MIND CAFE
PSYCH-E-OPINION
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ISSUE 06

In this issue...

Mindcafe needs your help!

In five short months *Mindcafe's* readership has swelled to well over 1,500 monthly readers nationwide. We wish to spread our free service to your colleagues around the country and for this we request your assistance.

Your help in growing the readership will help keep *Mindcafe* a free service for all. The request is a simple one—please forward this issue to as many colleagues as you can and encourage them to *click here and register online* to receive *Mindcafe's* unique free CPD service. Whilst we have the support of so many well regarded clinicians, we rely on networks and word of mouth to really grow the service for the benefit of our Mental Health Community.

We break for December and January and will have the next issue out in the last days of February as usual.

Let me take this opportunity to congratulate you this year for working towards the shared noble goal of better mental health for the Australian people.

It is something we should be proud to be part of. *Mindcafe* wishes you a wonderful and joyous festive season and an enriching year ahead.

We thank you for your help with supporting and growing the *Mindcafe* service by spreading the word...

Dr Ranil Gunewardene FRANZCP
Psychiatrist/Clinical Director/Director *Mindcafe*

Worried about risk? Relax!

When you think about risk, what worries you most?

A couple of weeks ago I spoke at the annual Redbank Conference at Sydney's Westmead Hospital. This year the topic was 'risk' and the organisers asked registrants to provide an answer to the question above. They gave me a copy of everyone's responses.

Perhaps these were a group of particularly Nervous Nellies, but plenty of people seemed plenty worried.

From what I could tell, most of the concern stemmed from the idea that it was somehow possible to make a useful assessment of a patient's likelihood of future harm, and that if, as a clinician, you failed in this task and if the patient were then to kill themselves or hurt someone else, you would be in someway responsible, or at least, that might be how a court would see it.

I should say that relatively few of the attendees were psychiatrists. Most were listed as nurses, case managers, psychologists or social workers, but the same sorts of worries affect Psychiatrists too, so I suspect. It is unpleasant to be anxious at work, and anxiety can get in the way of people's ability to provide the best care for our patients. Therefore, I think, we have a duty to reassure our colleagues that risk is not a bogeyman.

Fortunately reassurance, at least regarding one's own responsibility, is easily to hand. When people worry that they will be personally responsible for disaster based on a their risk assessment, they assume that it is possible to conduct a useful assessment of the likelihood of future harm. In reality, it is not. The low base-rate of suicide and serious violence, at least in civil psychiatric populations, means that no matter how good our risk assessment tools get, they will always be inadequate to the task. So-called risk assessments simply have too many false positives and too many false negatives.[1-3] No one should use them to guide their clinical interventions.[4]

If you can't do something, you can hardly hold yourself responsible for not having done it properly. Providing good clinical care has nothing to do with assessing risk. It's about carefully coming to grips with our patients' predicaments and then working with them and their families to try to meet as many of their goals as possible. I see suicidal people at Westmead everyday. I never do a risk assessment. I know that I can't. I just stick to what (I like to think) I can do—providing good clinical care.

When it comes to the attitude of the courts however reassurance is more difficult to provide. Here we are somewhat at the mercy of forensic 'experts', and some of them are still to come to grips with the maths on this thing. One thing is clear though. If you are going to claim it's not possible to do a useful risk assessment (and you should, because its true), it will (not unreasonably) confuse a court if you did one as part of your assessment. This means staying away from those dinky tick-a-box risk assessment forms.

Listen to your patients. Carefully document your assessment. Provide a detailed justification for the plan you put together. That will stand you in much better stead.

You can't tell who might eventually come to harm, but you can do your best to protect all your patients.

Christopher Ryan FRANZCP, Consultation-Liaison Psychiatrist and Senior Clinical Lecture, Westmead Hospital and the University of Sydney, Westmead NSW

References on page 02.

Dousing depressive symptoms with anti-inflammatory medicines?

Effect of Anti-inflammatory Treatment on Depression, Depressive Symptoms, and Adverse Effects: A Systematic Review and Meta-analysis of Randomized Clinical Trials. Köhler O, Benros ME, Nordentoft M, Farkouh ME, Iyengar RL, Mors O, Krogh J. *JAMA psychiatry* 2014 Oct 15. doi: 10.1001/jamapsychiatry.2014.1611. [Epub ahead of print]

Depression in conjunction with arthritis or other chronic pain is a problem that occurs frequently in both general practice and psychiatry populations. It would be tempting and simple enough to explain this association as a consequence of the impact of chronic pain on mood. However another intriguing possibility is that inflammation is a common element in the pathophysiology of both arthritis and depression. If not all depression, this may apply to at least some relatively common types of depression. What if anti-inflammatories could act as augmenting therapy for depression?

The idea of reducing inflammation as a neuroprotective strategy is not new. It ties in with ideas of oxidative stress and inflammation acting at a cellular level to produce the structural and functional brain changes associated with chronic depression. To date most studies of therapies aimed at reducing this process such as fish oils and antioxidants have been underwhelming. A transatlantic collaboration led by Danish researcher Ole Köhler decided to examine how the direct treatment of inflammation affects mood symptoms.

Anti-inflammatory analgesics have become a mainstay in the management of arthritis and chronic pain conditions. As such there are many patients taking antidepressant medications and anti-inflammatories concurrently. Over time contradictory reports have emerged on the role anti-inflammatories might play with respect to depression. One of the papers published using the STAR D data suggested that the presence of anti-inflammatories diminished the effectiveness of antidepressant therapy[1]. Similarly another recent paper suggested that anti-inflammatories may oppose the effective antidepressants in animal and human models[2]. Conversely there are now longitudinal data suggesting that patient on anti-inflammatories have a reduced risk of depression and Alzheimer's[3,4]. There have also been several studies (the best of which have been included in this meta-analysis) which have linked concurrent anti-inflammatory therapy to a reduction in depression severity.

With this mixed picture of the relationship between anti-inflammatories and mood it was pleasing to see that this group of researchers have explored this relationship in a detailed way. Using Cochrane collaboration like methodology this meta-analysis of randomised double blind trials represents the most carefully done research to try and gather together findings on a possible role for anti-inflammatories in treating depression. However the decision to use trials including patients with concomitant diseases and those with depressive symptoms or a diagnosis of depression adds a naturalistic, 'real world' clinical element to the study.

The authors first reviewed the evidence of links between inflammation and depression. These links include evidence of altered cytokine functioning in depression, increased associations of depression with autoimmune conditions and the demonstrated depressogenic effects of pro-inflammatory agents. They were interested in addressing the effects of anti-inflammatory co-treatment on depressive symptomatology. They noted two previous meta-analyses of celecoxib add on therapy and NSAID monotherapy suggested benefit from anti-inflammatory treatment but did not assess bias. This trial was larger and did consider bias.

Although outcome assessments were blinded the researchers did note a high risk of bias in most of the included trials.

The group sought to examine not only whether anti-inflammatories assisted in the management of depression and depressive symptoms, but whether they were associated with adverse effects. They examined the evidence for all anti-inflammatories as a group (14 trials including 6,262 patients) and each major class to tease apart any benefit or risk that may ensue. Aspirin and other classical anti-inflammatory medications plus selective COX-2 inhibitors (celecoxib) were grouped as (NSAID's) with 10 trials being included. Most were 6–12 week trials on patients with co-morbidity such as active osteoarthritis. One examined healthy individuals with a family history of Alzheimer's on NSAID monotherapy for a year. A further four trials looked at monotherapy with a disparate group of cytokine inhibitors including infliximab and entanercept.

The pooled effect suggested that anti-inflammatory treatment was associated with significantly reduced depressive symptoms whether one was considering patients with depression or simply depressive symptoms. This effect was significant for NSAID's but only a non-significant trend for cytokine inhibitors. The effect was strongest and most reliably demonstrated in the subgroup who took celecoxib as an add-on therapy, showing improved depression remission and response rates. Revisiting the literature, the authors noted that these antidepressant effects were;

1. large and thus clinically relevant
2. greatest in patients with inflammatory conditions or raised pro-inflammatory markers
3. independent of pain relief

Although these findings are preliminary, this suggests clinical utility, a target group for anti-inflammatory treatment and a preferred agent.

Importantly no increased risk of gastrointestinal or cardiovascular adverse effects was reported with NSAIDs over six weeks. However not all trials reported adverse effects and, as these problems may emerge with chronic treatment, this may be too short a time frame to fully assess risk of co-treatment.

Watch this space as we consider whether anti-inflammatory medications and COX-2 inhibitors in particular, make their way into depression treatment algorithms.

Josh Geffen FRANZCP

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Applied mindfulness

In the last article (refer to Mindcafe issue 04, September 2014) we explored how depression can be looked at as a disorder of attention. As such it makes a lot of sense to learn to manage attention better, not only for managing depression but also for other mental health problems and for dealing with the pressures of daily life. This article will be the first in a series to explore how to apply mindfulness, but before we consider what it means to be mindful, we will look at what it means to be unmindful.

Being unmindful

Have you ever been waiting for the weather report and when the temperature forecast is given you don't hear what the weatherperson says? Have you ever driven your car from point A to point B but you don't remember much of the journey? Do you ever find yourself in a conversation with somebody and find that you are not taking in a word of what they are saying? Well, that's unmindfulness and although we often take such experiences for granted, our tendency not to be present to our life has many implications. Among other things, unmindfulness means:

- wasting time
- missing important information
- risk of accidents
- communicating superficially
- vulnerability to stress
- poor mental and physical health

When we are unmindful the attention unconsciously unhooks from whatever the priority was at that moment and wanders off to something irrelevant. Looking at the pace of modern life, multitasking and the constant bombardment of information, it would not be unreasonable to think that we have created a world that literally drives us to distraction.

What's the remedy?

Perhaps the simplest way to express what mindfulness is would be to say that it is a practice in paying attention. It has three main aspects:

1. knowing where our attention is,
2. prioritizing where it needs to be,
3. and for the attention to go there unless a more important priority comes along.

More sophisticated terms for this include 'attention training' or 'attention regulation'. We can pay attention to our minds and bodies as well as the environment but the attitude with which we pay attention is just as important from a mindfulness perspective as the paying attention itself. The mindful attitude includes things like openness, curiosity, non-reactivity and acceptance. Why this attitude is important will become increasingly obvious.

You could take a few moments to notice what your body is experiencing right now. Notice its posture; how it feels; what it's doing. Notice what is going on in your mind; its thoughts, attitudes and emotions—you don't need to do anything about it; just notice it. Notice whether things that take place in the mind have effects on what happens in the body even if those changes are very subtle. You may come to notice the mind-body interaction. It's going on all day, every day. Notice also what is happening in the environment right now—the sounds, movements, smells, and sensations. Just be the impartial observer of your inner and outer worlds and how they merge and interact. This is not dissociation but observing with interest and non-attachment to what is being observed.

Mindfulness can be described as:

- a form of meditation (formal practice),
- a way of living with awareness (informal practice),
- and a foundation for psychotherapy (cognitive practices).

Mindfulness is not Buddhist.

Formal practice—mindfulness meditation

All meditation practices could be described as mental disciplines involving attention regulation although they vary in the focus of attention they use and the aim of the practice. The body is commonly used in mindfulness meditation. Why? Well, it's always in the present moment—never the past or the future. As the body communicates with the outside world through the senses in mindfulness meditation practice, the attention is grounded in the present moment by using the senses. This brings the mind out of its distracted preoccupation with a past and future (default mode). Any of the five senses could be used as a gateway back to the present moment. Hence, in everyday language we say 'being in touch', 'being sensible' or 'coming to our senses'.

Mindfulness is not Buddhist. There is nothing particularly Buddhist about the present moment, consciousness, the body or the mind. Having said that, practitioners from the Buddhist tradition have made an enormous contribution to the research, application and teaching of mindfulness. We don't need to have an affiliation to any particular wisdom tradition to apply and derive benefits from mindfulness. Indeed, most people come to mindfulness for pragmatic rather than spiritual reasons. We all need awareness no matter what our philosophical attitudes are.

Although mindfulness meditation is primarily a practice in awareness, because relaxation is a common side-effect many think of it as a relaxation exercise. That is understandable, but it is also a potential problem. If we get preoccupied with the goal of relaxing we may find ourselves becoming extremely tense and frustrated if the practice does not unfold the way we expect it to. Aiming at a goal, as desirable as it may be, is the antithesis of mindfulness practice. A goal is inevitably in the future, whereas mindfulness is all about the present. Better is to be mindful of both pleasant and unpleasant experiences as they come and go. If we learn to be at peace with that, or at least are less reactive to it, then anxiety, pain or other unpleasant emotional or physical experiences will agitate us less when they're present. This helps us not to amplify their impact.

The formal practice of mindfulness meditation refers to setting aside other activities of the day to practice being still and mindful. It is really the cornerstone to being able to apply the informal and cognitive aspects of mindfulness more effectively.

Punctuation marks are like little spaces between one thing and another. Just as a book without punctuation makes little sense, a day that is not punctuated makes no sense either. Mindfulness meditation is like the punctuation marks. Meditating for 5 minutes or more can be compared to punctuating our day with a 'full stop'. Short mindful pauses of anywhere between a few seconds to a few minutes can be compared to 'commas'. The more we practice it the better it will be and the more that it will reveal. If you don't have a strong motivation then a good 'starting dose' for those who are new to mindfulness meditation is to practice for 5 minutes twice daily. Before and after our working day are good times, preferably before food because after food is a low-point for the metabolism and sleep can occur more easily then. The duration of practice can be built up to 10, 15, 20 and even up to 30 minutes or longer depending on one's time availability, needs and commitment.

Here are some links and resources for you to get started with a mindfulness meditation practice.

- www.monash.edu.au/counselling/mindfulness.html
- The Centre for Mindfulness at the University of Massachusetts
- The Centre for mindfulness at Bangor University
- UCSD Center for Mindfulness

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- The New Psychology of Depression by Oxford University: Podcast Series (MBCT)
- Smiling Mind: http://smilingmind.com.au/
- Mindfulness with Jon Kabat-Zinn
- Wellbeing Series May 2011 Part 2: Main Introduction
- Oxford Mindfulness Centre YouTube Channel

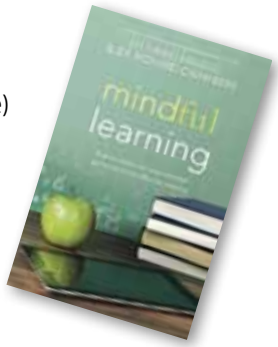
Books

– by Jon Kabat-Zinn

- Full Catastrophe Living
- Wherever You Go, There You Are
- Mindfulness for Beginners
- The Mindful Way Through Depression (with Zindel Siegel, Mark Williams and John Teasdale)

– by Craig Hassed

- Know Thyself
- Mindfulness for Life (with Stephen McKenzie)
- Mindful Learning (with Richard Chambers)
- The Mindfulness Manual



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Women's Mental Health: The Pill and Mental Disorders

Have you thought about the potential effect of the oral contraceptive pill on your female patients' mental health? Possibly not—since psychiatrists generally do not take extensive histories about contraception, and many practitioners tend to dismiss patients' own observations about links between 'The Pill' and mood. However, the link may be stronger than you imagined and certainly the patients' observations warrant validation.

'The Pill' has been in common use since the 1960s and revolutionised many aspects of society. Women became free to control their own reproduction and along with the social changes achieved by the feminist movement of the 60's and 70's, women's roles changed dramatically.

However, over the several decades since the pill was first produced, many women have noted a correlation between lowered mood and the use of certain types of the pill.

Research in this area is still inconclusive and results of previous studies have been contradictory (see Oinonen and Mazmanian, 2002), and hampered by methodological inconsistencies. But despite the wide discrepancies in results, it is apparent that a significant sub-group of women do continue to experience contraceptive-associated negative mood changes. Oinonen and Mazmanian propose that 'specific individual differences and contraceptive-related variables' must mediate the relationship between contraceptive medications and mood and also give consideration to how contraceptives influence mood over the course of the menstrual cycle. Yet little research over the past decade has attempted to ascertain what exactly these variables may be, such that still 'we have no way of predicting which women are likely to experience adverse effects of contraceptives on mood...nor which contraceptive formulations are more likely to be responsible'.

Not all oral contraceptives (OCs) are the same. We speak about 'the pill' as if it is one medication, when in fact there are many variations. All contain synthetic analogues of estrogen and progesterone with various side effect profiles. As the estrogen component is similar in the majority of brands (ethinylestradiol), the progestin component is responsible for much of this variation (Singh 2013). Older first and second generation OCs contain progestins structurally related to testosterone (e.g.levonorgestrel, norethisterone), with corresponding androgenic side effect profiles; whereas newer third and fourth generation OCs contain progestins more closely resembling progesterone itself (e.g. cyproterone acetate, drospirenone) with neutral androgenicity or even anti-androgenic effects.

Our research has revealed that certain types of progesterone in the combined oral contraceptives (COCs) are often correlated with irritability and lowered mood in women who have a heightened sensitivity to hormonal factors (Kulkarni 2005). In particular, progesterone-only pills or implants seem to precipitate or perpetuate dysthymia. Similarly, low dose estradiol pills also seem to precipitate lowered mood possibly because of the lack of balance with the progesterone in terms of CNS impact.

To further complicate matters, the condition of 'premenstrual dysphoric disorder' (PMDD) is a severe form of cyclical depression experienced by some women. It is typically treated with the combined oral contraceptive pill. The idea here is to stabilise the cyclical fluctuations of the gonadal axis and hence improve mood. However, if a COC is used that has a form of progesterone that worsens the depression then overall the woman's depression worsens.

From my clinical experience I suggest the following practice tips:

1. Take a detailed history of contraceptive use. In particular get specific information about the type of oral contraceptive used (most practitioners will usually note that 'the pill' was taken for a while), the length of time that each OC was used and note any observations about irritability, lowered mood during its use.
2. Often women observe important improvements in mood once they stop taking that form of OC, so it is important to take a history of all contraceptives used.
3. Discuss her contraceptive needs with her and work with her General Practitioner to use a contraceptive that does not worsen or cause depression.
4. Check for dysthymia symptoms related to using the pill, not just major depressive symptoms
5. The newer forms of oral contraceptive tend to have better forms of progesterones in terms of mood impact

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6. The lower dose estradiol pills tend to be worse for mood
7. The progesterone only pills and in particular implants tend to worsen mood.
8. Be aware that anger, irritability and hostility are usually the early symptoms of depression that can be worsened by the COCs.
9. Women with a history of trauma in their backgrounds tend to be more sensitive to mood effects of the COC.
10. If your patient says that she feels worse on certain brands of the pill—believe her.

We anticipate publishing our latest pill study results in the very near future and hope that this will help psychiatrists and other doctors when treating women suffering with depression.

Professor Jayashri Kulkarni FRANZCP
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If your patient says that she feels worse on certain brands of the pill—believe her.

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Monkey Luv demystified...

well somewhat anyway.

Build it and they will come...

I can remember a biology teacher exclaiming that a recent zoological discovery headlined that dolphins mate for pleasure and not just for the procreation of the species. I replied with 'Don't all animals with a brain?'. Animals enjoying 'mating' a neural truth. It's simple really: it feels good. Our neuro-chemistry is designed to make sex feel good so that we keep looking for the opportunity; but let's talk about 'attraction': the drive towards sex, for a moment...

There is a reason certain people are considered universally attractive: Symmetry. Approximately one percent of people fit into this criteria: they have facial and physical symmetry and in the world of genes this is the 'mother lode' because symmetry means good gene stock and that all leads to healthy offspring. No surprises, therefore, most of these people are in movies, splashed across magazine covers, harassed by paparazzi, serial dating and running for presidential selection, and the rest of the 99 percent can't get enough of them. Sigh...

Robert Sapolsky, a neuroscientist and primatologist, from Stanford University would describe himself as having his fair share of asymmetry but that didn't stop him trying to find out what sexual behaviour is all about. Sapolsky supports the 'feel good hypothesis' of mating. Certainly he acknowledges that the secondary benefit to mating is that individuals and species genes ultimately benefit by way of populating the globe, but this is not why we mate. Sapolsky introduces the dichotomy between distal and proximal explanations of behaviour. For example, distal cues are the long term, underlying explanations for why a behaviour happens where proximal cues are the short-term, nuts and bolts explanations of why a behaviour happens. Sapolsky correctly states that most behaviour is driven by proximal cues and that 'never is this more the case than when thinking about the motivation for sexual behaviour'. Behaviours that are evolutionary critical and result in great cost to the organisms, the motivation to participate in that behaviour cannot be abstract or delayed. Sapolsky uses the perfect example of this 'imagine how few elephants there would be on earth if elephant sex were motivated purely by the cognitive recognition that, do this, and, shazzam, two years later some kid pops out'. Hence, mating behaviour must be driven by proximal cues and therefore chemically; mating feels good.

Looking at the chemical responses to sexual activity help to better understand why proximal cues are so important for desired sexual behaviour. According to informative studies from Kirkpatrick et al, Winston, Buss and Sapolsky, our bodies are a chemical-mating-machine designed to optimise the feel good nature of a sexual encounter. Makes sense to make mating feel good in the hope that conception will take place eventually, right? Well it seems our brain (and bodies) are rather selective on that front.

Firstly, we are capable of detecting incredibly subtle asymmetries in eyes, ears, wrists (yes wrists), ankles and the good old hip-to-waist ratio. As explained earlier such symmetry in these areas signal optimal genes and organism health. What we are also very good at doing is detecting subtle asymmetry in another's genetic load by way of chemical cues such as pheromones. Yes even your pheromones give your asymmetry away. Seems us homo sapiens are as gifted in 'sniffing out defects' as other animals after all. You're not consciously aware of these said pheromones, but your brain (and body) is. It's best to explain these remarkable evolutionary phenomena by way of examples using the concepts introduced earlier.

Example one: asymmetrical male meets symmetrical girl. Asymmetrical male has small shoulder girth, average height, uneven ears, uneven ankle size but has a nice expensive car, big expensive house, high paid important job, no dependents, loves animals and is an overall nice guy. Symmetrical girl has hip-to-waist ratio of 0.7; even eyes; ears that start and finish evenly and nice even wrists (it actually doesn't matter what job, car, house or personality she has). Symmetrical girl thinks 'nice assets' and asymmetric male thinks 'nice assets' and they embark on a sexual encounter. Then the body takes over. Asymmetrical male body detects symmetry of female and immediately supersizes sexual activation by way of new sperm production and warming the integral body parts to optimal temperature and hence performance. Female body detects asymmetry (despite noted distal cues) and immediately cools things down, her hormone production sets about protecting any viable ovum from fertilisation by way of sperm-toxic chemicals. Her body temperature drops to an unwelcoming temperature and her ovaries shut shop. Overall not great conditions for conception but they will have a nice time nonetheless because it feels good and will be encouraged to keep seeking same behaviour, most likely with each other if distal cues are of interest to girl. This example works for the converse. Example two: Symmetrical male meets symmetrical girl. Symmetrical male has broad shoulder girth, is tall, even eyes, strong jaw line with perfectly placed ears and strong even wrists. Symmetrical girl is same as example one. Symmetrical male thinks 'nice assets' as does symmetrical girl and they embark on a sexual encounter. Then the body takes over. As described in example one the male body goes into sexual super drive. The female body responds in kind: her body temperature rises, ovum are released and a welcoming committee of chemicals ensures a ovum-sperm introduction is made. Overall perfect conditions for conception and they will most definitely have had a feel good time and will no doubt be chemically driven to repeat the behaviour again and most likely with each other.

So at a chemical glance; child-bearing hips, symmetrical faces and limbs and buffed-up alpha bodies gives us vertebrates the chemical hots and provide particularly strong proximal cues towards mating, favouritism, blind rapture, or as Sapolsky aptly puts it 'listen with rapt attention to someone's raving gibberish, just because they have gorgeously symmetric wrists' (we've all been there right...) but are we really that chemically determined? The quick answer is yes and no. When it comes to proximal cues then the answer is yes because this combination is chemically powerful; but when it comes to distal cues the answer appears

to be no. It seems the quality of a relationship is also important. Here is where our neo-cortex finally makes an appearance. It seems when people are contemplating long term, child rearing, home making futures it makes neo-cortex sense to choose a mate that is kind, loving, capable of being a good parent, reliable, has good financial prospects and who remembers to put the cap back on the toothpaste. This redeeming feature of our neo-cortex is that despite the limbic-symmetry-drive we, men and women equally, seem to prefer the long term company of other people that are kind and who love us. Now love is culturally defined so we won't open up that can of semantics here, rather, I'll qualify this by saying; whatever you deem love is you'll be looking for it in others when it comes to long term relations. Does it seem the heart can be a proximal cue too?

All this reminds me of a quote from Howard Hughes, renown for dating some of the words most beautiful women yet he never settled down with them. During a rare interview this observation was enquired about to which he replied 'It doesn't matter how beautiful they are, someone somewhere is sick of their shit.' I digress...

Going back to distal cues, remembering they are the long term underlying explanation for why behaviours happen: interestingly there are examples of this in the animal kingdom (particularly with birds and primates) where non alpha (or asymmetric) males set up an elaborate home or area of turf and then waits for a symmetrical female to notice. He is more than happy to accommodate the offspring of the alpha males because in between her occasional flings he gets to mate with her and have his genes transfer into their offspring. She is equally happy to hang around because she gets a lovely home, food on the table and a loving father to the children. In an interesting social experiment, Winston selected five unattached females of varying age and gave them a lunch voucher to a London cafe with simple instructions: rate the males from 1 to 5 (1= not attractive and 5=very attractive) who pull up in the loading zone outside the cafe. Winston then borrowed a beat up old car, wore a torn jumper and tousled his hair. He pulled up outside the cafe and walked past the females. Winston then borrowed a Bentley car, Armani suit and had a professional do his hair and nails. Again he pulled up outside the cafe and walked past the females. Not surprisingly, the females all rated the 'Bentley Winston' as more attractive. What would have made this study more accurate is if Winston had Fabio pull up in the old car. I suspect the ratings would have shifted dependant on female age and hence life stage.

Going back to the birds and primates distal manipulation I mentioned earlier: there is the obvious catch— the female still gets to mate via proximal cues and hence have genetically healthy offspring to jettison the species. Infidelity is not an absent behaviour to us homo sapiens, so does this mean we may still be part of the animal kingdom after all? Where are those studies showing infidelity occurs primarily as proximal cue drive? In the round filing draw perhaps.

Demystifying sexual attraction for the one percent of symmetrical people out there seems simple enough: proximal cues, symmetry, nice genetic lode—the trifecta for sexual attraction. As for the remaining 99 percent, they are left with this intriguing reality: who they want to grow old with (neo-cortex) and who they want to leap into the sack with (limbic system) are two different people. No surprises there is a call for psychotherapy and generations of cognitive therapies. With globalisation and the expanding gene pool, effective resolution of these competing cue drives are needed more than ever, not to mention what does this all mean for future genetic health and viability of our species? Meanwhile, don't panic. If you can't be accepted for your genetic prowess, then buy the big house and sports car instead...and wait.

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The Three 'D's—Dementia, Delirium, and Depression

In older persons, the issue of whether the patient is suffering from Dementia, Delirium or Depression is one of the most common conundrums in geriatric psychiatry. It is very difficult to find any psychogeriatric text that does not cover the issue, which invites one to ask as to why it is such a frequent question. I shall therefore start by showing how the question can be answered, and then explain why the question cannot be answered.

How to tell between the 3 'D's.

Many authorities have provided comparison tables along the line of indicating that there are varying time courses between the three conditions and degrees of fluctuation. Gagliardi (2008, Virtual Mentor) provides a unique and useful insight, in that she takes into account the patient's subjective experience and performance with ADLs as well:

	Depression	Delirium	Dementia
Onset	Weeks to months	Hours to days	Months to years
Mood	Low/apathetic	Fluctuates	Fluctuates
Course	Chronic; responds to treatment	Acute; responds to treatment	Chronic, with deterioration over time
Self awareness	Likely to be concerned about memory impairment	May be aware of changes in cognition; fluctuates	Likely to hide or be unaware of cognitive deficits
Activities of Daily Living (ADLs)	May neglect basic self-care	May be intact or impaired	May be intact early, impaired as disease progresses
Instrumental Activities of Daily Living (IADLs)	May be intact or impaired	May be intact or impaired	May be intact early, impaired before ADLs as disease progresses

The fluctuating consciousness of delirium is well known to be a key delineating feature between it and the other two conditions, but the fluctuating mood issues are also significant to note.

Why we can't tell between the 3 'D's.

1. *The three conditions are usually assessed and managed by conflicting authorities*

   Dementia refers to an irreversible, progressive illness. Differential diagnosis of the individual subtypes still has an arguably academic role, as there are no interventions which have been demonstrated to change the course of any form of dementia. The clinicians who are used to dealing with dementia are often therefore pragmatic individuals who are focused on functional optimisation and maintenance of integrity.

   Delirium is a medical emergency, with a high rate of mortality if untreated, and refers to an acute confusional state with an underlying potentially reversible cause. This then brings a wide range of clinicians who are interested in treating reversible causes within their areas of interest, but start from a vague position of nonspecific confusion. When one considers that some studies find that as many as 30% of bedside diagnosed deliria do not have an identifiable underlying cause, it is no surprise that it is a contentious diagnosis with a wide degree for intepretation. Depending on the classificatory system used, a patient may be diagnosed as having delirium as a result of fluctuating consciousness and attention alone, or only after a contributory 'medical' cause is identified. It is rare to find a consultation-liaison psychiatrist who hasn't built up a considerable armamentarium of defences regarding their physician colleagues' opposition regarding a delirium diagnosis.

   Depression is an entirely reversible psychiatric condition with considerable overlap into 'normal' functioning. Clinicians who achieve considerable satisfaction from the recognition and treatment of mental health issues may not have much awareness of organic pathophysiology, adding further to the aforementioned conflict. There is also the problem that there is no real end to treating a suspected depression—one can easily progress along the therapeutic ladder from SSRIs to combination antidepressants and electroconvulsive therapy whilst being entirely wrong about the diagnosis. Psychiatry is also rife with the 'watch and wait' approach of responding to diagnostic dilemmas by monitoring for progress—which can be fatal in the setting of confusion between these three.

2. *The three conditions often coincide*

   The patient who undergoes dementia is often aware of their progressive deterioration, regardless of whether the diagnosis has often been shared with them. The chronic stress of progressive inevitable loss easily can create a secondary dementia. They additionally may have reduced self-care leading to vulnerabilities to infections and electrolyte disturbances, predisposing to deliria.

   The delirious patient is in an acute confusional state—by definition, this allows them to manifest every potential psychiatric phenomenology there is, from mania to psychosis to depression. A sudden blunting of affect certainly permits a diagnosis of a depression secondary to delirium—as, although we historically consider psychiatric diagnoses as being 'functional', this by no means prevents them from having organic causes.

   The depressed patient certainly can reduce their self-care, primarily their intake of food and fluid. Other misadventures, such as medication noncompliance or attempted overdoses, can also lead to them developing delirium.
   Plus, while delirium and depression should not initiate cognitive impairments, there are meta-analyses nevertheless identifying an association between the two (Snowdon, Oct 2011, *Australasian Psychiatry*). These have been thought to represent misdiagnoses, which is still not comforting.

3. *Treating one condition can compromise some of the others*

The treatments for most dementia (cholinesterase inhibitors) improve some forms of delirium. However, many studies (particularly van Eijk's landmark 2010 Lancet study) have found that they can increase confusion and even mortality.

Many guidelines advocate for the use of low dose antipsychotic agents for reducing the severity and length of delirium—studies are actually very mixed regarding this query (compare Hawkins et al 2013 with the 2014 Hope-ICU trial), however as antipsychotic agents sedate and dull all patient responses, they can be lethal for masking severe depression masquerading as delirium.

All antidepressants (with the exceptions of reboxetine and mirtazapine) can cause prominent hyponatraemia in vulnerable elderly patients, leading to a spiral of delirium and increasing confusion in a patient having escalating treatment for a suspected functional illness, who therefore would not be getting regular blood tests to monitor for same.

Therefore, the question will probably never be easily answered and will remain in psychogeriatric textbooks for some time yet. It is possible, however, to take the above into account to create a pragmatic approach for the ambiguously presenting patient:

1. *Start with the SQID*
The 'Single Question in Delirium' (Sands 2010, Palliative Medicine) simply involves turning to the patient's friend or family and asking 'Do you think [name of patient] has been more confused lately?' While it seems ridiculous, it performs remarkably well against many other instruments, and responds sensibly to the key issue of monitoring for fluctuating confusion by using collateral information.
2. *Delirium screen*
For reasons that are not clearly known, elderly patients are remarkably sensitive to urinary tract infections, even those which may not appear 'positive' on urine testing (low leucocyte count and lack of a clear bacterial growth).
In addition to this, a recommended minimum screen is—
FBC, EUC, B12, Folate, LFT, TFTs, CMP, Serology (Syphilis, Hepatitis)
An EEG is in fact reasonably diagnostic for delirium by identifying new onset slow waves; however differentiating this from encephalopathy related to dementia is difficult and does not usually add more to the assessment process than the bedside assessment.
3. *Geriatric Depression Scale*
The 15 point Geriatric Depression Scale has very high sensitivity and specificity for assessing for depression in patients capable of communication—different authorities advise 5 or 8 as cut-offs, but sensibly using it in conjunction with a bedside mood assessment greatly assists with diagnosis.
4. *Do not test memory via any cognitive testing*
No bedside standardised brief test of cognitive screening will adequately distinguish the cognitive deteriorations associated with the 3 'D's. Even lengthy neuropsychological testing can often yield ambiguous results. Dementia should be seen as a diagnosis of exclusion only.
5. *Re-evaluate the patient repeatedly, particularly following an intervention*

The three 'D's pose an interesting challenge as an intersection of culturally and phenomenologically distinct entities between which misdiagnosis is common and associated with potentially catastrophic outcomes. It is worth recollecting that 'Pseudodementia' originates from Leslie Kiloh's 1961 paper, as part of his effort to educate the community to think about conditions that are potentially treatable, rather than having therapeutic nihilism as the rule. None of our patients would want to know that their clinicians are having trouble understanding them; but they'd probably forgive us if they knew we were doing everything possible to find something treatable.

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Changing health behaviours: Models, predictors and the importance of affect

Recently Professor Mark Connor was in Australia as a keynote speaker at the Annual APS Conference in Hobart. His primary area of research is in health behaviour models and determining behaviour change factors. Motivating clients to change certain health behaviours in addition to managing presenting psychopathology has long been a difficulty in psychological practice. Health behaviours that may be seen in clinical practice range from increasing physical activity, smoking cessation, alcohol or substance reduction or cessation, and improved diet or weight loss. This is frequently in addition to comorbid psychopathology that may be the primary cause for treatment seeking. It is therefore important to understand the underpinnings of changing health behaviours so as to work with clients towards their goals.

There is much research into the psychological determinants of health behaviours, particularly health cognitions. There are a number of, often overlapping, theories used to explain health behaviours such as the Social Cognitive Model, Theory of Planned Behavior, Health Belief Model, and Protection Motivation Theory (see Connor, 2010). These models suggest that the thoughts and feelings regarding a behaviour are predictive of whether that behaviour is subsequently performed in the future. Some models, such as the Theory of Planned Behaviour, suggest that our behaviour is determined by intent, which is determined by subjective norms, perceived control of behaviour and attitudes. However such models focus almost exclusively on cognitive aspects of health behavior excluding other aspects, such as the role of affect. A study by Lawton et al (2009) found that affective attitudes were stronger predictors of intentions and behavior when compared to cognitive attitudes. Much of the research highlights the importance of affective attitudes in determining intention and action for health behavior change. In his paper, Connor (2013) examines the literature to assess the role of affective influences, such as affective attitude and anticipated effect (such as guilt or regret), on health behaviors in the context of models such as the Theory of Planned Behavior. Current literature reinforces the importance of focusing on affective influences when working with clients on health intentions and motivating behaviour change. The research also found that a combined approach of focusing on affective attitude and anticipated affect did not add value.

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A study by Lawton et al. (2009) found that affective attitudes were stronger predictors of intentions and behavior when compared to cognitive attitudes.

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