Arrivals and Departures

We are very sorry to see Christine Murdoch, our administrator, leaving us at the end of this month. Christine is moving away from the area. For the last 2 ½ years Christine has been a sympathetic first point of contact for many of you. She reorganised our systems and has kept them running smoothly. We will miss her. At the time of writing, a replacement has not been appointed. Jackie Benjamin will deal with most APP administration.

Dr. Ian Jones returns to the University this month from a year spent at the Virginia Institute for Psychiatric and Behavioral Genetics, at Virginia Commonwealth University, USA where he has been learning more about statistical approaches to genetics. Ian has been looking at mood changes in pregnancy and the postnatal period. Following his return to Birmingham, Ian will be continuing to work on molecular genetic research on puerperal psychosis.

Molecular Genetic Studies

A great deal of work is being done in the lab on samples of DNA given by APP members. Work is focussing on the COMT gene and the serotonin transporter gene. Serotonin is a neurotransmitter which affects mood.

Because so much work is going on in the lab., fewer visits are being made to involve new volunteers in the study. We do intend to contact as many of you as possible in due course but it may be quite some time before you hear from us.

Women who already suffer from bipolar illness (manic Depression) are known to be at particularly high risk of suffering puerperal psychosis. Emma Robertson will starting working soon on a project comparing the clinical information for women who have experienced puerperal psychosis with that for women with bipolar who have not been unwell immediately following childbirth.

Recent Research papers

Long Term follow up study of severe post-natal illness

A study was done of 64 British women admitted to a psychiatric hospital within 6 months of childbirth. They were followed up between 17 and 28 years later and
interviewed and their medical records examined. 75% had had further psychiatric illness, mostly unrelated to childbirth. Further illness was less likely to occur where the puerperal illness had been with the woman's first child, where illness started within 1 month of delivery and where the illness had been depressive.

It should be emphasised that this study wasn't confined to women who suffered puerperal psychosis as we would define it. They looked at all admissions to psychiatric hospital within 6 months of childbirth, not just those where psychosis was diagnosed and started within the first 6 weeks after birth. Cases of non-psychotic depression were also included.

*Long-term outcome of severe puerperal psychiatric illness: a 23 year follow-up study*


**Puerperal Psychosis - analysis of 35 cases**

35 patients admitted with psychosis either immediately or within 6 months of delivery were analysed for various risk factors. First child, previous psychiatric history, antenatal complications, caesarean section, perinatal death and female baby were all associated with an increased risk of psychiatric admission.


**Molecular study of estrogen receptor gene**

This study looked at 2 polymorphisms (variations) in the estrogen receptor alpha gene in a sample of 219 people with bipolar affective disorder (including 26 with puerperal psychosis) and 219 "normal" controls. There was no evidence that these polymorphisms are a factor in bipolar affective disorder. However the numbers with puerperal psychosis in the study were too small too exclude the possibility that the polymorphisms play a part in making women vulnerable to it.

*Molecular genetic studies of bipolar disorder and puerperal psychosis at two polymorphisms in the estrogen receptor alpha gene (ESR1)*

JonesI, Middle F, McCandless F, Coyle N, robertson E, Brockington I, Lendon C, Craddock N


**1st World Congress on Women's Mental Health**

The 1st World Congress on Women's Mental Health took place in Berlin from 27th to 31st March. This was a large international congress with delegates from all over the world. There were a number of presentations relevant to puerperal psychosis which are listed below.
This paper may be of particular interest to our readers. It involved 2 women who both had bipolar affective disorder (manic depression) and were followed through late pregnancy and the 1st postnatal year. One of the women had had a previous episode of puerperal psychosis. A treatment strategy was used consisting of close monitoring of sleep/wake cycle beginning in the third trimester of pregnancy, stimulus reduction in the immediate postpartum period, and prophylactic treatment of insomnia. Both women remained well for over 1 year following childbirth.

The author suggested that disruption of sleep may be an important trigger of postpartum psychosis and that early recognition and treatment of insomnia may
prevent the occurrence of psychosis in women with or without a history of postpartum psychosis.

The study is very small with only 2 women participating so the conclusions do not carry much weight. These women only had a risk of about 20-30% of suffering psychosis so might have remained well in any case. A much larger number of women participating in a study would be needed for any firm conclusions.

**Neurotransmitters.**

Brain cells communicate with each other by sending out chemicals called neurotransmitters.

Neurotransmitters are chemicals which are released from nerve endings and transmit impulses from one nerve cell (neurone) to another neurone or muscle cell.

When an electrical impulse travels down a nerve cell axon it causes the release of a chemical neurotransmitter at the axon terminals.

The neurotransmitter is released from tiny swellings called synaptic knobs at the axon terminals.

The neurotransmitter then crosses the gap or synapse between the two cells, to the surface membrane of the target cell, where it binds to a receptor.

If sufficient target cell receptors are activated by neurotransmitter binding, an impulse is initiated and passes in turn down the target cell’s axon.

The transmitter is then either removed by enzymes or recycled. When this happens the nerve is ready for the next message.
There are many types of neurotransmitters, but the ones that have attracted most interest in researching mood disorders are serotonin and noradrenaline (also known as norepinephrine).

**Noradrenaline (Norepinephrine)**

Noradrenaline or norepinephrine circuits originate in the brain stem, and run to many areas of the brain, including to the limbic system – areas which play a significant part in regulating emotions.

From the 1950s onwards, researchers have put forward hypotheses which suggest that irregular levels or chemical imbalances in these neurotransmitters can lead to depression and mania.

In the 1960s Schildkraut of Harvard University came up with his classic ‘catecholamine’ hypothesis of mood disorders. He proposed that depression stems from a deficiency of chemicals including noradrenaline in certain brain circuits and that mania arises from an overabundance of the substances. The theory has since been refined, acknowledging for instance, that decreases or elevations in these chemicals do not alter moods in everyone. Nevertheless the proposed link between noradrenaline depletion and depression has gained much experimental support.

**Serotonin**

Serotonin-producing cells are found in many brain regions that are thought to play a part in affective (or mood) symptoms: for example, the amygdala (an area involved in
emotions), the hypothalamus (involved in appetite, sleep and libido) and cortical areas of the brain that are involved in memory, planning and organisation tasks.

Abnormality of the serotonergic system has been implicated in a number of human diseases such as depression, migraine, epilepsy, Obsessive Compulsive Disorder, eating disorders and affective disorder (Bipolar Disorder).

The role of serotonin in mood disorders has been investigated for almost 30 years, since Prange, Coppen and their workers put forward their so-called permissive hypothesis. This view held that synaptic depletion of serotonin was another cause of depression, one that worked by promoting, or ‘permitting’ a fall in noradrenaline levels.

Studies have found that depressed patients had a lower level of serotonin than non-depressed patients and a lower number of serotonin cells that are only found in the brain. Further evidence for the role of serotonin in mood disorders comes from the therapeutic response of drugs like Prozac, which acts on serotonin on reducing depressive and anxiety symptoms.

**Anti-depressant medications.**

Anti-depressant medications work by trying to correct this imbalance of brain chemicals. One of the ways to do this is to stop the chemical being recycled, allowing more chemicals to be available in the synapse to ensure a stronger message is passed to the cell, and activity in that part of the brain is increased.

Different types of medications work slightly differently, so :-

Tricyclics block the recycling of serotonin and norepinephrine e.g. amitriptyline (also known as Elavil, Endep, Tryptizol) and clomipramine (also known as Anafranil)

SSRI’s – (Selective Serotonin Reuptake Inhibitors) – block the cycling of just serotonin e.g,Prozac (fluoxetine) and Seroxat

MAOI’s (Monoamine Oxidase Inhibitors) – block the enzyme which breaks down norepinephrine, serotonin and some other neurotransmitters e.g. moclobemide.

At the moment, scientists are not sure why some people respond better to certain types of anti-depressant drugs than others, but research is taking place which may help to understand this phenomena.

**Puerperal Psychosis & Neurotransmitters.**

As discussed in previous newsletters, in the molecular study of puerperal psychosis, we are studying genes for neurotransmitters which are influenced by steroid hormones. We believe that steroid hormones such as oestrogen are likely to be involved in puerperal psychosis e.g. the serotonin transporter gene which is influenced by oestrogen is the site of action for Prozac and other SSRIs.

Emma Robertson
New Zealand Mother & Baby Unit

Last year Professor Brockington spent 3 months working at a Mother & Baby unit in Christchurch, New Zealand. He gave a talk recently comparing the Christchurch Unit with the Unit in Birmingham.

Christchurch is a city of 300,000 people on New Zealand's South Island.

The Christchurch Unit has about 200 mothers referred to it each year compared to almost 600 a year in Birmingham. Both teams have 2 consultant psychiatrists and 2 registrars. In New Zealand they have more psychologists and also have social worker, physiotherapist and dietician. Staff have their own offices. The Birmingham Unit is completely self contained and has 8 beds plus a flat for families. The Christchurch Unit has 7 beds and is shared with a service for anorexia patients.

In Christchurch the waiting time for admission is 4 to 12 weeks compared with 1 to 6 weeks in Birmingham. This is due, in part, to different approaches to the assessment of a patient's needs. In Birmingham a psychiatrist, often accompanied by a medical student, visits the woman at home and assesses her needs. There is no team discussion. In Christchurch, an interview by medical staff is followed by full discussion of the case with all members of the team. This is more thorough and doesn't assume that only doctors have the answers. However the drawback is that it is more costly and leads to delay.

In terms of treatments available:

- Both units offer in patient treatment for mothers to be admitted with their babies
- Both offer therapeutic groups, anxiety management and play therapy
- Christchurch offers more psychological treatments, help from a social worker and all around better teamwork
- Birmingham offers home assessment and, in some cases, home treatment

In conclusion, Professor Brockington thought the relative advantages and disadvantages of the two units were fairly evenly balanced. Christchurch has the major advantage of a multidisciplinary team offering more comprehensive treatment. Birmingham, on the other hand, had speedy home assessment, a better in-patient environment and medical training and research.

National Mental Health Drugs Telephone Helpline

- It is based at the Pharmacy at the Maudsley Hospital in London
- Funded by an unrestricted grant from Zeneca Pharmaceuticals for 1998, 1999 and possibly beyond
- Staffed by experienced mental health pharmacists
- Available 11am to 5pm Monday to Friday (excluding Bank Holidays)
- Open to patients and carers
• Can provide independent advice and information about mental health drugs
• Has been set up the UK Psychiatric Pharmacy Group, which has over 350 pharmacist members

Telephone 020-7919 2999

contacts

You can write to Jackie Benjamin or any other member of the team at

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University of Birmingham
Queen Elizabeth Psychiatric Hospital
Mindelsohn Way
Edgbaston
Birmingham B15 2QZ

You can also reach Jackie by e-mail at j.f.benjamin@bham.ac.uk

Our web page is at http://www.bham.ac.uk/app.

Telephone 0121 678 2354 (Most of the time this is a voicemail service. We will return calls as soon as we can)

Changes of Address

Please let us know if you change your address.

Newsletter by e-mail

If you would be happy to receive this newsletter by e-mail instead of post, please e-mail Jackie.