

Journal of Mental Health for Children and Adolescents with Intellectual and Developmental Disabilities: *An Educational Resource*

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The aim of this Journal is to improve the mental health of children and adolescents with intellectual and developmental disability through enabling academic debate, research and commentary on the field.

Description and purpose

This journal is a modification of the previous newsletter; a vehicle of expertise about mental health information of children and adolescents with intellectual and developmental disability. As a product of CHW School-Link, this journal is supported by School-Link and a collaborative effort with a multi-agency editorial group from the Statewide Behaviour Intervention team of the Department of Aging Disability and Home Care, NSW Family and Community Services, and NSW Department of Education and Communities. We are extremely proud to present these ideas and invite you as authors to help develop this field and the knowledge base to help support children and adolescents.

On our Website:

www.schoollink.chw.edu.au

The website will be playing a crucial role in the information that CHW School-Link can provide to you.

- The collection of previous and current editions is located there with the ability to download articles separately.*
- An invitation for contributions can be found on the website with instructions for authors.*
- Upcoming training at conferences, workshops and other professional development opportunities will be continuously updated.*



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Welcome to the final edition of the journal for 2016.

Firstly I would like to acknowledge and thank our editor Hebah who prepared this edition before embarking on maternity leave. We wish her all the very best with parenting her newborn son. We will miss her amazing work on the journal, and hope we can keep things on track whilst she is away.

The collaboration on our Meet Jessica, awareness raising project was once again recognized by receiving a 2016 quality and innovation award from the Sydney **Children's Hospital Network**. We were thrilled to be given the highly commended award for collaboration.

As the Christmas/January holidays approach I encourage you to reflect on your self-care. Self-care is so important in the work that we do. Reachout.com has a useful self-care assessment tool for professionals. I hope that you will be able to recharge your batteries over the holidays in preparation for an eventful 2017.

Enjoy reading this edition of the journal and please send any feedback or your own contributions to schoollink@chw.edu.au

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The Future of Our Children's Mind, Spirit and Soul: Will the digital revolution destroy them or can schools grow them?

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2 book reviews: **Mind Change** by Susan Greenfields & **Beyond Happiness** by Anthony Seldon.

I encountered two books that deserve to be considered seriously about changes in the way we bring up children. The first is **'Mind Change' by Baroness Susan Greenfields**, the chief scientist in the UK (Penguin Random House UK, 2014). Greenfields first raised her concerns on the effect of digital technologies on the development of the brain and mind in a speech to the House of Lords in Feb 2009. Her book examines the current evidence base. The title is reminiscent of **'climate change'**, as she is concerned that, like climate change, the changes we are witnessing from the influence of digital media on brain development are global, unprecedented, multifaceted and complex, and like climate change it takes a long time and plenty of research to reach a conclusive understanding. She focuses on the influences of **Screen Time, Social Media and On-line Gaming**.

First there is **screen time** which the average young person uses an average of 53 hours/week. Research shows that the amount of screen time in children and pre-teens predicts psychological issues, behaviour

problems, attention problems and physical health, even factoring out the effects of diet and exercise! Bill Clinton described civilization in three stages: first *isolation*, then *interaction* when communities started to exchange ideas, and *integration* with the globalization of thought in the **'noosphere'** with internet connectedness of minds. However, this connection with and reliance on facts and ideas available on the internet risks diminishing human capacity for train-of-thought and use of memory. People are constantly interacting and social networking through **Social Media** with their smartphones and computer, such that the real world becomes less relevant. The outdoor playground is replaced by virtual reality sedentariness, with the loss of physical exploration and freedom of the imagination. This can be seen as the erosion of childhood. Internet and video games expose kids to inappropriate content that is difficult to protect them from. In moderation screen time and the internet can help with stimulation, socialising and can benefit some cognitive functions. But in excess, on-line socialisers are alone, representing a false self to others and themselves. Further, the power of the immediate gratification/stimulation prevents deeper thinking and learning. Children of the new generation are easily distracted with shorter at-



“In moderation screen time and the internet can help with stimulation, socialising and can benefit some cognitive functions...”

attention spans, lack face-to-face social skills and are dependent on digital connectivity. On Social Media, people present an unrealistically confident, often hostile and critical persona. Meta analysis of online student communication over 30 years indicates a steep decline in empathy in the last 10 years. Impulsive communication with an average of 500 Facebook friends risks the exclusion of real relationships.

The next digital cornerstone is **video games** which for the average 10-13-year-old boy takes 43 hours per week! There are case examples of gaming addiction leading to childhood death from neglect; violent content breeds violence in the home, and some significant suicides.

Greenfields pursues underlying explanations of the biology of the brain: addictions such as compulsive gambling is related to the dopamine release in the nuclear accumbens, associated with recklessness and this can be at the expense of dopamine activity in the higher centres of the frontal lobes. The brain may be complex but we know that external inputs change the organisation of brain cells and in turn your thinking. For example, the expert geographical knowledge of London Taxi drivers gives them a bigger hippocampus. Such skill-related brain enhancement is also depended on the principle of use it or lose it. Experience in the first three years of life can determine whether a child will grow up to be peaceful or violent citizen. Different

expert skills are associated with the development of different brain capacities. Particularly the richness or lack of early stimulation affects brain development and future capacity for inter-neuronal connectivity. Thinking itself can provide such stimulation. Dopamine is the final common pathway of all psychoactive drugs of addiction, giving a sense of reward or excitement in the (primitive) brainstem. In particular, the dopamine can be diverted from the prefrontal cortex, the crucial centre of cognition. Adolescence is described as one hypo-frontal risk-taking state. Childhood, schizophrenia, overeating, autism and gambling all involve hypoactive dopamine frontal activity. All such states involve reduction in reality testing and looseness of thinking. Conversely excessive screen time and trauma increases the size of the amygdala, part of the midbrain or reptilian automatic brain.

Greenfields contrasts Mindlessness with Mindfulness: **Mindlessness** is linked to sensation, strong feelings, immediacy, driven by external environment, little meaning or self-consciousness, no time or place reference, high dopamine, prefrontal under function, world meaninglessness and a small neurological assembly of consciousness. **Mindfulness** involves cognition, thinking, past-present-future connection, driven by internal perceptions, personalised meaning, robust sense of self, clear sequentiality, less dopamine, frontal activity, world meaningfulness and greater assembly of consciousness.

Social Networking: “do I even know my 800 Facebook friends that I don’t hang out with? Facebook has turned us into paranoid neurotic masses who are afraid of a real human connection”. The average smartphone user checks Facebook 14 times a day! Zuckerberg argues “relationships are how we discover new ideas, understand our world and ultimately derive long-term happiness... More than 800million people



“You are in a continuous state of high arousal, craving novelty and stimulation, but vulnerable to manipulation...”

have mapped out 100 billion connections and our goal is to help this rewiring accelerate”. This notion challenges identity as an internalised construct in close conjunction with others but suggests we function as nodes in a complex machine and this wiring is superior! This is the ‘noosphere’.

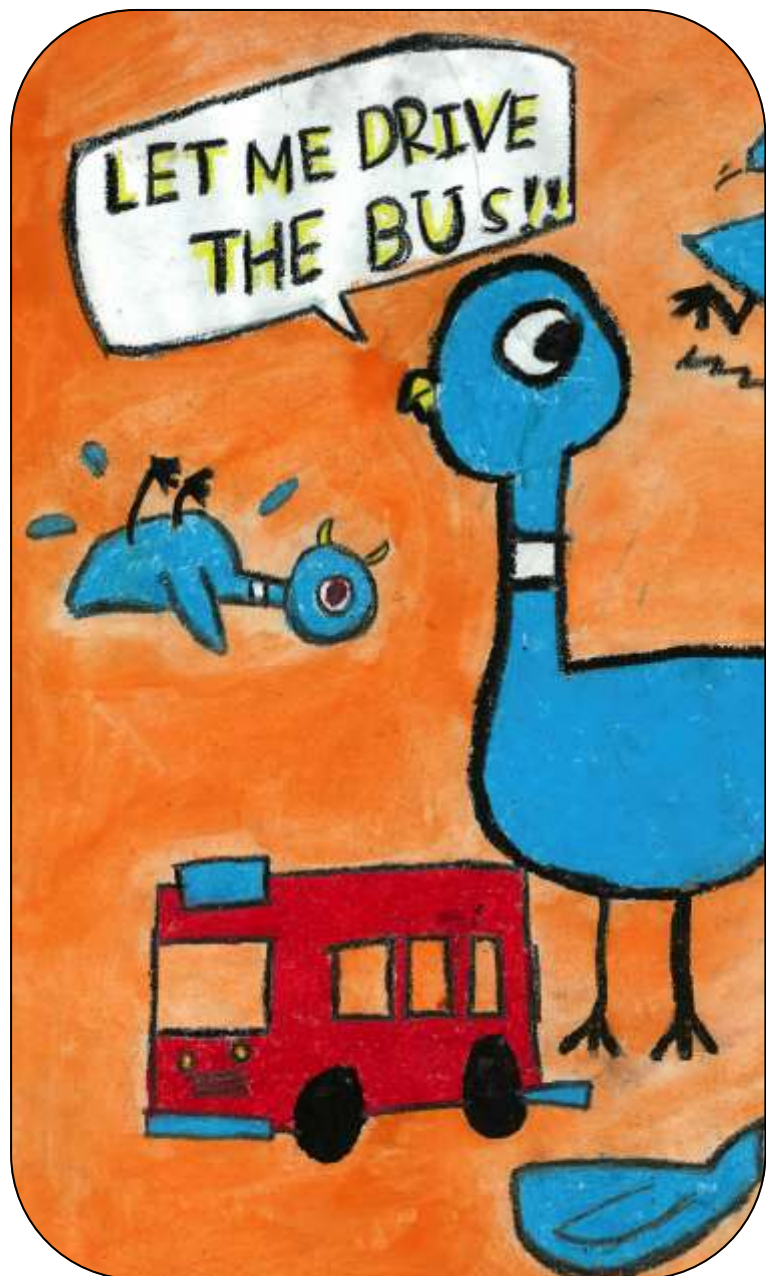
Loneliness and social isolation is bad for health, for example doubling the risk of strokes. We have 209 genes differentially expressed by the level of social contact. Evolution has led to the genetics of immune and cardiac system effectiveness to be sensitive to socio-environmental conditions. These effects are more related to the subjective experience of loneliness than actual social network size. However social contact stabilises heart rate through a burst of the hormone oxytocin. The frequency of living alone has grown dramatically, and the proportion of face to face contact versus electronic connection which was 6:4 hours in 1987 has now reversed! Loneliness predicts attachment to Facebook!

Richard Watson argues that universal connectivity means that we tend to be alone, even when we are together, with social networking even with other people present. Could child care by placing children in front of a screen lead to a primary insecure or avoidant attachment to media? The consequent lonely anxious nature lacks an openness to imagination, new experience and attentiveness to inner feelings as characterises a curious mind. Self disclosure activates brain reward systems which may create the drive for Facebook. However the discrepancy between the public unrealistically positive, narcissistic, false self and the private self may be a key cause for a feeling of disconnection and loneliness.

We all, but particularly the young, use digital communication in greater frequency than face to face. However, face to face communication has physiological correlates from facial expression and body language that reduces stress hormones such as cortisol and raises empathy hormones such as oxytocin, but this chemistry is not seen in digital communication. Further, both genes and emotional communication are key for

growth of mirror neurons which underlies developing the skills of empathy and leads to emotional intelligence. The rise of digital connectedness is inversely related to the availability of friends you can talk to about important matters. Is this the reason for the measured decline in empathy in student cohorts and could this contribute to the rise in psychopathy and autism? Indeed, there is some link between early screen watching and the rise of autistic-like traits associated with lack of eye scanning, lack of emotional communication and theory of mind, with a loss of frontal lobe brain coordination that can be explained by the literal and concrete experience of the screen. Further, excess time on Facebook is related to less time on human relationships which is shown to lead to increased negative, jealous relationships, divorce and destroyed careers. One law firm cited Facebook as a cause in 1/5 divorces.

Rousseau’s notion of the social contract involves an agreement to surrender individual freedoms or rights



for mutual protection and safety. Social networks impact on social morals and cyber bullying has become a new form of violence which is harmful to both bully and victim. Studies suggest 20-40% of young people are victims at some stage. Gossip so easily turns into libel on-line.

Video games are designed to reward users in the same way rats can be conditioned to keep pressing an electric bar to provide food pellets using conditioning behavioural approaches, such that it is designed to be an addictive substitute for real friendship fun. Approximately 8% of young people have all the hall marks of addiction with gaming. They show the growth of ventral striatum, where dopamine is produced in the mid brain, along with desensitisation and tolerance such that gamers lose the dopamine response, in the same way that Ecstasy addicts lose the effect from their fix. In the same way, video games stimulate the user and provides an escape from the real world. Violent video games increase violence in those predisposed. ADHD is a hyper dopamine state, and flooding dopamine receptors with Ritalin re-sensitises you to endogenous dopamine.

Internet Surfing: “without the internet I feel so stupid”. The availability of so much information on the internet means that your capacity for memory and meaningful internalisation of information is less. We bombard ourselves with random information, when knowledge comes from purposeful questioning based on previous perspective.

The impact of the Screen: causes eye strain and presents a world in motion at the expense of visual spatial perspective. **You can't write notes in the margin, you can't see where you are on the page or in the book.** Memory retrieval is aided by locating the book in your mind in the library and remembering the page with its notes. The consequence of internet knowledge is that you remember less. The average student spends 6 minutes on task before switching to an alternative input. Multi-tasking contributes to short lived attention and this failure to internalise information. Books are **generally needed for 'effortful learning'**.

Leaving aside measurement issues, only 20-40% of 'g' or general IQ is inherited. Environment has a big impact. The Flynn Effect is the increase in measured IQ over the last 60 years due to a stimulating environment. An alternative explanation is that we have got better and faster at doing tests that are measured in the IQ. Digital natives are better at abstracted pattern recognition and rule recognition independent of context, as found in computer games. However, the Flynn Effect is reversed and intelligence is reduced over time

when one considers appreciation of context rather than facts, and the importance of growth of knowledge from a personalised conceptual framework of understanding. The human capacity for appreciating symbols and meaning which develops into a framework of deeper understanding over time is what characterises the mature mind. Gifted people have greater brain interconnectivity and also analogical thinking. It is thinking in the context of history/memory and experience, along with perspective and empathy that gives us a capacity to imagine and create. Stories and narrative are the staple of culture, requiring time and concentration, not just information. Such deeper thinking is necessary for creativity and meaning. The digital world is not conducive to developing such personalised human higher capacities, in fact risks damaging these key contributors to culture and civilization.

Other potential consequences of greater digital connectedness include the lack of personal privacy, or even internal narrative. The associated loss of personal identity leads to a lack of relationships. Digital sensory stimulation substitutes for meaningful sex and a decline in dating and birth rates as seen in Japan and South Korea. Cyberspace is a 2-D substitute for a 3-D world with a sedentary lifestyle which is contributing to the epidemic of obesity. Your cyber bubble is protected from other real people, yet you are never alone or independent. You are in a continuous state of high arousal, craving novelty and stimulation, but vulnerable to manipulation, as you seek constant approval, with a blurring of fact and fiction, reality and fantasy with a growing ambivalence. Will the growth of bio-technology enhance or harm health and culture, will it promote or frustrate deep thinking, creativity and real fulfilment?

10 years ago Facebook, Twitter and Wikipedia did not exist. 6 of the 7 billion people of the world now have access to a mobile phone, while only 4.5 billion have **access to a working lavatory. Did George Orwell's '1984' herald our future world of surveillance and manipulation of thought? Could machines really take over from human creativity? Could biochemistry and genetics transform humans leading to marriage by numbers and eugenics? Humanity has always had a love-hate relationship with progress. Socrates was concerned that writing would destroy mental prowess, 'creating forgetfulness in the learners' soul'. As H. L. Menchen said: 'for every complex question, there is a simple answer and it is wrong!' We have to adapt to change and use the opportunities for greater digital connectedness. It can contribute but not substitute for our nature as social beings. This involves appreciating our need for personal narrative and acknowledged as special, to be accepted as a member of a tribe and part of a larger collective identity, and to experience gratification and**

fulfillment. In our efforts to shape the future we need to be aware that cyberspace can be used for both good and for ill.

Although Greenfields raises important questions of childhood neuroplasticity, the strength of her scientific evidence has been challenged, particularly when she suggests media usage may be a factor in the increase in ASD. In particular, the well-respected Dorothy Bishop has criticised Greenfields on her website: <http://deevybee.blogspot.co.uk/2014/09/why-most-scientists-dont-take-susan.html>. This confirms that more research is needed to refine our understanding.

Beyond Happiness: the trap of 'happiness' and how to find deeper meaning and joy by Anthony Seldon.

(2015. Hodder & Stoughton Ltd; London). Anthony Seldon is a prominent headmaster, author of history and biographies, founder member of "Action for Happiness" (2011) and president of the International Positive Education Network. He has implemented his ideas in creative learning, bridging the gap between state and independent schools, holistic education and the teaching of happiness, wellbeing and character education in his latest school, Wellington College. His writing is based on his own experience of life. He posits that we can live our lives on three distinct levels:

I Pain/Pleasure of the body, which involves narcissism and egotism, akin to the behavior of animals; it is amoral, self centered, experiencing the world as if we are the centre of the universe.

II Happiness is based on relationships with others and our deeper selves, a by product of living wisely, treating people as we treat ourselves well, morally and as equals. However, happiness is often built on impulse and gratification, from consumerism, food and excitement. It is often linked to exploitation eg of drugs, alcohol and sex.

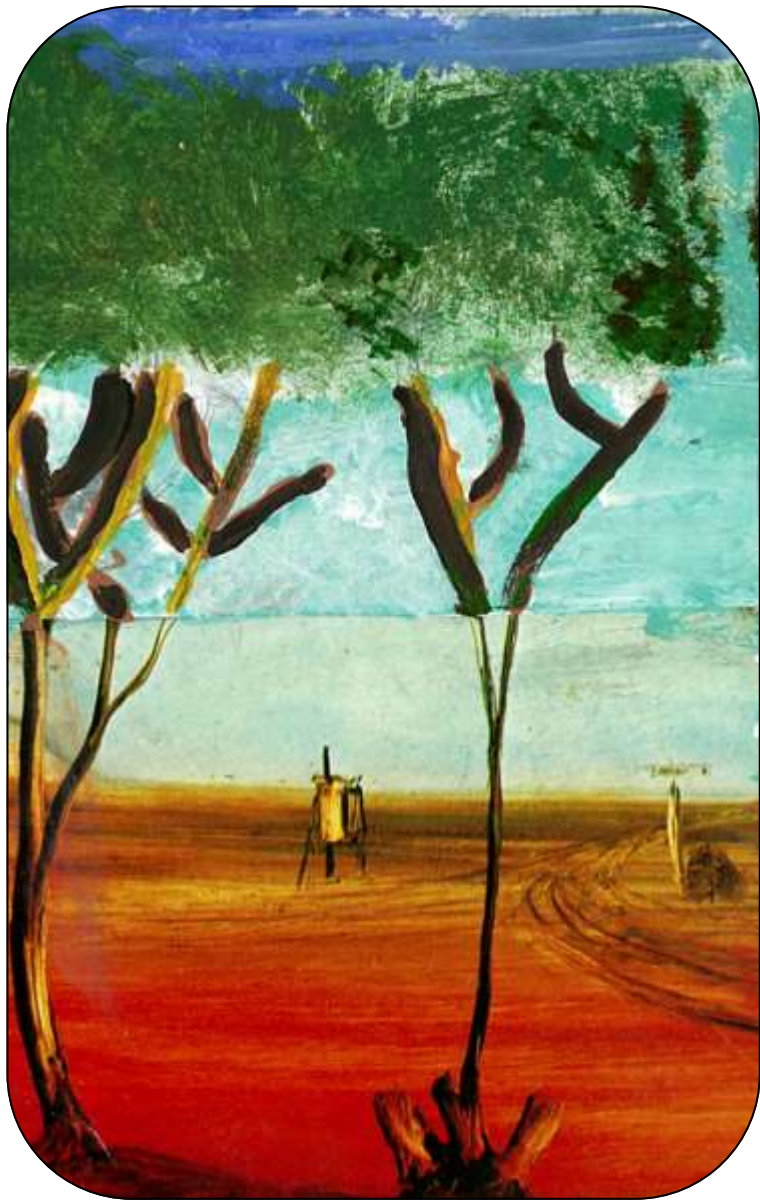
III Joy: once we have built secure egos from happiness, we learn to dissolve our egos into deeper selves in harmony with creation.

The origins of these ideas come from Socrates self-knowledge, Aristotle's virtuous life, the Stoics' use of rational thought and objective perspective, the Utilitarians' greatest happiness for the greatest number, followed by the positive psychology of Maslow with his hierarchy of needs and Seligman and his research 'flourishment'. It is learning about what is right about people, in addition to what is wrong. We can't but feel that there is something deep down that is about more than happiness. The problem is religions often become egotistical and self serving and claim a monopoly on

truth, even as a justification for war. Religion may provide signposts but not a destination. Joy involves enlightenment, experiencing reality beyond our subjective minds, to true freedom and enduring security, towards a more altruistic fulfillment and a more caring society. Life is a journey created by our choices from narcissism to wholeness, such as not blaming others but taking responsibility for who we are and who we resolve to become, from pleasure to self-knowledge to happiness and joy. Eight billion people are each on their unique journey to travel either to greater self-absorption and personal aggrandisement or to immersion in compassion and love for others. We chose the direction we take. Unhappiness and depression does not seem to be reducing with greater affluence and advances in medicine. What is our individual 'song', unique mission or opportunity in life? Have you become more materialistic or more spiritual? Has your aim changed over time? Who has influenced/inspired you and what about? What company do we keep and what is their influence on a road of discovery? In a life well lived death is not to be feared.

We often experience pain from not looking after our bodies well and unwise living. The primary cause of unhappiness is attachment to happiness and dependence on pleasure. Martin Seligman summed happiness in the acronym PERMA: Positive emotions, Engagement, Relationships, Meaning and Accomplishment; if embraced happiness will follow. Unpleasant and selfish people are so often miserable. Karma and good actions bring us happiness.

I Pleasure/Pain: If we maximise pleasure and minimise pain, we are no better off than animals. John Stuart Milne wrote: "it is better to be a human dissatisfied than a pig or a fool satisfied. And if the fool or the pig are of a different opinion, it is because they only know their own side of the question". There are higher ends than the chemical sensations of relief of pain. Pleasure should not be denied, as some puritans believe. Pleasures are not evil except if we are obsessed with avoiding pain. Mindfulness of our body contributes to pleasure: eating, flavours, smells, exercise, pets, recreation, relaxing, reading (often in solitude), art, music, architecture, culture, exploring, travelling, appreciating beauty. These things can be enhanced by education teaching more about them. Pleasure in excess can be harmful: overeating, too much wine, drugs, the objectification or exploitation of sex, excessive possessiveness e.g. of children. A balance in work/recreation is necessary so as not to become stale, dull, tired; taking our team sport too seriously. Mindfulness involves travelling lightly through life; a bearable lightness of being.



“The primary cause of unhappiness is attachment to happiness and dependence on pleasure...”

II Happiness in the context of this book results from relationships, gained from sharing and giving. The more we grow in our capacity for relationships the happier we become. Happiness is central to individuals, families and wider organisations. Modern life may be characterised by an emphasis on pleasure at the expense of happiness with a consequent society cost in depression and anxiety. At the other end of a -5 to +5 scale of happiness is flourishing, inner fulfilment and radiant energy: a life lived in joy, immersed in love, with an understanding that we are part of a connected and transcendent world in which there could never be anything to fear. The eyes are the window into the heart of where someone is on this scale: a +5 joyful person shows spiritual peace and wisdom, radiates love, compassion and understanding. The eightfold pathway to a happy life is not wealth, possession and power, but:

1. Accepting self, physically and mentally (our past, parents, personality), and others;

2. Belonging to good organisations, being part of something bigger and connected (growing from club, religion or country to a part of humanity);

3. Character virtues (eg hard work, kindness, politeness, punctuality, loyalty, courage, optimism and obedience, and are too often substituted for exam performance);

4. Discipline is vital to resilience and to help us live to our aims and values rather than succumbing to being a victim or pessimist, in different spheres: in body, mind, finances, use of time, relationships, energy and jobs, versus temptation and addictive behaviour in alcohol, bullying, control and cleanliness, drugs, exercise, food, gambling, sex, shopping, status and theft;

5. Empathising and compassion to deepen relationships: that which opens your heart eg music, a memory, a poem or a book, appreciating, forgiving, connecting, taking action, giving thanks, versus judgement, prejudice, cynicism, criticism, detachment and rejection which leaves us isolated and unhappy;

6. Focusing on goals and search for meaning; consider focus under 5 headings, like an archer having 5 arrows to fire: work, family, friends, home and leisure, and any causes you believe in. Aims should be realistic, and achievable within your resources of finances, circumstances, bodily and mental powers. Compare real aims and aspirational, short term and long term. Consider the seven stage model to maximize performance and happiness: read from the 7th upwards: 1. The Crown- be still and reboot; 2. The 3rd eye- appreciate and grow; 3. The Throat- Optimise your wellbeing; 4. The Heart- Empathise with others; 5. The Solar Plexus- Take the initiative; 6. The Sacral- manage your time; 7. The Roots- Set your targets.

7. Giving and serving elevates others and energises us. Seldon lists 10 ways of giving: 1. Acknowledge others; 2. Acceptance without judgement; 3. Using our Expertise for others; 4. Selfless Love is the greatest gift. 5. **Money spent wisely on others;** 6. **‘One-minutes stands’** where we help a stranger; 7. Make Peace with those you have hurt; 8. Smiling makes others happy as well



as ourselves; 9. Giving time, being there for someone; 10. Empathic touch.

8. Health in minds, bodies, and emotions maximises opportunities for happiness. We can all live healthier lives; **don't want to only pollute your bodies. What does living at peak effectiveness require?** Mastery of breathing prevents fear or anger. Stretching, yoga, relaxation, food, exercise and drink all contribute to bodily and mental health.

III Joy: Deeper, joyful and spiritual vision of happiness is championed by **Action to Happiness. the 'eight paths to happiness' are a stage to somewhere, not a place of its own, and will create greater happiness.** Happiness can have limitations. It can be superficial, complacent, indifferent, and stultifying to creativity and the depth of human experience. In fact, many religions can be stale and inert. Jesus created a revolution with a focus on a humility and a spirit full of love and joy. Ideology can become a trap of limitations. Is the universe random or a grand design? It is all chance or predestined? A belief in choice suggests it is in between the two. Similarly, Freudian analysis is based on understanding the misfortune of repression, whereas Jung saw misfortune serving a purpose connecting to a collective consciousness. For Freud individuation was freeing us from our unconscious past, whereas for Jung individuation was through achieving inner harmony. For Freud biological necessity was the drive, for Jung life had a divine purpose. Despite the modern cult of individualism, mindful awareness of our consciousness makes aware that the mind is bigger and connected to humanity. **"The only wisdom we can hope to acquire is the wisdom of humility. Humility is endless" (TS Elliot).**

James Fowler, developmental psychologist, provided a seven stage journey of spiritual growth: 0. Primal/undifferentiated: learning about safety and trust in the first two years of life.

1. Intuitive-projective: fluidity of thought before ego restricts access to sub-consciousness.
2. Mythical-Literal: a strong sense of justice and meritocracy, taking parables literally.
3. Synthetic-Conventional: Teens tend to conform to religious authority.
4. Individuative-Reflective: Struggle with faith, **becoming open minded to faith's contradictions.**
5. Conjunctive: mid-life adults accept the paradoxes of faith by transcending literal meaning.
6. Universalising: the wise embrace the universals of love, justice, compassion and become enlightened. Simon Walker, a spiritual teacher simplified this to: Lack of Awareness, Awareness, Responsibility and Choice, and Freedom. It is important not to confuse atheism and the rejection with religion, with the existence of a spiritual realm. In fact, fundamentalists have much in common with atheists. They use their understanding of science, philosophy or religion to have a monopoly on truth, and reject spiritual insights of artists, poets, thinkers and statesmen.

Further paths to joy are described:

Inquiry including of our unconscious, learning to live consciously. **'A unexamined life is not worth living' (Socrates). Don't get trapped in happiness and self-satisfaction. Life remains a mystery, but 'whoever knows himself knows his lord' (Islamic saying). Self examination can be aided by looking at your negatives and positives as seen in your: Actions, Beliefs, Fantasies, Fears, Hates, Hopes and Influences, Pressures, Pride, and Traits; all areas of uncertainty of self.**

Journey: beyond your own limitations to discover the world afresh. We all have a duty in life to find a purpose. There are two halves of life: as builders of qualifications, careers and families, and the second half we let it all go, becoming less assertive and more compassionate. Pilgrimage is a journey to places of special significance; what are those places for you?

Karma: we are all interconnected in profound and complex ways and the ripples we send out will rebound on us; i.e. **'do as you would be done by' or love others as you would be loved yourself.** We are therefore responsible for our actions and it is important to acknowledge harm we have done to others. You can still make peace with those who have died or their families. Acknowledge habitual selfless actions such as volunteering. *Agape* or selfless love is the natural

expression of all on the highest spiritual path and the natural state of those in the highest state of evolution.

Compassion is at the heart of all religions and helps us overcome the drives of feeding, fleeing, fighting and reproduction which are at the heart of our reptilian brain. Karl Jaspers described the Axial age (800-200 BC) when a spiritual awakening occurred including Confucianism, Buddhism and Platonism. CS Lewis describes love of family, of friends and sexual love, which all entail a degree of self-centeredness, whereas Agape alone is the love that yields not benefit but is a sovereign path to joy.

Liturgy: derived from leitōs (public) and ergon (work) or corporate worship. Unfortunately, liturgies are often tightly controlled by religious authorities, often formulaic and rigid. Prayer is a form of liturgy and the main kinds are gratitude, devotion and supplication. We need to be grateful for everything, including what we do not like. We need to think about what we need to give thanks for: food, fresh air, jobs, friends, family and the energy that gives us life.

Life's heroes include those that face horror mindfully, such as those who 'faced' the holocaust like Anne Frank. Devotion can be dangerous whether it be to possessions, sports stars or religious fundamentalism.

However, devotion to the infinite, the source of all love, peace and wisdom is worthy, as it will never harm any being. What are your blocks to such devotion? Prayer as supplication is not to change the sick person who we pray for but to change ourselves and the way we respond to adversity. Facing death with equanimity is the ultimate liberation in life. The 14th century book of **'the cloud of the unknowing'** suggests that only by abandoning all our beliefs to feelings of

“Meditation, mindfulness and contemplations are suddenly everywhere...”

'unknowingness' can we begin to comprehend the realm of God. Great sermons, religious songs and holy texts can bind people together and elevate their spirits.

Meditation: meditation, mindfulness and contemplations are suddenly everywhere. They are not new fashions but ancient practices for spirituality, aiming for a quiet, alert mind focused on the present, not darting off. This still and receptive mind is attainable for us all and such a mind is constantly joyful. It is a non-judgmental awareness, to aid relaxation and improve chronic mental conditions. Mindfulness may be seen as secular tool, but awareness of the movement of thought and feeling is your soul, or Atman, a Hindu term. Meditation, in Tibetan means to become familiar with your soul, and is found in all the major religions. Rowan Williams, the former Archbishop of Canterbury is a recent convert. It may involve concentration on a mantra or religious text, an object or statue, a physical posture in Yoga, and includes breath control or pranayama, immersing ourselves in complete silence. Why is your ego preventing you practicing? Are we held back by a fear of extinction or attachment to things that prevent us being free or fully alive? Contemplation is allowing the soul to understand divine forms. Current lives are designed to distract from the present moment. It used to be a fear of attack e.g. of wild animals, now it is the lure of electronic media. Spirituality means waking up, whereas we pass through much of life in partial awareness, like driving automatically. We are inclined to miss the loveliness and beauty of human existence.



Iris Murdock invites us to ‘focus on what is good as doing so will connect us with the true nature of things’. Doing so makes us ‘humble, seeing oneself as nothing and seeing other things as they are’. For Anthony Seldon this journey enabled him to find total love for his dying wife. It taught him to reflect on his own learning and mistakes in life. In such a journey, the more you put into it, the more you get out. Maybe, he concludes, his role is as a teacher. At the end of it he felt lighter than in his previous life, and the future is filled with excitement and anticipation. He invites us to make a similar journey.

I find this book engaging. It brings together inter-denominational commonalities of the great religions and suggests they are still relevant to our post modern world. Great schools teach us about emotions, relationships, character and good, which are arguably more important components of preparing a young person for the future than exam grades. As Hugh Mackay, the social researcher, recently reported: over two thirds of Australians describe themselves as believing in a greater being or God, but less the 10% attend church regularly (Beyond Belief, 2016). This is because most of our society still believe in religious derived attitudes, but not the ideology. Inconsistencies of religious ideologies has sapped the authority each has.

A book such as this may well capture a wide audience, not just those using mindfulness in mental health and social welfare. The idea of a pathway to fulfillment, as here described, will similarly find many adherents. Such ideas are difficult to test empirically, because they derive from history, faith and experience. However, the strength of such an approach is dependent on **a spiritual leaders’ familiarity and linkage to a wider literature of ideas.**

I am inclined to recognise the descriptions of a pathway of awareness of happiness through life. I adhere to a notion of an evolution of our ways of explaining our purpose in life and the importance of our personal and wider connectivity. How does spiritual connectedness link to the growth of an awareness of the **noosphere, as describe in ‘Mind Change’ above?** Such global processes of our human connectedness could become a unifying humanising movement, to contrast **to other ‘truths’ we are fed, such as the inevitable political forces of global economics, resources and climate.** I have long felt schools should have a metric of wellbeing, resilience and flourishing against which to benchmark their progress and achievement. For those that believe in moral betterment of the individual and humanity, this book provides an experiential and secular guide to put the human spirit at the centre of life.

“Spirituality means waking up, whereas we pass through much of life in partial awareness, like driving automatically. We are inclined to miss the loveliness and beauty of human existence...”



Reading List

Brown, R.I. and Schippers, Al. (2016). Editorial. Special Issue: Quality of life and family quality of life: Recent developments in research and application. *Journal of Intellectual and Developmental Disability*. Volume 41, Issue 4, 2016.

This issue is the first part of two special editions with the second to appear in the *Journal of Policy and Practice in Intellectual Disabilities* in 2017. This particular edition covers a variety of family issues such as sibling experiences, friendships in adolescence, individualised support, perceptions and proxies, comparison measures and more.

Pennington, R., Courtade, G., Ault, M. (2016). Five essential features of quality educational programs for students with moderate and severe intellectual disability: A guide for administrators. *Education and Training in Autism and Developmental Disabilities*. Vol 51, Issue 3, Pp. 294-306.

This is an interesting overview for special educators to consider.

Below is the reference list for the article by Walsh, Samaras and Trollor (2016), found on page 24 of this journal. *Addressing Cardiometabolic Risk Factors in People with an Intellectual Disability*.

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Notes from the 19th Society for the Study of Behavioural Phenotypes (SSBP) International Research Symposium, University of Siena, 9-11th September 2016

Peter Wurth

Psychiatrist with a special interest in Intellectual Disability

The conference was held at the University of Siena, Italy, founded in 1240, set right next to the Piazza del Campo, the square where the famous horse race, the Palio, is held twice a year. We were taken on a tour of the original Hospital of Santa Maria della Scala, opposite the beautiful Duomo. Of the 80 delegates 16 were from Australia. The medieval hill town was a delight to walk around.

Sakkubai Nadiu from Boston opened the conference with an account of Rett Syndrome; from discovery to treatment. The syndrome was named by Andreas Rett 50 years ago, and has now been found to be due in mutations in the X-linked MECP2 gene. Common features include lack of abnormality at birth, then substantially delayed growth in head circumference within the first few months of life and apathy, often mistaken **as a sign of a 'good' baby. There is some early speech** which is then lost, and some early purposeful hand use which is then lost, with purposeless or stereotypical hand movements common. Major features include microcephaly, seizures, respiratory anomalies, and autonomic dysfunction and severe intellectual disability. No one feature is invariable however. The severity is proportional to the pattern of X inactivation. 5% of patients are male who are generally much more severely affected. Most are de novo mutations, but there are occasional carriers with very skewed X inactivation who are mildly affected. The proportion of immature neurons is much higher, with MECP2 necessary for neuronal maturation. Neurons are small with a reduction of synapses by 50% and reduced dendritic arborization. Vision is preserved as ocular neurons do not use MECP2, and parents will observe that their children **'speak with their eyes'. There is an excess of both glutamate and glutamate responsive NMDA receptors,** leading to excessive excitation and cell death, in children under 10 after which they appear to burn out, and these glutamate and NMDA receptor excesses are no longer seen. This centre conducted a trial of dextromethorphan, an NMDA antagonist, and found an improvement in language skills which reached significance. Other changes did not reach significance. Test-

ing the outcomes of intervention in this population is compromised by the difficulty assessing changes in cognitive function at such low levels of ability.

Alessandra Renieri from Siena then spoke on the genomic complexity underlying Rett spectrum disorders. Her team identified the FOXP1-related Rett variant syndrome, which shows a shorter period of perinatal normalcy and more severe microcephaly. This gene is located on chromosome 14 and the sex ratio of affected individuals is therefore equal. Both this gene and MECP2 dysregulate GABA. Her team has investigated molecular mechanisms via the study of induced pluripotent stem cell (iPSC)-derived neurons, a technique involving cell cultivation of skin fibroblasts from affected individuals and the creation of stem cells, from which can be grown simple neural networks for detailed analysis. They found shared pathways in the neuronal damage produced by both genetic variants involving dysregulation of GABA and of genes producing the enzyme histone deacetylase 6 involved in the formation of microtubules. There are drugs currently under development that can target these abnormalities. The current clinical implications are unclear. The dysregulation of GABA suggests that carbamazepine should be avoided in Rett syndrome children under 10, after which the excess of glutamate no longer operates. The two speakers in subsequent discussions gave contradictory views about the safety of valproate in this population, one advocating it as inhibitory of GABA, the other concerned about its adverse effect on already dysfunctional mitochondria.

Dafin Muresanu from Romania presented on brain protection and recovery after stroke and traumatic

“Structural connectivity precedes functional connectivity although the process is probably bi-directional...”

brain injury, emphasising the need to take a whole brain approach. He described the very poor evidence base for many interventions aimed at promoting brain recovery, with unclear concepts and very poor study design. He emphasised that factors promoting immediate neuroprotection could work in contradictory ways from those which optimise long term recovery. He described the **'anti-correlation' common to many processes** following an acute brain lesion, and the need to take an approach focused on large scale networks rather than molecules. There are many homeostatic mechanisms operating within endogenous neuromodulation, and interventions aimed at one process can have unforeseen consequences in upsetting this balance. Data support the idea that even a small lesion can trigger progressive disorganisation of axons even at a distance from the core site of injury, with possible mechanisms being widespread inflammation and dysfunction of neurovascular units. Promoting long term recovery via neuroplasticity and neurogenesis is currently a major field of endeavour in neurology.

This and the three subsequent talks, all by neurologists, served to highlight current foci of neurological research, but required significantly more basic knowledge than possessed by many conference delegates.

Antonio Giorgio from Siena spoke on brain connectivity during development, the study of which has been immeasurably facilitated by the development of non-invasive imaging including functional MRI, especially in the resting state, and diffusion tensor imaging. Structural connectivity precedes functional connectivity although the process is probably bi-directional.

Massimo Filippi from Milan spoke on the **'vegetarian brain'**, describing the differences in regional brain activation to relevant stimuli between omnivores and a group comprised of vegetarians and vegans. Vegans for example activated the fusiform gyrus to faces of animals more than to faces of people, whereas omnivores did not activate this facial recognition gyrus on presentation of pictures of faces of animals. Pictures of suffering humans or animals evoked different responses depending on whether the subject experienced empathy versus cognitive understanding. While there were some differences between vegans and vegetarians, the overarching conclusion appears to be that both anthropomorphise animals to an extent and mount more empathic responses to animals than do omnivores.

Stefano Cappa from Pavia spoke on brain connectivity in neurodegenerative diseases. He described the idea of excessive attribution of functions to specific cortical areas as akin to a modern phrenology, and emphasised that the reality in most degenerative disease is one of large scale dysfunction in cognitive networks. The process of degeneration starts well before presentation and diagnosis. The old model of specific **histopathology as described in Alzheimer's, Picks and motor neurone diseases** is a late stage phenomenon. Investigations into functional impairment provide far more information when compared to normals than do investigations into those without any remnant of the relevant skill. Behaviourally disturbed patients will not tolerate MRI scanning especially when this is combined with an expectation of task performance. Brief scanning of resting states however can provide valuable information, with evidence that resting state deficits parallel those deficits seen during specific task



performance. He highlighted the concept of biological reserve as underpinning the variability in age onset of many diseases. There is early evidence for example **that individuals with language variant Alzheimer's have a history of delayed childhood language acquisition, creating later vulnerability.**

David Zee from Johns Hopkins gave a very informative account of eye movements in degenerative disorders, incorporating compelling reasons to study saccades, i.e. the rapid eye movements we make to scan the environment. Accurate localisation of brain lesions according to the type of disorder of saccades can lead to early diagnosis. He listed many reasons to study them, including that various saccadopathies are signatures of a number of degenerative diseases such as **Parkinson's or Huntington's, that they can be studied in a wide range of animals including zebra fish, and that their formation is dependent on healthy functioning of a wide number of brain functions, including the cerebellum, brain stem, basal ganglia and cerebral hemispheres.** He noted that nystagmus is common in various neurodevelopmental disorders, is poorly studied, and may be secondary to ion channelopathies.

Andreas Chiochetti from Frankfurt described the complex genetic architecture of autism spectrum disorders. Little real progress has been made. There are no biomarkers and no models of pathogenesis. Greatly differing genetics can produce a similar phenotype, and the mechanisms underlying this remain unknown. Most inheritance appears to rely on either multiple common variants acting together, or is unknown. The prevalence of ASD is now estimated at 1%, with the risk of ASD in a sibling of a proband being 20%. Perhaps 3-10% can be explained by rare or de novo variants. Genetic patterns appear to differ between those with ASD with and without ID.

Terry Naerland from Oslo spoke on autism symptoms and gender ratios across different disorders, focusing on the role of aetiology and the degree of ID. This team is analysing ASD symptoms in different groups, including **idiopathic ASD, Down syndrome, Angelman's and Smith-Magenis syndromes, and Fragile X.** They note that the range in ratio of male to female subjects is 2 to 7:1. They are investigating the connection between gender and ASD symptoms.

Stephan Huijbregts from Leiden discussed executive functioning and the hypodopaminergic state in adults with PKU. In Europe this disorder has an incidence of 1:10,000. Degrees of severity occur. Elevated phenylalanine levels block the blood brain barrier and stop other neurotransmitter precursors entering the brain. Phenylalanine also directly damages white matter. Die-

“Ernesto Burgio from Palermo gave a fascinating but alarming presentation on the rise of neurodevelopmental disorders...”

tary treatment must be started before one month of age. Executive function is damaged much more than IQ, and the extent of damage depends on the serum level of phenylalanine. Problems compound with the expectation of performance of simultaneous tasks of executive functioning (EF). His study found a direct link between low levels of dopamine availability and EF, but further contributions were probably made by low levels of serotonin and white matter damage. Stimulant medications may therefore be helpful in PKU. The presence of a hypodopaminergic state raises the question of whether patients will be more vulnerable to the development of Parkinsonism. He also made mention of the medication sapropterin or BH4, a co-factor which directly lowers phenylalanine levels in those intolerant of dietary management. (This medication is very expensive in Australia, and is subsidised by the PBS only for specific deficiency states and not for PKU.) He recommended maintaining phenylalanine levels in PKU below 600 as sufficient, with no evidence supporting the lower levels commonly recommended as necessary.

Friederike Ehrhart from Maastricht described biological pathway analysis of Rett syndrome transcriptomics, a highly technical exposition which illustrated the extent to which multiple pathways are either up or down regulated in this disorder. She noted the availability of WikiPathways as a repository of biological pathways freely available on the Internet. Further work will help understand the link between the MECP2 deletion and the development of the phenotype of Rett syndrome.

Shruti Garg from Manchester presented a randomised control trial of simvastatin in Neurofibromatosis Type 1 (NF1) autism. This condition has an incidence of 1:3000 and is a single gene disorder, a mutation at 17q11.2. 50% are the result of dominant inheritance and 50% sporadic. Over 50% have ASD. Previous trials of simvastatin have been negative. This study focused on earlier intervention than previous trials, with 26 patients aged between 5 and 10 years dosed at a rate of 1mg/kg completing the study. The results were negative. Rather than the reduction of GABA expected in frontal white matter there was an increase. There were



no differences in parent behaviour ratings but no adverse effects. NF1 inhibits GABA in animals and NF1 animal models show an excess of GABA. Recent human studies however have found a relative deficiency of GABA in NF1. This disappointing result highlights the problems of extrapolating from animal models to humans in genetic disorders, and the pitfalls in the development of specific drug treatments.

Ernesto Burgio from Palermo gave a fascinating but alarming presentation on the rise of neurodevelopmental disorders, highlighting the shift in emphasis from genetics to epigenetics. He described brain hardware as genetically derived but the connectome as epigenetic. Genes require regulation and this is not done by DNA. The difference between cells is secondary to epigenetics given that all have identical DNA. He described environment as a continuous stream of outside information, and the brain as the most plastic organ of the body throughout life. Many rare genetic variants show substantial overlap between disorders such as schizophrenia, bipolar disorder, ASD and ID. He noted that autism is a disorder of the connectome not of the hardware. The concordance between dizygotic twins is increasing, whereas that between monozygotic

twins is decreasing, indicating an increase in the contribution made by epigenetic factors. He noted that early maternal childhood abuse leads to an increased risk of autism in her child. He also noted that pregnant women in Europe have been found to contain between 200 and 300 exogenous chemicals or pollutants. Living near a freeway increases the risk of autistic offspring by 2 or 3 times. There has been a major increase in the prevalence of cancer in the first two years of life over the last 40 years, likely also to be due to epigenetic factors such as pollutants and obesity.

Jim Harris from Johns Hopkins described progress over the last 25 years in understanding Lesch Nyhan syndrome. This is an X-linked recessive disorder of purine metabolism, with an incidence of 1:300 000. There is a spectrum of severity, with only those with classic LNS who have less than 1% of expected enzyme levels exhibiting the classic pattern of severe self-injury. Less severely affected individuals show motor abnormalities varying from clumsiness to severe dystonia, and cognitive impairment ranging from problems of attention to intellectual disability. Impulsivity is common. All individuals have increased urate levels with the standard complications that require active medical management. Lowering serum urate with allopurinol does not improve the behavioural phenotype. A few patients in an open label trial have benefited from S-adenyl methionine. The dopamine D1 agonist ecopisan has shown early promise in reducing SIB. Deep brain stimulation in the globus pallidus can be transformational, with one patient still doing well 15 years later.

Petrus de Vries from Cape Town presented the results of a large trial of everolimus adjunctive therapy for the treatment of refractory seizures in patients with Tuberous Sclerosis Complex (TSC). 70% of these individuals have epilepsy, often refractory. Everolimus is an inhibitor of mTOR. The study was strongly supported by the manufacturer Novartis. No studies are done with the related medication sirolimus (rapamycin) which is significantly cheaper and off patent. Everolimus is already licensed for use in shrinking SEGAs and renal AMLs. Subjects in this trial had to have at least 16 seizures in a four-week period, but the median seizure frequency pre-treatment was 35-40. 40% of subjects on high dose everolimus showed a greater than 50% reduction in seizure frequency, while 5% became seizure free. Discontinuations were low despite the high prevalence of side effects, including stomatitis, diarrhoea, mouth ulceration and hyperlipidaemia. Vineland and quality of life questionnaires have been administered and analysis is ongoing. Future trials are planned to look at the impact on psychiatric and behavioural disorders.

Bissell from Birmingham compared difficult behaviours in younger and older children with TSC, finding that young children had high rates of self-injury and aggression, indicating that behavioural problems in TSC begin early. The average age of diagnosis of autism in 2000 individuals with TSE was very late at 7. Inflexibility and resultant tantrums are very problematic.

Shahid Zaman from Cambridge discussed evaluating **the earliest manifestations of Alzheimer's disease in Down syndrome**. 40% of those with Down syndrome aged between 50 and 59 will have clinical dementia, compared to 5% of those over 65 without Down syndrome. **15% of Down individuals have Alzheimer's histopathology at the age of 15, with 100% by the age of 35**. Pathology is evident perhaps 20 years before clinical onset. Clinical onset however is not inevitable and this remains unexplained, but must be due to neuroprotective factors. The cascade of damage appears to result from the existence of three copies of the amyloid precursor protein (APP) gene on chromosome 21.

Ines Pote from Kings College described the developmental trajectory of glutamate in the human brain from the foetus to the early infant. Glutamate is an excitatory neurotransmitter, which is vital for the development of neurons and circuits. It is the immediate precursor of GABA which is inhibitory, and in the healthy adult brain these two neurotransmitters are in balance. In the immature nervous system however GABA is excitatory, and possibly switches functions at birth. In this small sample size of individuals at high risk of autism because of a sibling with autism, glutamate levels were higher. Remarkably proton magnetic resonance spectroscopy (HMRS) can measure glutamate concentrations in foetal brains. Data are preliminary and the sample size is so far small.

Rosalyn Hithersay from UCL described the use of functional near infrared spectroscopy (FNIS) to measure executive functioning in adults with Down syndrome. This technology relies on the fact that near infrared light passes through brain tissues and bone, but is differentially absorbed by haemoglobin depending on the degree of oxygen saturation. Light penetrating the skull traverses an arc before exit. A cap similar to an EEG cap can be used to both transmit and detect light, with the depth of imaging dependent on the distance between source and sensor. Thick hair creates problems with using this technology in older children and adults. The investigation is well tolerated. The machine automatically subtracts the part of signal due to variations in skin blood flow. Healthy adults were scanned while performing four tasks involving different executive functions. This is a preliminary study to determine the likely effectiveness of the method in individuals

with Down and other syndromes. Larger cross-sectional and longitudinal studies are planned.

Jessica Penhallow from Birmingham described long term predictors of quality of life for adults with genetic syndromes. Various measures were used in a sample of 69 parents and carers of individuals with **Angelman's, Cri du Chat, Cornelia de Lange, FXS and Prader Willi syndromes**. The mean age was 29 and 68% were male. Measurements were taken in 2003 and 2015. The main predictor of poor quality of life was low mood. Quality of life was measured across four domains, physical, psychological, social relationships and quality of the environment. The presence of autism and challenging behaviours were significant negative factors. Self-reported quality of life in those able to participate closely paralleled the information obtained from informants. (Pat Howlin described finding surprisingly good quality of life in adult autistics, leading her to question the validity in that population of the tool used.) This study suggests that early intervention to address low mood may have significant later benefits.

Anne Bassett from Toronto described the neurocognitive profile of 22q11.2 deletion syndrome. She emphasised that this is now the universal name for this syndrome, and older terms such as VCFS are now discouraged. This is a multisystem syndrome. The facial features can be subtle. There are high rates of ASD, ADHD, seizures, mood and anxiety symptoms, Parkinsonism and schizophrenia, which the incidence of the latter approaching 30%. Lifespan is reduced. IQ ranges from normal to intellectual disability, but those with an average IQ still have impairment in social and communication skills. Adaptive function is proportional to IQ and to the presence of schizophrenia. The onset of schizophrenia in this population has a mean age of 21, the same as in neurotypicals. The neurocognitive profiles of those with schizophrenia with and without this syndrome are similar. There are no tests that will predict which individuals with this syndrome will develop schizophrenia. This investigation included individuals with schizophrenia who had been stabilised on medication. The impact of that medication on these results is therefore unknown. Most cases of this syndrome occur de novo but 5-10% are inherited.

“Quality of life was measured across four domains, physical, psychological, social relationships and quality of the environment...”



Kate Woodcock from Belfast described the development of a pilot battery to measure executive function (EF) (also termed cognitive control) in individuals with genetic neurodevelopmental disorders. Three EFs are typically studied – inhibition, task switching, and updating working memory. EF is typically measured indirectly by task performance, which requires competence in numerous components. EF measures therefore have poor reliability, and rely on a number of assumptions. All of those individuals we study have patchy cognitive skills. She described the development of a battery of 25 tests designed to minimise the influence of cognitive strengths and weaknesses, piloted with 125 neurotypical children aged 6-12, and 12 with genetic syndromes. This tool, the CAN measure assessment battery, has now been administered to almost 700 neurotypical 6-12 year olds and should facilitate more valid assessment of EF in those with neurodevelopmental disorders. At this stage assessment of children aged less than 6 is not possible. Her team plans to make this instrument freely available.

Kate Wolfe from UCL described the phenotypic features of individuals with the rare CNVs 2q13 and 4p16. Microarrays of 202 adults recruited from ID psychiatry services across England showed 11% with CNVs classed as pathogenic. 4 patients were examined with 2q13 deletions and 5 with 4p16.3 duplications. A deletion at the latter site causes Wolf Hirschhorn syndrome. No consistent phenotypes emerged. In one individual the non affected parent also had a deletion on two q13, raising questions about pathogenicity, and raising questions about the concept of benign familial variants. It is of course possible that the CNVs found in these individuals were not causally linked to the clinical

presentation. The study of CNVs revealed by genome wide microarrays remains in its infancy, and there are many uncertainties about interpretation.

Andre Strydom from UCL presented on cognitive decline and dementia in Down syndrome. 40% of those with Down syndrome in the UK are now aged over 40. The lifetime risk of dementia in this population is now estimated to be 80-90%. The median age at diagnosis is 55 years, with 25% occurring before 50 and 25% after 60. Down syndrome results from an overdose of 300 known, normal genes. There are problems of microcephaly, hypofrontality, small hippocampi and impaired memory, executive function and expressive language before the onset of Alzheimer's disease. In contrast to those with Down syndrome and therefore three copies of the APP gene, the syndrome of duplication of the APP gene results in the universal onset of dementia by the age of 60. The overproduction of amyloid cannot therefore be the whole story in the dementia of Down syndrome, so there must be neuroprotective factors that prevent a similar outcome. Good sleep protects the brain and 40% of individuals with Down syndrome have obstructive sleep apnoea. Intervention in children is likely to be more successful than in adults. Anti-amyloid treatments are often toxic and need to be given early. Most trials are conducted in those already exhibiting clinical dementia, and these trials have typically failed. Trials of antioxidants have been ineffective. Trials of a green tea extract which inhibits the kinase DYRK1A are underway as a result of successful treatment in Down syndrome mice. Andre and his team are developing a cognitive scale for Down Syndrome focusing on memory, EF and language, and will be exploring the use of this in early prediction.

Sophie van Rijn from the TRIXY (trisomy of X and Y) Centre in Leiden presented on social, emotional and behavioural problems in individuals with an extra X chromosome. Most are shy, timid, with poor peer relationships and high social anxiety. There is an increased prevalence of autism. There are global deficits in communication skills and language problems. Affective socialisation depends on intact executive function, including attention, inhibition, flexibility and working memory, which are all diminished in these syndromes. Similarly poor cognitive skills, such as recognition of facial expression and body language, contribute. Individuals with TRIXY but without autism show reduced social attention but heightened social arousal, whereas individuals with ASD show reductions in both domains.

Paul Hagerman from UC Davis spoke about pathogenic mechanisms of FXTAS, a condition caused by a pre-mutation expansion of the FMR1 gene. This is a multi-system disorder with problems likely to be a result of the development of inclusions in the cell nucleus. These are prominent in neuronal nuclei and are considered to be the result of the accumulation of problems associated with chronic DNA damage repair. The FX gene is too active in the pre-mutation, whereas it is inactivated in the full mutation. Levels of FMRP are normal but there is a substantial increase in mRNA which is toxic to DNA, in part through the formation of transcriptional loops. Individuals manifest an intention rather than a resting tremor, gait ataxia, peripheral neuropathy, cognitive decline, Parkinsonism and problems with bladder and bowel control.

Carol Samango-Sprouse from Washington DC presented results from a large prospective study of the neurodevelopmental outcome in prenatally diagnosed males with 47 XXY. She stated that it is naïve to think that low testosterone levels in these individuals are not significant, yet many have not been offered testosterone replacements. Individuals with this syndrome treated with testosterone show improvement in fine and gross motor skills, reasoning, self-esteem, muscle tone and comprehension, with reduced anxiety levels and a lower risk of osteoporosis. This syndrome is not associated with intellectual disability, and if this is present there should be a further search for additional causes. Motor milestones are delayed even with testosterone treatment and physical therapies are still required. This was a placebo controlled trial of 158 individuals. Treated subjects were given three shots of testosterone between the ages of 4 and 12 months. Booster shots between the age of 5 and 8 are recommended, with regular supplementation from the age of 11. No adverse effects were reported.

Randi Hagerman from UC Davis reported on advances in research in Fragile X. She noted that advances include both new treatments and the elucidation of new mechanisms of dysfunction when FMRP is missing or deficient. FMRP has been found to control the translation of multiple measures important in other disorders such as schizophrenia and ASD. A number of these new mechanisms have potential for identifying new targets for treatment. Intervention in early childhood is the best way to demonstrate treatment efficacy. She notes that there are early deficits in tryptophan pathways whatever the aetiology of ASD. SSRIs increase BDNF, but sertraline is the only one to increase dopamine in the striatum and nucleus accumbens, which can result in improvements in attention. Individuals with FXS have a GABA deficiency, which results in a reduction in habituation to stimuli and therefore an increase in sympathetic tone. As a result, they disproportionately attend to threatening information in their environment. Sertraline 2.5-5mg a day in children aged 2-6 with FXS can improve language especially in those with comorbid ASD.

The Novartis trial of AFq056 failed to demonstrate a statistically significant benefit, but some individuals experienced benefit from this mGluR5 antagonist. It is possible that administration in the trial did not occur at an early enough age. There is further work ongoing. There is a controlled trial underway of lovastatin in 10-18 year olds. This medication lowers RAS-ERK 1/2 and reduces excess protein synthesis. Benefit has been shown in FXS in an open label study. Parent Implemented Language Intervention (PILI) has been included in all participants in this lovastatin trial. This is conducted by therapists via Skype twice a week, and assists parents develop skill in stimulating language in the FXS child.

Studies of the GABA agonists ganaxolone and alphaxolone are underway, with one study producing only very modest results. Two companies are considering undertaking trials of cannabidiol, a non-psychotropic component of cannabis. Perhaps 10% of individuals with FXS show a Prader Willi phenotype and metformin can help with the insulin dysregulation seen in this population. There may be additional benefits on mood stimulation. Acamprosate has benefited some individuals. The challenge in all of this research is choosing robust outcome measures. High placebo responses can obscure subsets of individuals who respond positively to these agents. Eye tracking data can be a useful outcome measure.

The next meeting will be held in Leiden, Netherlands from 14th to 16th September 2017.

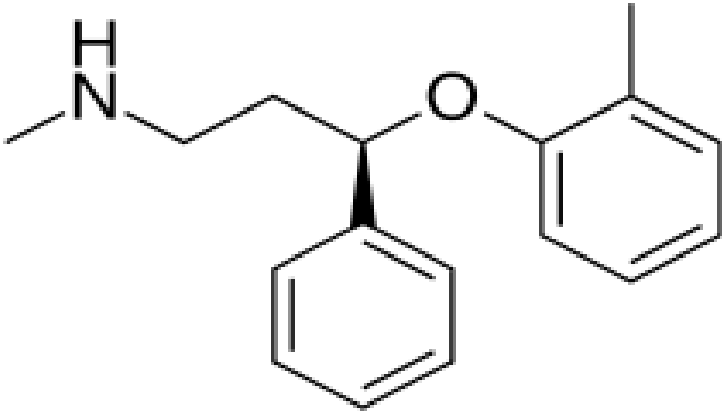
The Medicine Cabinet: Atomoxetine.

Judy Longworth

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Atomoxetine was originally developed as an antidepressant (tomoxetine) by Eli Lilly in 1990s but not found to be very efficacious whilst undergoing phase III clinical trials for depression. As a noradrenaline (norepinephrine) reuptake inhibitor had potential in ADHD and thus further clinical trials were performed from 1996 and finally released onto the Australian market in 2004 under the brand name of Strattera® and now there are generic versions also available such as Atomoxetine Amneal®.

When treating ADHD the psychostimulants (dexamphetamine and methylphenidate) are the first line agents and then atomoxetine is considered.

Atomoxetine clinical trials have included children and adolescents as well as adults with ADHD and smaller number of case studies has been reported in the developmental disability populations.

Some studies suggest that it's because of dopamine and noradrenaline dysregulation in the prefrontal cortex activation that ADHD patients have poor response to cognitive tasks of attention and executive functioning. It has also been shown hypothetically that low to moderate levels of dopamine and noradrenaline stimulation lead to better working memory and to reinforce learning and reward conditioning. Deficient dopamine and noradrenaline input will theoretically lead to increased noise and decreased signal, thus preventing a coherent signal from being sent. Hypothetically this causes ADHD symptoms such as inattention, hyperactivity, impulsivity and in some a combination. Strengthening prefrontal cortical output is hypothesised to be **beneficial in restoring patient's ability to tease out important signals from unimportant ones, and to manage to sit still and focus.** Adding stress from environment can further affect the noise and signal leading to noradrenaline and dopamine release thus inefficient information processing. With chronicity this can lead to a difficulty in treating as the patients present with dopamine and noradrenaline depletion.

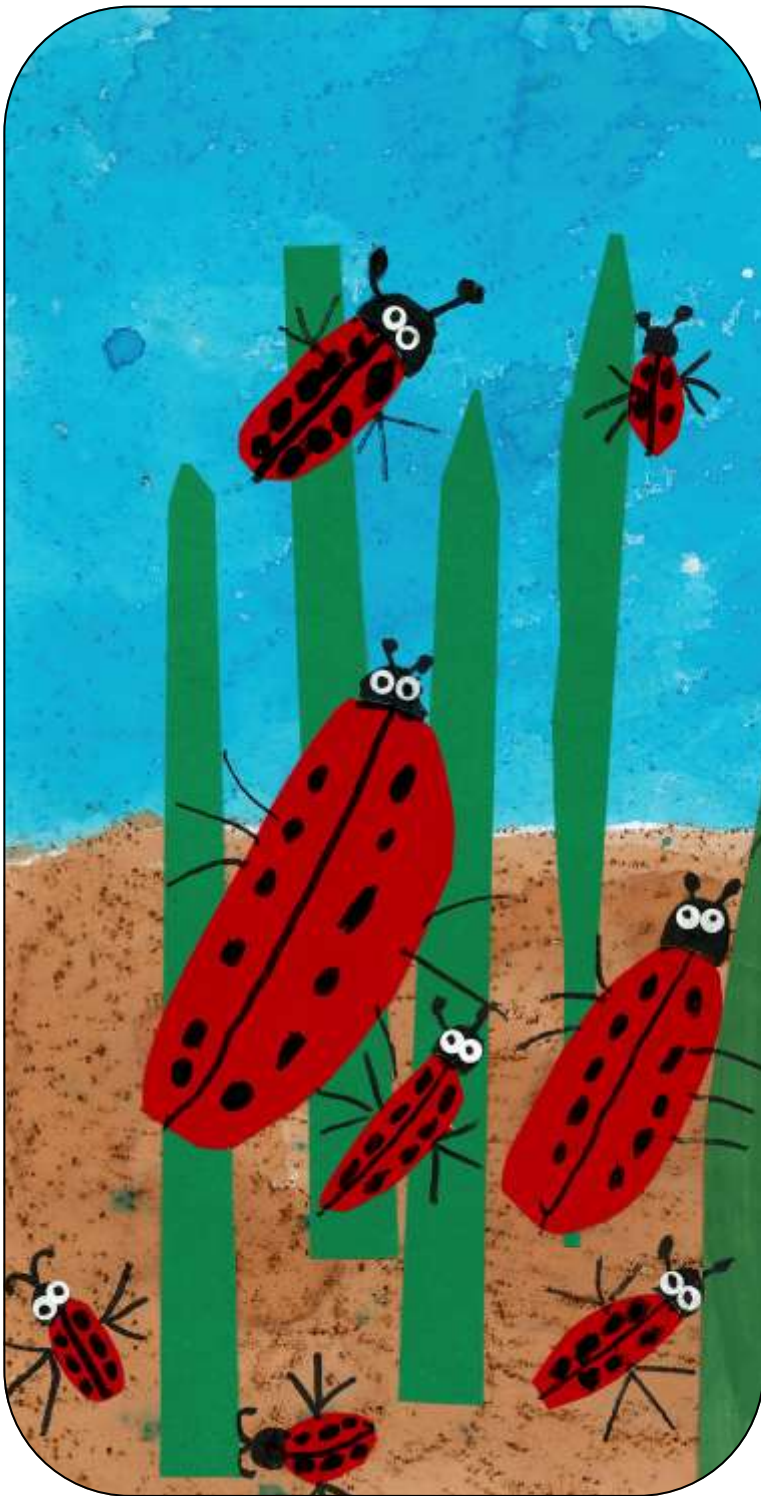


“Adverse effects are mostly dose related but can include insomnia...”

Atomoxetine clinical trials enabled atomoxetine to be licensed for use in children over the age of 6 years. Atomoxetine is available in Australia in 18mg, 25mg, 40mg, 60mg and 80mg capsules. It has a slow onset of action and response can take up to 4 weeks and the dose needs to be titrated slowly to minimise adverse effects and to see full optimization of the drug. It is not a controlled substance so unlike the psychostimulants has not shown any potential for abuse. Care should be taken in prescribing for poor metabolisers of CYP450 2D6 about 10% of the population as there could be increase in the adverse effects and only require a smaller dose as well as when given concurrently with fluoxetine or paroxetine – potent inhibitors of CYP450 2D6.

Adverse effects are mostly dose related but can include insomnia so taking the dose in the morning can help with this, dizziness and this can be helped by slowly getting up from a lying position or taking the dose at night when drug levels are highest. Others include fatigue and headache and these can be helped by again taking the atomoxetine at night or reducing the dose. Emotional inability and suicidal ideation have also been reported in the clinical trials and these should be reported back to the prescribing team. Atomoxetine as a raw product is known to be a gastric irritant so there is also upper abdominal pain reported as well as nausea, vomiting and even anorexia. As drug levels are not affected by food taking the dose with food or after food may help reduce these adverse effects. As there can be small increases in heart rate and blood pressure in the beginning of therapy these might be monitored by the prescriber and if one experiences a racing heart rate then the dose might be adjusted. Dry mouth, constipation and urinary retention as well as dilating of the pupils of the eyes have also been reported.

Serious life threatening liver damage problems with elevated liver function blood tests such as ALT and bilirubin have also been reported in small number of patients. The raised bilirubin causing jaundice will normalise once atomoxetine is ceased. The liver injury/damage may occur several months after initiation and persist for awhile after discontinuation. There is also a



Atomoxetine is a selective noradrenaline reuptake inhibitor or selective NRI or NET inhibitor. The mechanism of therapeutic action in ADHD, since the prefrontal cortex lacks high concentrations of DAT (dopamine transporter) dopamine is inactivated in this part of the brain by NET (noradrenaline transporter) thus, inhibiting NET increases both dopamine and noradrenaline in prefrontal cortex. In ADHD patient with weak noradrenaline and dopamine signals in prefrontal cortex, atomoxetine increases both noradrenaline and dopamine in the prefrontal cortex. As there is no effect on the noradrenaline and dopamine levels in the nucleus accumbens there is no abuse potential.

warning about increased risk of suicidal ideation with atomoxetine and thus increased monitoring should be done in the initial stages of therapy.

Summary of trials in ASD populations

The general population trials published in 2004 were for acute treatment (9 weeks) and long term (2 years) treatment for ADHD. There have not been the clinical trials in the younger age group or patients with comorbidities.

In a review by Fung et al in the journal, *Pediatrics*, the authors looked at irritable behaviour as measured by Aberrant Behavioral Checklist – Irritability (ABC-I) in various clinical trials and concluded for atomoxetine showed no significant difference from placebo for irritability but effect improvements in hyperactivity and impulsivity when measured on Aberrant Behavioural Checklist – Hyperactivity. (1). Irritable behaviour was defined as an excessive response to stimuli and also a consequence of emotional dysregulation. The atomoxetine study patients also had lower baseline scores for ABC-I when compared to other trial participants.

A larger study of nearly 100 children by Harfterkamp et al looked at the effect of atomoxetine on stereotyped behaviours and communication after small scale trials should some positive effect in ameliorating symptoms of ADHD in ASD patients. (2) When looking at the **Children’s Social Behaviour Questionnaire (CSBQ) sub-scale** for fear of change there were better effects with atomoxetine than placebo but no beneficial effects of atomoxetine on social functioning after 8 weeks of treatment. When this study was continued as open label extension for a further 28 weeks, the adverse effects of atomoxetine subsided and there was continued improvement in ADHD symptoms in children and adolescents with ASD. With some patients with ASD improvements take more time even up to half a year longer than in typical ADHD before the full response to atomoxetine has been established. (3)

The original small studies for atomoxetine in ASD patients found some effect in ameliorating the effects of ADHD in children and adolescents especially in core symptoms of ASD and social functioning. (2)

Problems with taking atomoxetine

Atomoxetine capsules cannot be opened as the contents can be irritant to the eyes. There is NO commercial liquid preparation so if unable to swallow capsules, atomoxetine use should be reconsidered. Food does not affect the absorption so can be taken with or without food but as it can cause gastric upsets giving with or after food helps to minimise stomach ache, nausea and vomiting.

Tips

Atomoxetine therapeutic action may continue to improve for 8-12 weeks and onset of therapeutic action can be seen as early as first day of dosing but is not working within 6-8 weeks then it may not work at all. The actions of noradrenaline on acetylcholine can cause decreased appetite, increased heart rate and blood pressure, dry mouth as well as urinary retention. Most side effects are immediate and often decrease with time. Although original trials were twice daily dosing efficacy has been for single daily morning dose. Unlike other medications used for ADHD atomoxetine does not have the abuse potential or have the ability to be diverted.

Atomoxetine should also be used with caution with patient with known cardiac disease due to its ability to increase blood pressure and heart rate. Monitoring should occur for cardiac parameters as well as liver and other physical health measures such as weight and height.

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For further information on atomoxetine:

1. www.nps.org.au

Addressing cardiometabolic risk factors in people with an intellectual disability

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The Department of Developmental Disability Neuropsychiatry (3DN) at UNSW Australia recently launched *Positive Cardiometabolic Health for People with an Intellectual Disability: an early intervention framework and resources*. Resources are free to download from the 3DN website.

Cardiometabolic health in people with an ID

People with an ID have poorer physical health than the general population and die at a younger age, often from preventable causes (Coppus 2013). A major cause of this health disparity is cardiometabolic illness (Draheim 2006). People with an ID have a higher rate of psychotropic medication prescription than the gen-

eral population, are prescribed psychotropics from a younger age and experience polypharmacy, all of which have major impact on their cardiometabolic health (Matson & Maham 2010). People with an ID have higher rates of mental health disorders than the general population (Cooper et al 2007); however psychotropic medications are also often prescribed for treatment of challenging behaviour. There is little evidence to support the effectiveness of prescribing for challenging behaviour in people with an ID (Tyrer et al 2008 & La Malfa et al 2006). Psychotropic medications frequently lead to weight gain, increased blood pressure and deteriorated metabolism of blood sugars and fats, even in children and adolescents

POSITIVE CARDIOMETABOLIC HEALTH FOR ADOLESCENTS WITH AN INTELLECTUAL DISABILITY: an early intervention framework

ADAPT YOUR PRACTICE while addressing STANDARD TARGETS¹

Plan for: communication adjustments; engagement with support networks; extra time; consent; teamwork.

Activity	Diet, lifestyle weight/waist	Socioeconomic resources	Blood pressure	Glucose regulation	Fasting blood lipids	Psychotropic prescription
<ul style="list-style-type: none"> Physical activity: (e.g. >60 mins per day) Screen-based activities: <2 hrs per day 	<ul style="list-style-type: none"> Non-smoker, balanced diet, no alcohol or other drug use BMI²: <85th centile Waist: height ratio: <0.5 	<ul style="list-style-type: none"> Socioeconomic status is associated with cardiometabolic health Ensure adequate access to housing, healthcare, transportation, education and employment opportunities 	<ul style="list-style-type: none"> <90th centile Use appropriate cuff size for arm circumference 	<ul style="list-style-type: none"> For most: FPG <5.5 mmol/L; HbA1c <42 mmol/mol (6.0%) For people with diabetes: HbA1c <58 mmol/mol (7.5%) For aversion to venepuncture see over 	<ul style="list-style-type: none"> Total Chol <4.4 mmol/L LDL <2.85 mmol/L HDL >1.56 mmol/L Trig <1.02 mmol/L 	<ul style="list-style-type: none"> Evidence based prescription to treat symptoms of defined mental illness and/or when challenging behaviours are severe and non-responsive to other interventions Minimum effective dose and length of treatment³

Any values outside of target range: DON'T JUST SCREEN – INTERVENE

Tailored intervention brochures can be downloaded from <https://3dn.unsw.edu.au/positive-cardiometabolic-health-id>

Using a person-centred approach PROVIDE TAILORED LIFESTYLE & NUTRITIONAL INTERVENTIONS:

If arranging multidisciplinary follow-up falls outside your practice scope make appropriate referrals to the person's GP and ensure proactive follow-up.

For physical health interventions create a **GP Management Plan** (MBS item: 721) and a **Team Care Co-ordination Plan** (MBS item: 723).

For Mental Health interventions consider using a **Mental Health Treatment Plan** (MBS items: 2700, 2701, 2715 or 2717) and referral to a psychiatrist and/or psychologist.

<ul style="list-style-type: none"> ↓ sedentariness; ↓ screen time; ↑ physical activity; Account for any co-existing physical impairments* Consider referral to exercise physiologist (MBS item: 10953) or physiotherapist (MBS item: 10960) 	<ul style="list-style-type: none"> ↓ energy intake; stop soft drinks/juices; ↑ vegetables and fibre Consider referral to dietitian (MBS item: 10954); exercise physiologist (MBS item: 10953); psychotherapist (MBS item: 10960); occupational therapist (MBS item: 10958)⁴ Referral to smoking or O&A cessation program 	<ul style="list-style-type: none"> Include social worker in multidisciplinary case conference (MBS items: 735 – 758). If the person has a diagnosed mental illness they can also receive individual social worker sessions (MBS item: 80150) Referral to disability support services 	<ul style="list-style-type: none"> Consider antihypertensive therapy if lifestyle intervention alone is insufficient* Limit salt in diet Education about blood pressure management 	<ul style="list-style-type: none"> Diabetes educator (MBS item: 10951) AT RISK: FPG 5.6 – 6.9 mmol/L; HbA1c 42 – 47 mmol/mol (6.0 – 6.4%); OGTT; if abnormal refer to specialist. Consider metformin if lifestyle intervention insufficient. DIABETES: FPG >7.0 mmol/L, RPG >11.1 mmol/L, HbA1c >48 mmol/mol Endocrine review 	<ul style="list-style-type: none"> Referral to paediatrician to consider Statin if lifestyle intervention alone is insufficient* Fibrate for triglycerides 	<ul style="list-style-type: none"> Consider switching, decreasing or discontinuing if metabolic side effects emerge; rationalise any polypharmacy; where possible avoid high metabolic liability medication as first line treatment⁵ (lithium, mood stabilisers – MBS item: 900); provide psychosocial education
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¹Certain causes of intellectual disability may alter baseline cardiometabolic risk. ²BMI may be inaccurate if person has muscle wasting caused by physical disability or an inability to stand upright. If BMI is >30, assume at risk waist circumference.

³Weight gain in first 3 months should be <5 kg (or <7% from baseline). ⁴In people with dietary insufficiencies consider checking folate and Vit B12. ⁵Consider referral to specialist if additional input required.

(Pramyothin & Khaodhiar 2010). Despite these potential side-effects, psychotropic medication is over-prescribed in people with an ID. Other cardiometabolic risk factors for people with an ID include higher rates of obesity and lower levels of physical activity than their peers (De et al 2008 & Emerson et al 2014), low socioeconomic status and certain genetic syndromes associated with ID (Barker et al 2007, Grotto et al 2008, Landesman Ramey et al 1999 & Wallace 2004).

Health professionals engaged in the care of people with ID would benefit from education on how to champion improved cardiometabolic health in people with an ID, including how to facilitate and support lifestyle and multi-disciplinary approaches to healthcare. It is essential that cardiometabolic health in people with an ID is monitored and appropriately managed, especially if psychotropic medication is prescribed.

Cardiometabolic Early Intervention Framework and Resources

Positive Cardiometabolic Health for People with an Intellectual Disability: an early intervention framework and resources has been adapted from a generalist monitoring framework* to address the specific cardiometabolic health needs of people with an ID (Troller et al 2016). Adolescent and adult versions of the Early Intervention Framework are available.

The Early Intervention Framework guides health professionals through screening of cardiometabolic risk factors in people with an ID. It provides age-specific, healthy target measures for each cardiometabolic risk factor as well as lifestyle and nutritional intervention strategies, including multidisciplinary referral options and relevant MBS item numbers. Additionally, the Early Intervention Framework outlines monitoring schedules for people with an ID on psychotropic medications, provides tips for overcoming fear or refusal of blood tests, identifies genetic syndromes associated with ID and their cardiometabolic risk profiles and links to consumer and clinician resources. The Early Intervention Framework has been endorsed by multiple professional colleges and consumer organisations including the Royal Australian and New Zealand College of Psychiatrists and the National Heart Foundation. A description of the development process and recommendations of the Early Intervention Framework has been published in the Australian Journal of Primary Health (Troller et al 2016). To access this publication, go to <http://www.publish.csiro.au/?paper=PY15130>

In order to empower people with an ID and their carers to encourage and engage their health professionals in monitoring cardiometabolic health, 3DN has also developed consumer postcards. These postcards provide information on staying healthy to people with an ID

“Postcards provide information on staying healthy to people with an ID ...”

and carers, and encourage them to bring the postcard to the doctor. The reverse side of the postcard has information for the doctor on how to access the Early Intervention Framework and Resources and the need to monitor cardiometabolic health.

The resources were launched at a forum at UNSW Australia in July 2016. To access the Early Intervention Framework and resources or view forum presentations from multidisciplinary experts in the field, go to <https://3dn.unsw.edu.au/positive-cardiometabolic-health-ID> All resources are free to download.

* The Early Intervention Framework was adapted from the psychiatry resource *Positive Cardiometabolic Health: an early intervention framework for patients on psychotropic medications*. This resource informs the assessment and management of cardiometabolic syndrome and related physical health issues in patients with severe mental illness. Adult and adolescent versions can be downloaded from <http://www.heti.nsw.gov.au/adolescentcma/>

References

Please see full reference list on page 13.

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The 15th World Congress of the International Association for the Scientific Study of Intellectual and Developmental Disabilities (IASSID) Melbourne 15-19/8/2016.

David Dossetor
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1300 people congregated in Melbourne for 15th World Congress of IASSID, a conference of both science and humanism, from the latest understanding on genetics, and mechanisms of the mind, to the importance of recognition and inclusion of people with intellectual disability, to sessions on spirituality and quality of life.

With 14 parallel streams there was plenty to choose from and different attendees experienced a totally different conference, although this was punctuated with plenaries and master lectures. Abstracts are published in *Journal of Intellectual Disability Research* 60: (7&8). The poster session with cocktails was a great source of networking with the international family of professionals in ID. Mental health was a more central theme than in the past, and indeed was children and adolescents. There were several messages indicating the importance of recognising mental health needs as something always to be considered separately to an intellectual disability, and an increased identification of pathways to wellbeing and achievement for example in people with Down Syndrome where inclusive education can lead to better self esteem, employment, participation and a positive wellbeing. Forrester-Jones reported on how faith-based spiritual organisations were more inclined to consider the spirituality of their clients with ID and in turn this led to clients having twice the size of social networks.

Here are a few of my highlights:

Jozef Gecz, from the Women and Children's Hospital in Adelaide gave an impressive update on the 'genetic architecture of neurodevelopmental disabilities (NDD)'.

His presentation highlighted how rapidly our understanding of genetics is changing, increasing our understanding of interactions between the complexity processes and the huge diversity of outcomes. He has been party to identifying 100 genetic disorders particularly of the X chromosome. He reminded us that the rate of developing genetic knowledge is remarkable: DNA discovered in 1953, first human genome sequenced in 1999, now a 1000 genomes are being sequenced a day and soon for \$1000 (although the opinion costs more). We have 21,000 coding genes which

is 2% of the genome, but the 'junk' ('non coding DNA') 98% may also be important, partly as the spacing can be important, but individual junk genes may have enhancing or modifying roles. For example, in a gene wide association study (GWAS) of obesity, a junk region gene related to enhancing white or brown fat. Some enhancer (but not coding) genes work through transcription factors. Eric Green predicted that the human genome would change the delivery of medicine, but currently cost prevents equity of access. We are 99.6% genetically identical with each other, 98% with a chimpanzee and 90% with a mouse. Deletions and insertions account for 15% of abnormalities. Everyone has 3-7 copy number variants (CNV).

Fragile X was discovered in South Australia and remains the most common identifiable cause of ID. The X chromosome has 800 genes of which 1/5 is involved in genes linked to ID. The proportion of people with ID that have a genetic abnormality has gone up to 62%: 12% have abnormality on chromosomal micro analysis, 27% on whole exome sequencing and 42% on whole genome screening, including CNVs and single nucleotide polymorphisms (SNP). Various epilepsies are genetic, but different mutations of the same gene can cause different NDDs. Conversely loss of a **functioning gene doesn't always cause disease**. Indeed, the same gene can have variable outcomes. Some cause several disorders, with different penetrance and environmental modifiers. BDNF (brain derived neurotrophic factor) polymorphism is a protective factor e.g. in Retts Syndrome. Nonetheless genetics explain 75% of the variance of educational progress. The Global Genetic Exchange helps identify single cases of ID. Gene DDX3X causes hypertension, movement disorder and behaviour problems, but is also a target for cancer treatment. There is an interesting overlap of 250 genes between NDDs and cancer. Most cancer genes are also involved in NDDs because they are involved in cell/neuronal proliferation and synapse formation. When one thinks that every cell has the same DNA, then the differentiation of structure and



function is due to enhancers and modifying genes/ processes.

He talked about PCDH19, a genetic deletion that leads to cluster epilepsy in girls. They appear to be healthy for 9 months, but regress developmentally with stress or infection and 62% have NDDs and 30% ASD. There seems to be a brain signaling problem involving androgen/progesterone, affecting GABA receptors and BDNF. They are deficient in allopregnanolone which is a brain steroid that is protective in PTSD, ASD and seizures. A novel neural steroid medication, Ganaxolone **is being trialed with some benefit. Boys aren't affected** because of a knockout effect of the gene, whereas the girls have two types of cells, some that are functioning **and some that aren't.**

We now know 700 different genes contribute to ID, 300 in Epilepsy, over 800 in ASD, 120 in Schizophrenia and over 20 in Cerebral Palsy (CP). However, there is huge overlap between the genes involved in NDDs such as ASD, ID, Schizophrenia, Bipolar, and Anxiety and more complex interactions between the genes, proteins and conditions. Many of these genes work at the synapse, and gene networks control the DNA, with environmental interactions. The gene TBCID24 can cause a NDD or deafness. A gene active in the perinatal period of brain development may become more active later in development. There has been no reduction in CP with the increase in rate of caesarians, indicating that CP is not due to perinatal insult. Indeed 14% have genetic findings, 10% de novo, 4% inherited and 40% more have candidate genes. A further 20% could be

explained by copy number variants (CNV). Environmental factors such as substance abuse, or too much or too little food affect genes and these effects are passed onto the next generation. This is part of epigenetics. Even with rare conditions, large cohorts are needed to elucidate genetic processes. For example, in schizophrenia an increase in sample size from 50 to 150,000 identified, instead of just 5 genes, 120 relevant genes. The heritability of Schizophrenia is 80%, Bipolar Disorder 60% but recent studies have brought it down for ASD to 50-55%. Some of these genes map to the immune system, for example complement 4A CNV increases pruning of neuronal dendritic spines. The Micro Biome of the gut also affects the human genome, e.g. in ASD development, including what we eat and what antibiotics we take. It is possible to transplant a metabolic syndrome with the Micro Biome. Some genetic metabolic syndromes can be treated. Ganaxolone has also been shown to improve Fragile X. Iceland now has a project to do the whole human genome on the total population. This presentation was impressive, as it introduces a much greater degree of complexity to genetic mechanisms. It may be that Fetal Alcohol Spectrum Disorder (FASD) is the archetypal epigenetic syndrome, partly by what alcohol does to moderator genes, and partly as some toxic effects from alcoholic in utero affect epigenetic processes and change development and behaviour in subsequent generations.

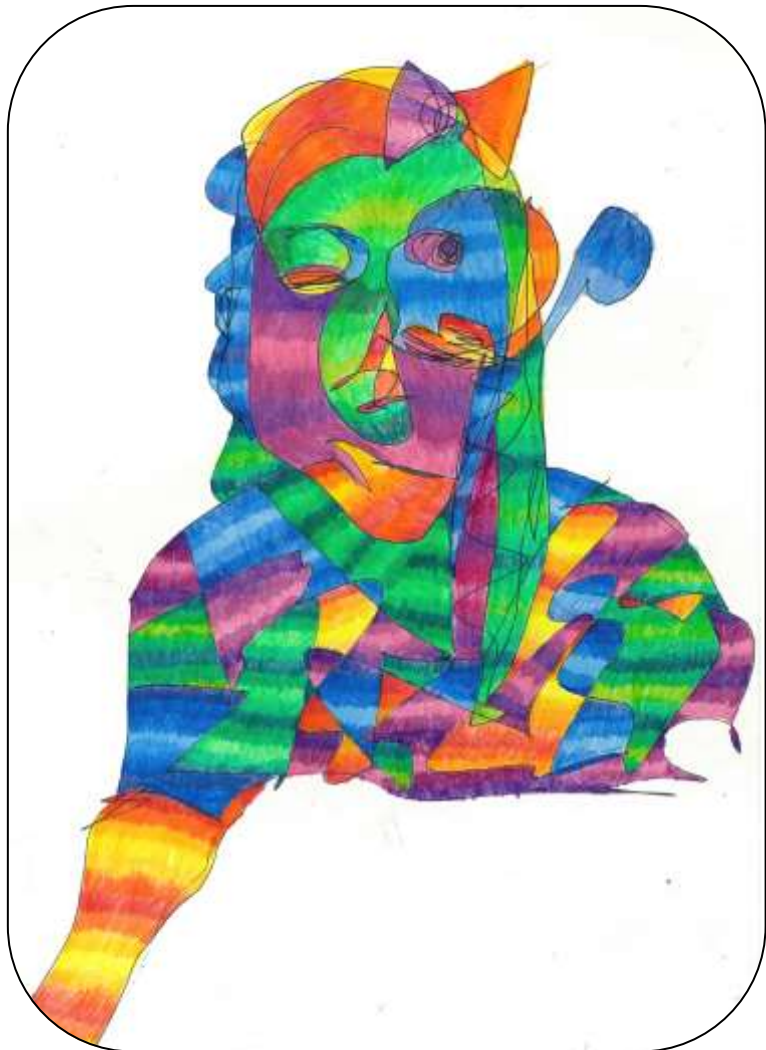
Irva Hertz Picciotto from the Mind Institute at UC Davis presented on the potential of environmental chemicals

to cause ASD. In California the incidence of ASD has gone up seven times in birth cohort studies between 1987 and 2002. Changes in diagnostic criteria, inclusion of milder cases and aging of the maternal population only accounts for a doubling. Prenatal exposure to toxins is important for neuronal development and migration. There is no single cause for ASD but outcomes are multifactorial. All factors only increase risk. Examples include lead poisoning, congenital rubella, thalidomide and valproate. Their 'childhood risk of environment' study is a case control study from 2002, looking at pesticides, heavy metals, organic pollutants, viruses and nutrients. Eg. Pesticides can interfere with GABA and Glutamine ion channels. Chlorpyrifos change parts of the brain involved in social processing, memory and IQ. Maternal metabolic conditions cause an increase in a range of conditions, such as inflammation, diabetes, hypertension, ADHD and ASD. Folic Acid is required for DNA synthesis and methylation. There are then genetic risk factors that interact with such environmental factors. Their study is looking for 1000 chemicals in blood samples, but there are 10,000 new organic chemicals registered a year! Even when a toxin is found to be relevant it only has a small increased risk and it is not specific for any NDD but increases the risk of them all. Accordingly, her study does not ac-

count for the dramatic increase. Although increased clinical skill/knowledge has clearly led to greater recognition of ASD, I was not impressed that she had **n't considered the environmental impact of the virtual digital world** on the neurodevelopment of attention and empathy. Other speakers commented on other big changes in our world e.g. the decline of infectious disease and the rise of immunological disorders like asthma. What is the influence of the epidemic of obesity on epigenetics and how does the MicroBiome interact?

In considering the mental health of people with ID, Prof Bruce Tonge recommended that the NDIS should include providing support to enable all to get an annual health check to improve the lack of equity of access. Although more genetic causes of ID are being identified, other predictors of mental health in ID include childhood disturbance, communication problems, lack of social networks, family functioning with disruption causing externalising behaviours and over protection causing anxiety, parental mental health, socio economic status, life events, accommodation and employment.

David Oppenheim from Haifa University presented his research on Mindful Parenting in ASD. While parenting does not cause ASD, the quality of parenting can have significant effect on the outcome and openness of a child with ASD. So often the diagnosis and burden of **care overwhelms a mother's capacity such that she loses her mindful parenting style**. His team did video assessments of mother and child interaction. **Mindful or insightful parenting: 'the capacity to see and feel things from the child's point of view' keeps a mother open to new information and practical approaches and greater anticipatory insight for their child, rather than an emotionality.** Only a third of mothers showed this level of acceptance and mindful parenting, which was not associated with the level of ASD functioning, was associated with a secure attachment in the child in 80%. However only 20% of children with ASD had a secure attachment. In a mainstream population attachment disorders are uncommon, although in deprived or abused populations it increases to 40%. He also found parent training intervention for behaviour problems could help a proportion of parents regain their mindful parenting style. Others have shown parent child interaction therapy (PCIT) can improve a **parent's mindfulness. His presentation makes clear that both the quality of parenting and attachment is of major importance in ASD and may well provide an early factor in the genesis of emotional and behavioural problems for which there could be intervention.**



“Both the quality of parenting and attachment is of major importance in ASD ...”

Sally Anne Cooper from Glasgow chaired a session on the DSM5 and DM-ID2 diagnostic manuals. The DSM5 diagnoses considers the influence of development, age, culture and gender on diagnosis but not ID, which is why the DM-ID needed renewal. Developmental perspective does consider children and adolescents and has a special section on severe and profound ID leading to limitations and needing adaptations. GAF has been changed for the WHO-DAS for assessing disability. It was encouraging to see UK and EU clinicians collaborating with the USA on the development of the diagnostic manuals, even if there is no funding available to look at diagnostic reliability and differences across the Atlantic. When should diagnoses be considered dimensionally rather than categorically?

Robert Pary presented on bipolar in ID. Levels of evidence remain low. Increased energy is a new primary symptom. Early presentation of bipolar diagnosis has gone up x40 in young people in the last 10 years. Disruptive Mood Dysregulation Disorder (DMDD) is hoped to be a better description of pre-pubertal mood problems which is not episodic like bipolar. Bipolar requires grandiosity for at least a week to distinguish it from DMDD in which mood changes are shorter.

Jane McCarthy presented on Trauma and Stressor related disorders, which now includes the attachment disorders of Reactive Attachment Disorder and Disinhibited Social Engagement Disorder, as well as PTSD. 42% of those with borderline IQ have an attachment disorder. By new definitions Adjustment Disorders resolve in 6 months and is found in 1% of those with mild ID and 2% in Moderate and Profound ID. It is underdiagnosed and suggested by behavioural change, although it is often difficult to identify stressors in ID and often has overlapping symptoms of inattention, impulsivity and hyperactivity. Those with severe ID may have more complex presentations.

Kieron O'Malley in his new book of FASD (Nova: New York; 2016) provides an interesting integration of comorbid psychiatric disorders in NDDs and has drawn attention to the recognition of Disorders of Arousal (in the Diagnostic Classification of Mental Health and De-

velopmental Disorders of Infancy and Early Childhood-Revised (DC: 0-3R)) which occur in infancy and in NDDs such as fetal alcohol spectrum disorders (FASD). Although FASD has not made it into DSM5, the appendix includes, under “Conditions in Need of Further Study”, a new disorder of Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE) which includes potential dimensions of: physical characteristics, Disorders of Arousal (including sleep and autonomic arousal), NDDs, and cognitive and executive function problems.

Jacob Burack from McGill University, who presented on **Zigler’s developmental models in Capetown IASSID**, presented on the importance of seeing ability and disturbance in a developmental context. He presented a paper examining the development of attentional cognitive skills in children with an intellectual disability compared to children matched for developmental age **which showed no deficit, upholding Zigler’s theory of developmental sequences.**

The controversy this evoked was extra-ordinary. 10 academics refused to review the article for publication, as it presented a challenge to the field of research in ID, which generally uses age and developmentally normal controls. Evidence demonstrating the importance of developmentally matched controls risks invalidating so much research in ID. They complain that a developmental approach invites too much complexity. Accordingly, most currently funded researchers fail to take account of this universal and humanistic framework which includes a social, emotional and physiological **‘whole person’, and which underlies every clinician’s approach to understanding their clientele.** Such an approach to theory and methodology can lead to major advancements in knowledge about the development of persons with ID and the intrinsic links across behaviour, brain, genes and environment. His experience revealed a deep division in methodology between those that consider developmental context, and those **that don’t.**

Helen Appleton from Giants Steps presented the framework for her research into anxiety in ASD/ID based on a dynamic model, proposed by Bartak et al in 2006, of tiers of anxiety from primary to quaternary.

1. due to deficits in processing information,
2. Ritualistic behaviour, poor sensory integration, obstacles to communication,
3. Internalising and externalising behaviours and
4. Psychiatric and personality disorders.

The implication is that all levels need considering, but the highest tier needs treatment in order to access the

“A real need for even single case reports on the value of intervention in severe or profound Intellectual Disability...”

issues of a lower tier. Her colleague Andrew Frakes described a pro-active audit tool for assessing challenging behaviour in ASD/ID by assessing the response to well-known strategies, rather than waiting for the results of an applied behavioural analysis. In the context of skilled teachers, this approach was a much quicker approach to providing intervention.

Flynn did a literature review of psychological and pharmacological intervention in severe and profound ID and using reliable measures of disturbance and outcome and found only 6 papers! This suggests a real need for even single case reports on the value of intervention in severe/profound ID!

Bob Cummins from Deakin University gave a powerful plenary on **Quality of Life: ‘the Essential Resources for a happy life’**. He has a huge database on QOL using his Personal Well-being Index (PWI). Short-term well-being is an emotion, but long-term well-being is a mood that is very stable and with a strong genetic basis for where you lie on the normative centile spectrum between 72-90% (mean 76.7). Those at 90th percentile are somewhat high and irritating, those at 72 are more introspective and show less affect. It is a **“normally positive state of mind”** that involves everything in our lives. His research has included those with (mild) ID, who generally do similarly well to others. The PWI is underpinned by the well-described 7 dimensions: adequate standard of living and health, intimate relationships, achieving in life, safety, community connectedness and future security. Multiple regression reveals **‘the golden triangle of happiness’**: the three most important factors of: sufficient money (those with disability need a bit more because of their needs), an intimate relationship, and achieving something each day. An intimate relationship is having someone you can winge to!

The other factor is internal reserves or resilience and the capacity to find meaning. His illustration: when you drop your coffee cup in the morning and think how stupid you are, it is the capacity to perspective take and say to yourself that despite that, you still have some

good qualities. PWI has data from 12 years old till old age. Alice Schiffers shows that family quality of life has similar domains, where support from others are important in maintaining a positive mood/PWI. Families also rely on trust and reciprocity which can vary considerably. Accordingly, maternal depression can influence everyone in the household. PWI slowly goes down through high school as schools do not focus on personal well-being but focus on exam performance, which in a lifetime perspective, has little benefit to PWI. Getting a job and finding an intimate relationship improves PWI; it goes down with children and a mortgage but improves in the over 55s and remains pretty stable. Bullying and discrimination is harmful to PWI. Depression and mental health problems directly affects the homeostasis of PWI or resilience. Politics should be about the greatest well-being for the greatest number. Curiously economists have realised that there is more to life than gross national product but their approach to quality of life is simplistic. Health-related quality of life is equally invalid: that quality of life is related to one particular symptom.

Chris Oliver from the Cerebra Institute in Birmingham gave a masterful lecture illustrating the multiplicity of aetiological factors can cause disturbance. Some of the examples include: physical difference such as different sleep architecture or causes of pain, temperamental difference, social attachment difference or cognitive difference. For example;



Pain: SIB in Cornelia de Lange's Syndrome (CdL) is often caused by gastro-oesophagitis. Similarly In Tuberose Sclerosis SIB is caused by pain especially from flank pain from kidney tubers. He recommends the Face, Legs, Activity, Cry, Consolability scale or FLACC scale (http://prc.coh.org/PainNOA/Flacc_Tool.pdf) for eliciting pain.

Social Attention and Preference: Angelmans is seen as a happy disposition with laughter and sociability but underlying this they have a strong drive for attention. This the drive for attention also leads to aggression when attention is withdrawn such as by turning away, leading to hair pulling and grabbing to greater levels than in other conditions such as Cri du Chat or CdL. Children with Smith Magenis Syndrome are found to have a strong attachment to their primary care giver. At school they tend to attach to one teacher. Experimentally when exposed to an unfamiliar care giver, they continually turned to their mother, in a way not seen in Down Syndrome. It was concluded that they have an excessive attachment to their mother, which causes considerable stress to their mother (on top of being awake at night with their inverted circadian sleep cycle). They may also have high sociability but low social cognition leading to problems in their teens, **because they can't read social cues.**

Cognitive Difference/Executive function: Prader Willi Syndrome get stressed with demands to switch attention, using executive function testing (EF), which was then correlated with increased repetitive questioning and need for routine. fMRI found related to reduced activity of the fronto-parietal tract compared with typically developing young people, explaining why they have to work harder on the EF task. In effect, carers seeing their behaviour as stubborn and obstinate was **pejorative, as they couldn't switch task, not that they wouldn't! Interestingly, whereas PWS have temper tantrums with change (after which they are remorseful), Fra X become anxious. Every year his research group**



add new observation to help understand individual differences from their study of syndrome specific clinical features. Each observation raises questions as to whether such phenomena may also apply in certain clinical cases without a gene specific phenotype.

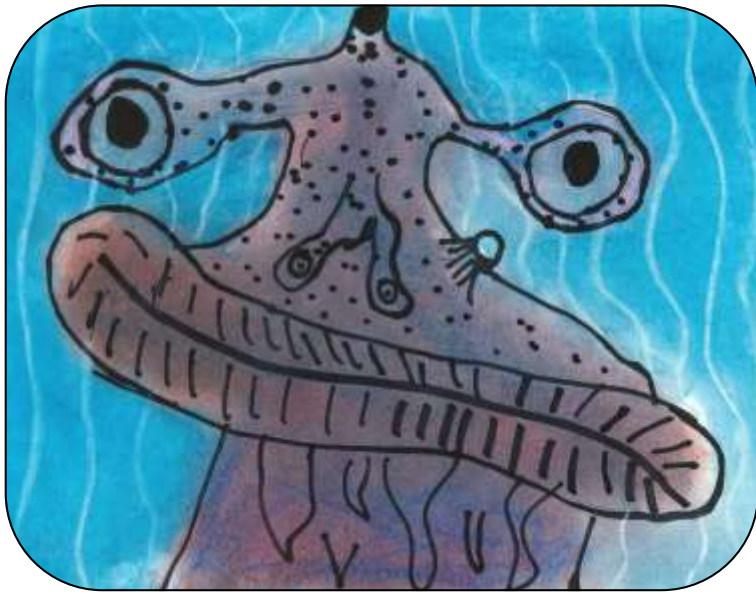
At the end of such a diverse array of presentations, I was forced to conclude that despite different approaches to deconstructing our understanding of emotions and behaviour, the syndromes of psychiatric disorder remain resilient constructs of a different type or order, which are important to study, diagnose and to treat: psychiatric treatment is still an important branch of medicine and recognising their importance in people with intellectual disability remains an important discipline that warrants continued development and investment.

“The syndromes of psychiatric disorder remain resilient constructs of a different type or order...”

International Association for the Scientific Study of Intellectual and Developmental Disabilities (IASSID) Symposium on Quality of Life in Children with Intellectual Disability.

Donna White

*State-wide Behaviour Intervention Service
Ageing, Disability and Home Care,
Department of Family and Community Services NSW.*



This symposium contained three papers from the Telethon Kids Institute in Australia focusing on the quality of life for children with intellectual disabilities. All three papers involved interviewing parents of the children (aged 6-18 years) by phone then transcribing and analysing the responses for themes related to their research questions.

The first paper by Murphy and colleagues (2016) described qualitative research that aimed to explore aspects of life that contribute to happiness and wellbeing in children with Down syndrome (n = 17). The research found that there were 11 domains that were important to quality of life for children with Down syndrome. These domains were broadly grouped into three areas,

- *Daily activities*: Communication and expression, movement and physical activity, routines and predictability, independence and autonomy.
- *Health and well-being*: Physical health (e.g. fatigue, pain, respiratory issues, infections, comorbidities), behaviour and emotional wellbeing, personal value.
- *Community and environment*: Social connectedness and relationships, variety of activities, nature and outdoors, access to services.

The second paper by Epstein and colleagues (2016) investigated quality of life as a composite of life experiences for children and Autism Spectrum Disorder (ASD). The results of the thematic analyses of parent responses (n=28 families) were then compared to the domains from the Pediatric Quality of Life Inventory (PedsQL) in order to develop a framework for measuring quality of life in children with ASD. They found 10 domains that were applicable to children with ASD that are described below.

Health and Well-being

1. Physical health e.g., body pain, sleep, energy levels, eating, gastrointestinal health.
2. Behaviour and emotional wellbeing e.g. body language, expression, sensory stimulation, aversion, repetitive behaviour.
3. Relaxation and reassurance e.g. calming and relaxing actions, cuddling, physical contact, “down time”.

Daily Activity

4. Communication and expression e.g., choice-making, sharing thoughts and feelings, non-verbal forms of expression.
5. Flexibility and routines e.g., familiar and predictable aspects of life, stopping a preferred activity with ease, topics of intense interest.
6. Leisure and recreation e.g., physical activity; “screen-time” via TV, computer, video-game; constructing with Lego; drawing; designing.
7. The natural environment e.g., time spent in nature and outdoors; contact with pets; interest in animals (e.g., park, zoo, aquarium).
8. Independence and autonomy e.g., mastery and achievement of different tasks; developing skills; learning something new.

Community Immersion and Services

9. Social desire e.g., social connectedness; shared enjoyment; social disinterest.

10. Services and associated outcomes e.g., access to supports and resources; advocacy; financial assistance.

The third paper of the symposium by Downs and colleagues (2016) used the results from the thematic analyses of the parent interviews (n = 86 families) to map quality of life domains across children with Down syndrome, Autism Spectrum Disorder with intellectual disability, cerebral palsy with intellectual disability, and Rett syndrome. They found that,

- Most quality of life domains were common across the four groups;
- **Some domains were related to the child's level of functioning** i.e., domains of personal value for Down syndrome; domain for independence and autonomy was not applicable for children with Rett syndrome.
- Some domains were specific to children with ASD, i.e., relaxation and reassurance; flexibility and routines; and social desire.

When Downs and colleagues (2016) compared these domains to other quality of life measures, they found **that there were two new domains that were "unique"** to children with intellectual disabilities. These were the domains of,

- Stability of daily routines (identified for children with Rett syndrome); and,
- The natural environment (common across all 4 groups).

Further analyses of the data and formulation of the domains into a quality of life framework revealed that,

- There needed to be systematic management of physical health;
- **That a child's behaviour was a critical marker for quality of life;**
- That social participation was multifaceted and that there needed to be a balance between the need for connectedness and a preference for solitary play or downtime for some children; and,

“A child's behaviour was a critical marker for quality of life...”

- Exposure to nature and animals promotes mental health and wellbeing.

The presenters concluded that the research identified specific domains that could be used as a framework to measure the quality of life of children with intellectual disabilities. This could also have the potential to more clearly identify the support needs of children with intellectual disabilities, be used as an outcome measure, **and develop a tool that obtains children's views about their own quality of life.**

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Inaugural meeting of the Bowral Mental Health Intellectual Disability Professionals Network

On November 15 2016, colleagues from a number of government and non-government agencies came together to explore the value of forming a network of professionals working to promote the mental health and well being of individuals with an intellectual disability. The group was co-convened by Claudia Tapia, Behaviour Support Specialist (Psychologist), Behaviour Support Team, Quality & Reform, ADHC, and Katelynd Turner, Senior Practitioner, Person Centred Behaviour Support, Client Programs Northcott. Support for the initiative was also provided by Angela Miller, Project Officer, Mental Health Professionals Network Ltd.

A number of organisations and agencies were represented at the meeting including The Disability Trust, Life Without Barriers, St Vincent de Paul, Schizophrenia Fellowship of NSW Inc, Child Youth and Family Services, NSW Health, Department of Education, **and** Family and Community Services. The range of professional backgrounds included psychology, special education, social work, speech pathology, mental health nursing, and social sciences. There was also significant operational and management experience in the room.

The first presentation of the day was delivered by Lesley Watson and Donna White, Statewide Behaviour Intervention Service (SBIS), ADHC, and Jodie Caruana, School Link Co-ordinator, **Children's Hospital at Westmead**. The presentation outlined the work of the Developmental Psychiatry Partnership, including the outcomes of recent collaboration and shared plans for the coming year. The afternoon was spent discussing the Terms of Reference for the Network and the schedule of meetings for 2017.

It was exciting to sit in a room of professionals so keen to work together, share ideas and promote the growth of knowledge and skills particular to working with individuals with intellectual disability and mental health issues. Developing a sustainability strategy to ensure the Network remains viable into the future will remain a priority in 2017. Groups of professionals such as the Bowral MHID Professionals Network will be critical sources of learning and support as we transition to the NDIS.



Carers Australia Carer Checklist



[Carers Australia](#) is the national peak body representing Australia's carers, advocating on behalf of Australia's carers to influence policies and services at a national level. It works collaboratively with partners and its member organisations, the Network of state and territory Carers Associations, to deliver a range of essential national carer services.

In regards to the National Disability Insurance Scheme (NDIS), it is common for carers to feel confused, nervous and a bit overwhelmed by how to help the person they care for to prepare for this new scheme. Carers Australia has developed the *Carer Checklist* to assist unpaid carers to describe the support they provide during NDIS assessment and planning meetings.

The questions throughout the *Carer Checklist* prompt carers to think about what they need to tell the National Disability Insurance Agency (NDIA) in their initial planning meeting. It is not compulsory, but will help **the NDIA to understand the carer's role, so that the support package allocated best meets the needs of the person with a disability and supports the carer's role.** The *Carer Checklist* covers the following areas:

The Caring Context

- Additional caring responsibilities
- Other supports
- Culture and religion

The Caring Role

- Personal care
- Mobility
- Meal preparation, eating and drinking
- Safety
- Domestic life
- Providing emotional support and behaviour management
- Health and treatment
- Communication and social participation
- Advocacy and representation
- Coordination of services and supports
- Employment education and training
- Time spent on all caring activities

The Impact of Caring on You

- Physical & mental health
- Financial
- Time
- Employment and education

Services & Support

- Continuing care
- Accessing services & supports
- Caring needs
- Emergencies/alternative arrangements

Additional Information

The **Carer's Checklist** can be accessed at <http://www.carersaustralia.com.au/ndis-and-carers/support-for-families-and-carers/>



KidsQuit

Smoking Cessation Brief Intervention E-learning

Change lives by talking to young people and their parents and carers about stopping smoking.

The Children's Hospital at Westmead and NSW Kids and Families are proud to announce the launch of the KidsQuit Smoking Cessation Brief Intervention E-learning program. This free online interactive program takes approximately 30 minutes to complete and is backed by evidence-based research.

The objectives of KidsQuit are to increase **knowledge of the 5A's of smoking cessation**, boost confidence in performing brief interventions and provide resources and support to professionals. The training is suited for not only health professionals, but any professional who comes into contact with adolescents or parents and carers who smoke.

Three additional modules have been added to focus on high risk adolescent groups including those with mental health conditions, pregnancy and Aboriginal people. KidsQuit is unique in its focus on adolescents, parents and carers and how to best educate and empower those working with young people to talk to them about quitting smoking.

Visit <http://www.kidsquit.org.au> to access this free training, and receive a certificate on completion for use in gaining professional development points.



The beautiful artworks in this journal are taken from the participants of the **Operation Art project** at the Children's Hospital at Westmead. You can find out more at <https://www.artsunit.nsw.edu.au/visual-arts/operation-art-2014>

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