIFFERENCES STUDY

PROJECT DESCRIPTION

Depression is a complex disorder with many factors implicated in the disease process. These include certain environmental stressors as well as genetic predisposition. Empirical evidence from studies has implied that genetic risk interacts with environmental stressors to increase an individual's overall risk of developing depression; however the mechanism is largely unknown. Epigenetic processes are one such candidate mechanism whereby the environment can directly have an effect on how genes are switched on and off. Using an existing data set from a large longitudinal twin study (G1219), we have identified pairs of identical twins discordant for depression across at least two time points for whom we also have DNA samples. Identical twins can only consistently differ as a result of environmental experiences specific to each, because identical twins share a common DNA sequence, the study of discordant twins represents an ideal design for investigating the contribution of epigenetic factors to disease aetiology as any differences seen in DNA methylation between twins will be independent of DNA sequence. This study plans to investigate genome-wide patterns of the epigenetic process of DNA methylation in this twin sample in order to explore the possibility that DNA methylation changes are associated with depression.

PROGRESS IN PAST YEAR

We selected monozygotic (MZ) twins from an adolescent twin study designed to investigate the interplay of genetic and environmental factors in the development of emotional and behavioural difficulties (G1219). Eighteen pairs of MZ twins were identified in which one member scored consistently higher (group mean within the clinically significant range) on self-rated depression than the other. We assessed genome-wide patterns of DNA methylation in twin buccal cell DNA using the Illumina 450K HumanMethylation array. Quality control and data pre-processing was undertaken using the WateRmelon package. Differentially methylated probes (DMP) were identified using an analysis strategy taking into account both the significance and magnitude of DNA methylation differences. The top differentially methylated DMP was successfully validated by bisulfite-pyrosequencing and identified DMPs were tested in post mortem brain samples obtained from major depressive disorder patients (n= 14) and matched controls (n=15). Two reproducible depression-associated DMPs were identified, including the top ranked DMP which was located within an intron of STK32C, which encodes a serine/threonine kinase, of unknown function. Our data indicate that DNA methylation differences are apparent in MZ twins discordant for adolescent
We have also generated genome-wide patterns of DNA methylation on an extra 23 pairs that are discordant for "diurnal preference" which is a depression associated phenotype that measures sleep quality The top differentially methylated probe in this analysis was in a gene named SRRM4 which negatively regulates REST (NRSF), a transcriptional repressor of genes required for neurogenesis, This additional study is currently being prepared for publication.

PUBLICATIONS & CONFERENCES ATTENDED

Publications
2014 **Biological Psychiatry**
Genome wide Methylomic Analysis of Monozygotic Twins Discordant for Adolescent Depression
Conferences Presented at:
November 2013 **Epigenomics of Common Diseases**

Donate online JustGiving

www.justgiving.com/psychiatryresearchtrust/

DONATIONS

If you would like to sponsor any of the above events or make a general donation please remember that, if you do not want to donate on line via Justgiving, we are always happy to receive cheque payments (if for a particular event please mark which one it is for). It's always very satisfying to open the day's post and finding a cheque!

PARTY IN MEMORY OF ZAK

Zak Shahdazzle was somebody that I really wish everybody had the chance to meet. She always had the biggest smile and such a warming presence with an ability to lift the moods of everyone she ever met. I was lucky enough to become a close friend to her and it was during this time that I became aware of the complexities of eating disorders and the difficulties in treating them. It was such a tragedy when anorexia took her away from us but the positive effect she left behind is truly everlasting.

It was in the wake of this tragedy that our friend and DJ, Chris, suggested throwing a party, not only in celebration of her life, but also to raise money and awareness for such an important cause. We decided to name the event 'Shahdazzled!' and when it came to booking the club the only available date was her birthday so I think it was meant to be! It was so moving to see so many people come together to make this happen, from people creating décor and designing flyers, to the many DJ's, (including Chris!) who offered to play at the party. There is nothing more she would have wanted than to see all her friends so happy, dancing to the music she loved and having an amazing time and that's exactly how we said our farewell!

The atmosphere and energy of so many friends coming together for Zak is something I will never forget and the fact we were able to raise money for the Psychiatric Research Trust made the night even more special. It was a great reminder to all of us that something positive can still be made out of the most tragic circumstances and I'm so glad the money raised will help combat eating disorders and prevent any more wonderful people like Zak from leaving the world too soon.

I would like to thank Chris Johnston for coming up with the idea and organizing the whole event as a lot of time and effort was put into it and it really paid off! I would also like to thank Janet Treasure for all the amazing work she does, especially for writing the book that really helped me gain an understanding of eating disorders and which helped me immensely in caring for Zak.

Tim Hall

TRUSTEE NEWS

This year we must say one goodbye and two hellos to Trustees.

Mr Jan Kingzett
The goodbye is to Mr Jan Kingzett who has been one of our specialised Finance Trustees since November 2007. Mr Kingzett works for Schroder Investment Management who recently acquired Cazenove Capital Management and who are our investment managers. This resulted in a possible conflict of interests resulting in Mr Kingzett's reluctant resignation. We would like to take this opportunity of sincerely thanking him for all the invaluable advice and support he has given the Trust over the last seven years.

Mr Ben Williams
Our first hello is to Mr Ben Williams who we welcome as a new Finance Trustee. Mr Williams is a fund manager at the global asset management company, GAM UK Limited and will be joining us for his first Finance Trustee meeting this month.

Professor Janet Treasure
Our second hello is to Professor Janet Treasure who many of you may already know as an internationally renowned expert in eating disorders and Professor of Psychiatry at the IoPPN. Professor Treasure was recently awarded a lifetime achievement award by the Academy for Eating Disorders (AED). She is also Director of the South London and Maudsley NHS Foundation Trust's (SlaM) Eating Disorders Unit at the Bethlem Royal Hospital. She has worked as part of clinical and research teams at SLaM and the IoPPN for over 30 years and much of her research has focused on the causes of eating disorders and the translation of these findings into new treatments. These include the development of the first ever self-care manual for bulimia. Professor Treasure is also the Chief Medical Officer for Beat (the main UK eating disorder charity), and was awarded an OBE for Services to People with Eating Disorders in the 2013 New Year's Honours.

Janet Treasure

HOARDING DISORDER

In August of this year, we received a generous donation from Esme Watson, in memory of her sister Lira Gregory. Our many thanks to Esme who we asked to write a small piece for the Newsletter about Lira and her reasons why she chose to donate to hoarding disorder research.

The following is a copy of what Esme kindly wrote for us and it is followed by information, originally written for us by Professor David Mataix-Cols, Head of the Psychobiology of Anxiety and Obsessive-Compulsive Disorders Group, about this often misunderstood condition.

Lira Gregory

My wonderful sister was a hoarder. Nearly every room was full. She was generous beyond belief to whoever asked for help and she was always going to sort it out. Only once did she ask me to help tidy the sitting room as family were going to see her and she kept putting them off, knowing the state of her house. Hoarders know there is a problem within themselves. They will not let anyone touch the clutter because they know where to find each item and they seem to feel safe with their belongings surrounding them.

If only there was a way of understanding the reasons of hoarding - it would be of the greatest benefit enabling to wean hoarders from this sad habit. Research is vitally needed. We who have, and had loved ones, can help by donating money towards the cause and prevention of hoarding.

HOARDING DISORDER RESEARCH

Like most human behaviours, saving and collecting possessions can range from being totally normal to excessive or pathological. Most children have collections at some point and approximately 30% of British adults define themselves as collectors. Hoarding and Compulsive Hoarding are some of the more commonly used terms to refer to an excessive and problematic form of 'collectionism'.

Problematic hoarding is highly prevalent (approximately 2-5% of the population – that is potentially over 1.2 million people in the UK alone) and, when severe, is associated with substantial disability and represents a great burden for the sufferers, their families and society.

Recent research conducted at the Institute of Psychiatry and elsewhere has shown that in most cases, hoarding appears to be independent from other neurological and psychiatric disorders. This means that a large proportion of sufferers may remain undiagnosed and thus not receive adequate treatment.

But all this may be about to change thanks to the inclusion of Hoarding Disorder as a new mental disorder in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the book that contains all officially recognised mental disorders. The DSM-5 is due to be published in 2013.

Symptoms of Hoarding Disorder include a persistent difficulty discarding or parting with possessions (regardless of the value others may attribute to these possessions) due to strong urges to save items and/or distress associated with discarding, resulting in the accumulation of a large number of possessions that fill up and clutter active living areas of the home to the extent that their intended use is no longer possible. For example, some sufferers are unable to sleep in their bedroom or cook a hot meal in the kitchen. Symptoms may also be accompanied by excessive collecting or buying or even stealing of items that are not needed or for which there is no available space.

Whilst some hoarders have good insight into the problems caused by their behaviour, others are completely convinced that their situation is not problematic, despite evidence to the contrary. These sufferers are often reluctant to seek help for their problems, causing great distress to family members. Sometimes, when possessions and clutter spill over to communal areas, e.g. front and back gardens, neighbours may be affected too and councils may be forced to intervene.

The hope is that the addition of this diagnosis in DSM-5 will increase public awareness, improve identification of cases, and stimulate both research and the development of specific treatments for this problem. It will also mean that sufferers should be able to receive help within the NHS.

The causes of Hoarding Disorder are currently unknown and there is a need to conduct research of the highest quality to further our understanding of this puzzling condition. In addition, existing treatments are known to be largely inadequate; clearly, new treatments need to be developed and tested before they can be offered routinely within the NHS. Any donations, large or small, will enable researchers at the Institute of Psychiatry to conduct this important work.